# THE BEARINGS

OF

# CHRONIC DISEASE OF THE HEART

UPON

# PREGNANCY, PARTURITION, AND CHILDBED

WITH PAPERS ON

*PUERPERAL PLEURO-PNEUMONIA AND ECLAMPSIA*

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*TO*

*JAMES MATTHEWS DUNCAN, M.D. LL.D. Etc.*

*WHO*

*BY EXAMPLE AND RESEARCH*

*HAS SO LARGELY*

*CONTRIBUTED TO THE ELEVATION AND PROGRESS*

*OF*

*GYNÆCOLOGICAL SCIENCE*

**This Work is Dedicated**

*AS A TOKEN OF RESPECT*

*BY*

*HIS FRIEND AND FORMER PUPIL*

*THE AUTHOR*

# PREFACE.

THE subject of Heart Disease in its relations to Pregnancy, Parturition, and Childbed, while it has attracted a considerable share of attention on the Continent, has hitherto failed to secure from the profession in this country an amount of study commensurate with its importance.

Recognising this fact, the author has, in the following pages, endeavoured to deal with the question in such a manner as may help to remedy this deficiency. How far he has succeeded in that object, he leaves to be decided by the judgment of his readers.

Papers on Puerperal Pleuro-Pneumonia and Eclampsia are included in the volume, on the ground that these diseases furnish important examples of the deteriorating influence exerted by pregnancy upon the blood and blood-vascular system, and are thus closely associated with the main theme of the work.

29 CHARLOTTE SQUARE,
EDINBURGH, 19*th October* 1878.

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# ON THE BEARINGS OF CHRONIC DISEASE OF THE HEART UPON PREGNANCY, PARTURITION, AND CHILDBED.

## CHAPTER I.

### INTRODUCTORY.

FEW subjects can be conceived calculated to attract to themselves more serious thought and attention on the part of obstetricians, than the bearing of organic disease of the heart upon the condition of the pregnant, parturient, and lying-in woman, on account of the important issues at stake; and yet scarcely a subject that borders on the mutual region occupied by the obstetric and the pure physician seems to have received less study from either the one or the other. The reason, no doubt, is partly due to the cramping effects of a too rigid specialism, which tends, on the one hand, to concentrate the attention of the obstetric practitioner exclusively upon matters purely obstetrical and gynæcological, whilst, on the other, it affords the pure physician only comparatively few opportunities to watch the influence of such disorders on particular cases, inasmuch as the patients are at such times pretty exclusively under the care of a physician-accoucheur.

But as every woman has not only a uterus and ovaries, but also a liver, heart, lungs, kidneys, etc., it is, I hold, the first duty of every obstetrician to make sure that he is a physician in the first instance, and an obstetrician only in the second place. Unless he does so, he can have only an extremely

inadequate idea of the scope and importance of his department of the medical art and science; and he is, at the same time, certain to allow his position in practice to degenerate into the cultivation of what at best is only an outpost of applied surgery. Much of the disfavour and affected hauteur with which obstetrical medicine has frequently been treated by pure physicians and surgeons, is doubtless traceable to the somewhat contracted notion that we ourselves have formed of the value and dignity of obstetrics, and can only be removed by our strongly asserting and proving that neither in loftiness of aim, in scientific precision, nor in practical importance, is gynæcology a whit behind either of her sisters. Only by entertaining an elevated idea of his department and its duties, can the obstetrician expect to practise with success and comfort the all-important branch of the healing art which he has selected as his own.

Being convinced that the general views entertained by the members of our department of medicine on the subject in hand are held by them with considerable looseness, and that a discussion upon the question might be the commencement of an inquiry which would eventuate in clearing up some points that are at present unsettled, I have for some time back been directing a good deal of attention to the matter, in the endeavour to form accurate views respecting it. With this object before me, I have collected together all the cases that I have notes of, which have occurred in my own practice or in that of my dispensary pupils, and to these I have added several obtained from medical friends, and also others selected from medical literature. I have thus been able to put together a considerable collection of what seem to me extremely valuable cases.

Now, I would begin by pointing out that nearly all systematic works on midwifery mention organic heart disease with more or less prominence as a serious complication of

pregnancy and of parturition, more particularly of the latter, and at the same time give suggestions for the management of such complications, especially when they interfere with labour. This is all well and good, and as the regulations for their management are, on the whole, tolerably appropriate, I hope this single reference to the point will suffice, and that I shall not be expected to recur to it.

But, so far as I know, there has been no attempt on the part of any English obstetrician or physician to differentiate the effects of the special cardiac lesions, and to define in any way their individual bearings upon either pregnancy or parturition. Or, to put it otherwise, I know of no English writer who has striven to put obstetricians in a position to answer with intelligence the question that is every now and again asked of us by a patient who knows she has heart disease,—Should I marry? or do I run great risk in marrying? Now, though I believe that under such circumstances ladies will almost invariably ask their doctor's advice and follow their own liking, yet we ought to be able to give sound counsel, so as to clear our own consciences of all blame in such an important decision. What is more, I think I shall be able to show in the sequel, that not only have physicians not written on this subject, but that a great amount of ignorance exists in the professional mind upon the matter,—ignorance that might and ought to have been greatly removed, if even a small share of attention had been bestowed upon the subject, and which is therefore much to be deprecated.

I do not intend to refer in this work, except incidentally, to the disease known as ulcerative puerperal endocarditis. I mean to restrict myself specially to those instances of organic defect in the heart which have come into existence independently of pregnancy, but which, as is well known, may be seriously changed in the course of pregnancy, or during parturition or the lying-in period, or may so influence the

current of events in these conditions as to require the most anxious attention and careful management on the part of the physician-accoucheur.

But even when so restricted to cardiac complications of a chronic nature, the subject is a very wide one indeed, and one that is extremely unmanageable, on account of the complex factors that come into action under the conditions presupposed.

It will be seen from these statements that my aim is not merely to refer to the general bearings of cardiac disease upon the conditions of pregnancy, parturition, and the lying-in period, but also and specially to ascertain, so far as our present knowledge will allow, the influence of the individual cardiac lesions upon these physiological states. I feel satisfied that you will agree with me in regarding this object as not only an interesting but a very necessary one; for the rapid advancement that has been made in the pathology, diagnosis, and treatment of diseases of the heart by pure physicians of late years, surely demands from accoucheurs something more exact than mere vague references in dealing with this important subject.

It is certainly devoutly to be wished that, if possible,—given that one of our patients is the victim of a special cardiac lesion,—we should be able to predict what are the special additional risks, if any, to which the pregnant, parturient, and lying-in conditions expose her, and what are the prophylactic or therapeutic measures we are bound to adopt, so as, if practicable, to avoid or diminish such risks. Such a result, I opine, can only be attained by a careful and unbiassed investigation into the physiological and pathological conditions involved, and perhaps even then can only be imperfectly secured.

Though at present we are not in a position to effect this object completely, yet I do think we ought to aim at such

as an end; and further, that we are a considerable way on the road to it if we only gather up with care and utilize the information that lies at our disposal, whether in our own experience, or scattered here and there throughout current medical literature.

With these ends in view, I have determined to adopt the following order in the management of this investigation :—

I. I shall endeavour to trace in historical sequence, and briefly estimate, the chief attempts that have been made to elucidate the special physiological changes that take place in the heart during pregnancy.

II. I shall try to trace historically, and criticise shortly, the efforts that have hitherto been made to discover the mutual bearings of cardiac disease and of pregnancy, parturition, and childbed upon one another.

III. Records of thirty-one cases will be given, accompanied with such remarks as they seem to suggest.

IV. I shall make some general observations, and endeavour to deduce some practical inferences, in regard to the prognosis and treatment of such conditions.

## CHAPTER II.

### HISTORY AND ESTIMATE OF THE CONTRIBUTIONS TO OUR KNOWLEDGE REGARDING THE NORMAL CHANGES OCCURRING IN THE HEART DURING PREGNANCY.

HAVING thus cleared the way by these preliminary observations, I proceed to the consideration of the first part of my task, which has chiefly to do with the question raised originally in the French school, viz., whether the left ventricle of the heart does or does not normally hypertrophy during pregnancy. Along with this come other questions that are ætiologically connected with it, such as the presence or absence of increased arterial tension during pregnancy, the condition of the pulmonic circulation, etc.

As to the subject of cardiac hypertrophy coincidently with pregnancy, we find that Larcher, in 1825 and 1826, during the time he served as Interne in the Paris Maternity Hospital, first directed attention to the circumstance that the left ventricle of the heart normally becomes hypertrophied during pregnancy. He examined during these years the alarming number of 130 hearts of patients who died at this hospital, the majority of them, he says, of childbed fever. Consequently it appears only just to hold, with Larcher, that the organs of these patients may be fairly assumed to have been healthy, except in so far as the fever interfered with them. The ages of the patients varied from eighteen to thirty-five years. From these observations Larcher concluded, first, that the heart in the human species is normally

enlarged during the period of gestation; second, that the enlargement affects almost exclusively the left ventricle, the left auricle and the right side of the heart being little if at all changed; third, that it varies in amount from a minimum of one-quarter the normal thickness of the ventricle, to a maximum of one-third of it; fourth, that it constantly occurs; fifth, that it disappears but slowly during lactation. Larcher also deduced many pathological results from this enlargement. He believed, for instance, that repeated pregnancies within short periods, more especially if the patients at the same time suckled their children, might give rise, even though the heart was perfectly sound in all its openings, to permanent hypertrophy; that it tended to produce epistaxis, cerebral congestions, and even cerebral hæmorrhages; that it aggravated pulmonary disease occurring coincidently with pregnancy, such as bronchitis, pneumonia, or phthisis, etc. etc. There is no doubt that Larcher exaggerated the effects of the hypertrophied heart in connection with pregnancy; but what discoverer of a new fact in science, real or imaginary, is not inclined to overrate its importance? It is further to be observed that, though Larcher explains the result of cardiac action on these complications of pregnancy in so restricted a manner, yet there is reason to believe he was quite correct as to many of his observations. As we hope to show, it appears to be the fact that the pregnant condition modifies and affects the conditions of the collective blood vascular system in an all-important manner, so that disease of lungs, kidneys, etc., are greatly conditioned thereby. Larcher saw this result, though he explained it by reference to one only of its numerous elements of causation.

Larcher's views were first published in 1828, but the full exposition of them is to be found best given in a memoir addressed to the Academy of Science, the 6th April 1857, and published in the *Archives Générales de Médecine*, V^e^

série, tome xiii. p. 291, 1859. These views, though generally accepted by his countrymen, were not universally so, and were denied by Dr. Rochoux, *Dictionnaire de Médecine*, t. iii. p. 504.

This led Dr. Beau, in 1843, to direct the attention of his then Interne, M. Ducrest, at the Paris Maternity, to the same point as had previously been investigated by Dr. Larcher. Ducrest examined 100 hearts, and makes the following statement relative to the thickness of the left ventricle. His plan was to measure accurately the left ventricle at its thickest part. The ages of the patients whose bodies were examined varied from twenty to thirty years. The maximum thickness of the ventricular wall occurred in five cases as 1·8 cent., with the exception of one case, when it reached the amount of 2·2 cent. The minimum thickness of 1·1 cent. was found in eight cases. In the majority of the cases the thickness was found 1·6 cent. The mean of all the measurements gave a thickness of 1·5 cent. M. Bizot gives the mean thickness of the left ventricle in the female as 1·0 cent.; and, comparing the results of his own observations with this anatomical fact, M. Ducrest argues that his results bear out to the full the truth of Dr. Larcher's views. It will be observed that the mean thickness of 1·5 cent. found by Ducrest is equal to 1½ of the mean thickness of 1·0 cent. assigned by M. Bizot to the average female heart. It would be interesting for us to know whether in all cases M. Bizot measured the thickest part of the left ventricle, as M. Ducrest tells us he himself did, as otherwise we are not sure whether the two authors are comparing the hearts on exactly the same grounds. M. Ducrest's results are found summarized in M. le Dr. Beau's 'Memoire sur le Bruit des Artères' (*Archives Générales de Médecine*, IV^e^ série, tome x. p. 28).

In a prize essay sent in to the French Institute of Medicine, Zambaco made similar researches to those of Larcher and

Ducrest in this inquiry, comparing the hearts of women who had died in childbirth with those of others who were not pregnant at the time of death, and found in the former that there occurred enlargement of the left ventricle.[1]

Béraud arrived at a similar result in like manner.[2]

Blot next attacked the problem from a different aspect. He did not compare the hearts by measuring the thickness of their walls, but by ascertaining their weights. After an examination of twelve hearts of women who died during pregnancy or the lying-in period, he found their average weight to be 9 oz. 38 gr. troy weight; whereas the average weight of the healthy heart of young women is about 7 oz. 120 gr. troy weight. It would thus appear that, according to Blot, the heart of the pregnant female gains in weight fully one-fifth during the pregnancy. He agrees with the other observers that the hypertrophy is confined almost exclusively to the left ventricle.[3]

It is important to notice in this connection that Bizot, in his estimate of the average female heart, is considerably under the average as given by Peacock, p. 4, vol. iv. of Reynolds' *System of Medicine*. It is there stated that the mean weight of the hearts of females dying between the ages of twenty and fifty-five years is 8 oz. 13 dr. avoirdupois = to 8 oz. 15·5 gr. troy.

The ordinary range in acute cases within the same ages is given as from 8 oz. to 10 oz. avoirdupois = 7 oz. 140 gr. and 9 oz. 55 gr. troy respectively.

The ordinary range in chronic cases, again, is put at from 7 oz. to 9 oz. avoirdupois = 6 oz. 182·5 gr. and 8 oz. 117·5 gr. troy respectively.

[1] Joulin, *Traité Complète d'Accouchements*, p. 383. Paris, 1867.
[2] *Loc. cit.*
[3] *Cazeaux's Midwifery*, edited by Tarnier. American edition, translated by Bullock, p. 160. 1870.

Still, Blot's results would indicate a certain though not so great amount of hypertrophy, even if we accept the more recent statement of Peacock in regard to the mean average as the more correct of the two.

More lately, in the 104th number of the *Gazette des Hôpiteaux*, Duroziez has attempted to demonstrate this hypertrophy on the living subject by an exhaustive examination, by percussion, of the cardiac region, in women shortly before and shortly after delivery. In pursuance of this investigation, he examined 135 women in the Parisian Midwifery Clinique, and is led thereby to the following conclusions:—

1. The hypertrophy of the heart in the puerperal condition is demonstrable; the heart does enlarge during the pregnancy.

2. During the first day after delivery the left cavities of the heart diminish in size, the right cavities remain enlarged.

3. At the time when the secretion of milk begins, about the third day of the lying-in period, the heart is lengthened. The heart remains during lactation enlarged; becomes small, however, if the woman does not suckle.

4. In women who have frequently borne children, the heart is larger than in such as have only given birth once or twice.

I am sorry I have been unable to get hold of Duroziez's original paper, although I have tried hard to do so, and have therefore been forced to content myself with an abstract of it contained in the *Monatsschrift für Geburtskunde*, Bd. xxxii. S. 479.

In this connection it is proper to state that Fritsch, in a paper published in the *Archiv für Gynäkologie*, Bd. viii. S. 376 and 377, while allowing that there is increased cardiac dulness, denies the possibility of arriving at such accurate results from percussion as Duroziez affects to have done,

in consequence of the insuperable difficulties presented to exact percussion by the presence of the enlarged female mamma. Fritsch is inclined to explain a considerable amount of the increased dulness by upward and forward displacement of the heart from the pushing up of the diaphragm; and is of opinion, further, that the statements of the French authors in regard to the increase of the muscular tissues in the left heart during pregnancy are, to say the least, somewhat overdrawn. Indeed, Fritsch, though in other passages he would seem to adhere partially to the view that there is normally some cardiac enlargement during pregnancy, expresses himself in this article as rather sceptical in reference to the existence of an eccentric hypertrophy of the muscular tissue of the left heart, and appears inclined to believe that a passive dilatation of the organ of a slight amount is sufficient to meet the necessities of the case.

But I shall leave him to express his views in his own words, translating as closely as I can: 'Now it is questionable if it is necessary to assume an eccentric hypertrophy, if new muscular fibres are actually formed. Though in general the hearts of lying-in women on post-mortem examination appear large, yet one would require to make accurate weighings and measurements of them. Furthermore, in consequence of the absence of fixed points in the measurement of the compressible substance, the thickness of which varies with the varying quantity of blood contained in the muscles, these direct measurements would be scarcely decisive. Still, one could seek for them in the shape of remains of the hypertrophy with the microscope. In fourteen sections of lying-in women who died at different stages of the lying-in period up to three months after delivery, I, however, found neither fatty degeneration nor pigment in the muscular tissues.'[1]

[1] *Loc. cit.* p. 377.

Again, in a more recent paper, supplementary to the one we have now been referring to, *Archiv für Gynäkologie*, Bd. x. S. 288, Fritsch expresses himself thus: 'In conclusion, be it observed that I can only admit as proof for a slight cardiac hypertrophy the estimation of the heart on section. To me the hearts of lying-in women appear on section constantly somewhat enlarged. But yet I must evidently withdraw this assertion, if more competent judges — pathological anatomists—contradict it.' To many points in these papers, which are of very great importance, I shall have to recur again and again, but I must leave them for the present.

Herman Löhlein,[1] one of the latest writers on this subject, has endeavoured to prove that the French authors, in demonstrating an hypertrophy of the left ventricle, are entirely mistaken, or at least have not at all proved their point. He quotes approvingly the statement of Gerhardt, 'that the measurements of Larcher and Ducrest fall within the limits of the anatomical statements of the average thickness established for the non-pregnant female,' and instances Peacock as authority. On turning to this author's statement, however, as found at page 5 of vol. iv. of Reynolds' *System of Medicine*, I find the greatest average thickness of the left ventricle in females given as 1·26 cent., whilst Ducrest assigns an average for the hearts of the pregnant females he examined of 1·5 cent., which bears out, to my mind, a distinctly appreciable difference in favour of the views of Ducrest and Larcher. The latter author, so far as I have seen, did not, unfortunately, proceed to exact measurements. Löhlein very properly, in my opinion, objects entirely to the possibility of attaining the definite results from percussion that Duroziez asserts he has obtained.

In attempting to settle the purely anatomical question, Löhlein objects to the statements of Larcher as being so

[1] *Zeitschrift für Geburtshülfe und Frauenkrankheiten*, S. 482. Stuttgart, 1876.

indefinite that they cannot be readily subjected to thorough criticism. But while not denying some value to Larcher's general statements in regard to the size of the heart, he maintains that without the data of the numbers and weights of the hearts he cannot regard such an important assertion as probable, whilst its discoverer holds it as superabundantly proved.

In opposition to the statements of Ducrest, Löhlein objects that the French author made no attempt at classifying the hearts he measured according to the cause of death and the time of its occurrence, which he thinks ought to have been done, inasmuch as thereby the condition of the muscular tissue of the heart is materially influenced. Among the diseases which produce such modifications he mentions eclampsia, chronic or acute affections of the respiratory and of the circulatory organs, and repeated and serious loss of blood.

Löhlein meets Blot's deductions from the increased weights of the hearts of pregnant women, by the results of an examination of the hearts of nine women that had died in the Gynæcological Clinique at Berlin during the previous year. These had met their death either from rupture of the uterus or from some acute cause that led to a termination of life within two days after delivery, and he finds the average weight of their hearts to be 247 grm.; whilst, according to Blot, the weight of the heart of the pregnant female is to that of the non-pregnant as 290·95 : 220·230 grm. He therefore maintains that if the cases in which the direct cause of death tended to cardiac hypertrophy—leaving the pregnancy out of account—were excluded as in his cases, the average weight of the hearts of pregnant females would be found not greater than the average weight of the hearts of non-pregnant females. He further supports this opinion by finding, in the case of six nephritic patients who had died

in eclampsic seizures, that the average weight of their hearts was 300 grm.; and states that if he had combined the weights of the hearts of those six with those of the nine cases mentioned above, his results would have agreed very closely with those given by Ducrest.

He then proceeds with great fairness, I think, to argue that authors, in maintaining the existence of an hypertrophy of the left heart, have clinically bestowed too exclusive attention to the cardiac dulness, which from the upward displacement of the heart in the course of the pregnancy is specially liable to mislead, and have not noticed whether the cardiac impulse was found to be intensified, the first sound at the apex specially loud, the second aortic sound accentuated, the radial pulse hard and difficult to compress, or whether the apex beat was thrown outwards or downwards from its normal situation, etc. He states that he submitted twenty patients to careful examination, with the view of ascertaining the correctness or incorrectness of these opinions, and formulates the results of these investigations as follows: 'Doubtless the absence of all the clinical symptoms by which we recognise the hypertrophy of the cardiac muscle is the rule at the end of the normal pregnancy; its occurrence, on the other hand, is the totally rare exception.' After expressing the opinion that a sort of teleological argument had had considerable influence with Larcher and the other supporters of the hypertrophy theory, viz., that such a condition being wanted to explain their beliefs in regard to certain symptoms, was accordingly found to exist, Löhlein comes to the following general conclusion: 'The proofs ever brought forward anew for Larcher's doctrine of the hypertrophy of the hearts of pregnant women are insufficient, anatomically as well as clinically. Our own observations of healthy women in advanced pregnancy and in the lying-in period have given us no support for this

assumption. It cannot, therefore, be laid down as an explanation of the increase of all the troubles that not infrequently come on in the case of women affected with disease of the heart during pregnancy, parturition, and the childbed period.'

It will thus be seen that the history of inquiry as conducted by French observers, who have bestowed much time and attention upon the subject, attacking the problem from various aspects, so as to test one method of observation by the results of others, in order thus, if possible, to eliminate the errors that might vitiate a single method, tends to establish almost beyond a doubt that there is a degree of hypertrophy of the heart as a physiological condition during the latter months of pregnancy. There seems also a very marked consensus of opinion among them that the gain in weight is chiefly, if not exclusively, confined to the left ventricle. From what has been stated, however, in this chapter, it cannot be doubted that the amount of this hypertrophy has been exaggerated. Still, notwithstanding the strong negative statements of Löhlein, and his specially incisive remarks regarding the usual absence of clinical evidence of cardiac hypertrophy during the latter months of pregnancy in the case of women with normally sized hearts, I cannot help feeling convinced that there is a certain amount of such hypertrophy. My reasons are the following:—

1*st.* I cannot conceive so many honest observers, as have examined such large numbers of the hearts of women dying during the childbed period, to have been completely mistaken in a matter of the kind, though they may have been so in regard to the amount of the increase of size. There is great liability to error in this direction, in consequence of the great difficulty involved in determining a reliable average for the normal heart.

2*d.* I think it can be shown that, though they have erred

in using tables that somewhat under-estimated the normal heart in respect both to weight and thickness, and consequently were led to make the difference appear greater than it should be, there still remains, even after such reduction is made, a substantial difference in favour of their views.

(*a.*) Thus, in regard to the average thickness of the left ventricle in pregnant and non-pregnant women—

Ducrest states the average thickness at the thickest part of the wall of the left ventricle in the puerperal woman at 1·5 cent.;

Bizot gives in the same cavity the average thickness for women generally as 1·00 cent.;

Peacock, again, makes the average thickness of the left ventricle in women generally, at base 1·102 cent., at apex 0·526, at mid-point 1·26.

It will thus appear that, granting that Bizot's averages are too low, and assuming Peacock's, as being the later, to be the more correct, there is still a margin of ·24 of a centimetre in favour of the truth of Ducrest's observations.

(*b.*) If, then, we take and compare the weights as given by Blot and by Peacock respectively, we find that—

Blot's average in the puerperal condition is 9 oz. 38 gr. troy.

Peacock gives the general average of healthy hearts in females that have died from twenty to fifty-five years of age as 8 oz. 15·5 gr. troy. In the case of those who died of acute diseases, the averages varied from 7 oz. 140 gr. troy to 9 oz. 55 gr. troy; whilst of those who died of chronic diseases, the averages varied from 6 oz. 182·5 gr. troy to 8 oz. 117 gr. troy.

No doubt puerperal deaths ought to be classed with the acute cases. But then it will be observed that, though we grant this demand, the average heart of the puerperal woman, according to Blot, is equal to the maximum of the

hearts in acute cases, according to Peacock, which certainly points towards the correctness of Blot's observations. Another remark has to be made in this connection, and it is this:

Since Peacock, in constructing his tables of the weight of the healthy hearts of females dying within the ages of twenty to fifty-five, must surely include a very large proportion of observations made on the hearts of women who have died in connection with child-bearing, as during the period of sexual activity the accidents attending pregnancy and parturition are unfortunately most fatal to female life, it is only reasonable to conclude that, by their addition to the gross weights of the healthy hearts of non-puerperal females, his average may have been unduly raised for acute cases. If this be true, Peacock's averages for acute cases being high ought rather to favour the doctrine of there being a certain degree of hypertrophy in the hearts of parturient females.

(*c.*) It is further to be noticed that, even on his own showing, Löhlein's average is 17 grm. over the highest average of the hearts of the non-pregnant females as given by Blot, which of itself indicates a slight increase in the average even in his nine cases. But, furthermore, we are not prepared to allow with him that the hearts of patients dying of eclampsia should be deducted, as renal disease in connection with fatal puerperal eclampsia is usually a very acute ailment, which could produce little change in the heart before death. And yet Löhlein's results, even if these are included, really confirm the views of Blot to a considerable amount.

If, therefore, we grant that the hypertrophy, though real, is somewhat less than the French authors believed it to be, we have, it appears to me, a sufficient answer to the criticism of Löhlein and others, that evidence of marked cardiac hypertrophy are absent, whilst we at the same time allow for such an amount of physiological change as shall explain

most of the phenomena that have been, as I believe, properly referred to cardiac hypertrophy during pregnancy.

(*d.*) On three occasions only, since I began this inquiry, have I had an opportunity to examine the healthy hearts of patients that died in childbed.

In reference to the first case, truth compels me to state that the left heart did not appear either to Dr. Wyllie or myself to be appreciably hypertrophied. But it was the heart of a very small woman, who died two days after delivery by craniotomy. We had only one opportunity of examining the heart hurriedly and by bad artificial light, our main object being to secure the pelvis, which was very much deformed. The second was the heart of a healthy young woman, who died of puerperal eclampsia a few hours after her first confinement, which occurred at the age of eighteen. In her case the normal hypertrophy of the left ventricle was demonstrably and indubitably present. The patient enjoyed the best of health till about thirty-six hours before death, so that there can be no valid objection founded upon the plea that the gradual effects of the nephritis had induced cardiac hypertrophy. The lesion in the kidneys was altogether acute, and every valve of the heart was normal. Dr. D. J. Hamilton, who performed the sectio for me, remarked at the time that the heart seemed enlarged and bulged very much to the left. On more accurate examination, he reported to me its condition as follows:—'M.'s heart was found to weigh 9½ oz. The left ventricle was decidedly hypertrophied. Its walls at the thickest part measured three-quarters of an inch. The right ventricle was only slightly hypertrophied.' The third case was that of a young healthy woman, aged twenty years, who died after her first confinement, which was slow, but terminated naturally, of an attack of acute non-septic peritonitis. In her case the heart was normal, but its left ventricular wall

measured three-quarters of an inch in thickness, and the whole heart weighed nine ounces. In this case, also, Dr. Hamilton performed the sectio and measured and weighed the heart. I am therefore satisfied that unbiassed anatomical experience supports very decidedly the French observers as against their German critics. My own first observation counts, it appears to me, for nothing, as it was imperfectly made. But both the second and third—particularly the second—afford evidence of an irrefragable nature in favour of cardiac hypertrophy during pregnancy. One such well-observed case is far more worthy of careful consideration than any amount of doubtful disputations, founded upon abstract principles.

3*d*. A certain amount of cardiac hypertrophy is not only rendered probable by the existence of greater work for the heart to perform during pregnancy, but has analogues in many similar changes that occur during pregnancy in other organs.

I have tested with some degree of care the limits of precision attainable by percussion in the latter part of pregnancy and in the lying-in period, and feel warranted in affirming that such an amount of accuracy as Duroziez affects is, in my judgment, unattainable, and that to attempt it can lead to no trustworthy or satisfactory results. With the view to determine, if possible, whether there is or is not such an amount of increased vascular tension during the latter months of pregnancy as the sphygmograph could detect and register, I have either taken, or had taken for me by my friend Dr. John Playfair, a very large number of pulse tracings. Part of these have been obtained by Marey's instrument, part by Mahomed's. The latter, having an arrangement intended to determine the amount of pressure with which the tracing is taken, and the amount of force necessary to arrest pulsation in the artery, is much more

exact than Marey's instrument. But, after all allowance is made for improvement in the instrument, there is reason to believe that the best applied sphygmograph gives only an extremely rough approximation to the real arterial tension.

The difficulties in the way of attaining accuracy are very great. Thus, for example, the resistance offered by the soft parts around the vessel is different in every arm. The spring of the instrument is liable to get weakened by use, and by variations in temperature. It is impossible for two different observers to say that each applies the instrument with exactly the same tightness in similar cases. Still, though it is impossible to look upon the expressions of pressure registered by Mahomed's sphygmograph as exactly representing the tension of the vessel in ounces, yet they may be with perfect fairness used relatively, as indicating whether a pulse is of high or of low tension. In this respect this instrument is superior for our purposes to any other I have yet seen.

The obstacles above mentioned prevent me from being able to draw so positive conclusions as I could have wished from my sphygmographic observations.

But, so far as I have been able to form an opinion, I believe they warrant us in asserting that the arterial tension in the latter months of pregnancy is unusually great; that it is somewhat diminished, but not greatly so, immediately after delivery, being still high during the lying-in period; and that it slowly returns to the normal during the latter. But there is not equal unanimity regarding the childbed period. Still the evidence afforded by the sphygmograph during that period satisfies me that the pulse tension is higher then also.

I may state here that the observations of Mahomed,[1]

[1] *Medico-Chirurgical Transactions*, London, vol. lvii. p. 223. 1874.

Meyburg,[1] Marey and Blot,[2] and others, agree with mine in regarding the sphygmographic curve of the pregnant female as one of high tension.

Thus during childbed I have usually been able to observe a well-proportioned and perpendicular up-stroke, or what is ordinarily called percussion wave, and have frequently found a peculiarly full and well-sustained tidal, or predicrotic wave. These conditions are held to indicate a pulse of considerable tension. Mahomed's sphygmograph has also shown, in the cases in which I have used it, that in women near the full time, or shortly after delivery, the best tracing is obtained by the employment of considerable pressure, usually 5 or 6 or even 7 ounces, and that to completely stop the artery a pressure of 13 to 20 ounces is needed.

In some cases—but these are the exception—the pressures necessary to obtain the best tracing, and to arrest the action of the lever, were quite as great, or even greater, after the delivery than before it. But this is not usual. The ordinary experience is to find the best working pressure about an ounce less after the delivery, and the obliterating pressure to fall along with it. Still, though the pressure after the delivery was usually less than immediately before it, in no case could it be called feeble. Even with its diminution it maintains a height fully greater than the average of that of healthy non-pregnant women. In this view I am in unison with Marey and Blot, and Mahomed, but at variance with Meyburg.

Many observations have been made by me in connection with this inquiry, but only a few tracings need be given. These will be found in the fifth chapter, when we make a general estimate of the conclusions arrived at in the work. It is so difficult to apply the sphygmograph so as to perform

[1] *Archiv für Gynäkologie*, Bd. xii. S. 114.
[2] *Bulletin de l'Académie de Médecine*, t. xxviii. p. 326. 1863.

well during a pain, that I have not thought those so obtained sufficiently reliable to warrant me in getting more than four of them printed.

The great tendency to various varicosities during pregnancy, indicating as it does exalted venous tension, cannot be overlooked, as substantiating the opinion that the circulation is conducted under abnormally high tension during this period. So also does the special liability to chest disturbances, and the difficulty with which these are recovered from during pregnancy. In short, it appears to me, from all we have seen under this heading, that the arterial tension during the latter months of pregnancy at least, is indisputably high, and is maintained in this condition by a left ventricle which is physiologically hypertrophied so as to be able to meet this end; or, to put it otherwise, hypertrophied because the gradually developed extra tension has to be supported by it.

It is plain that, as the amount of blood circulating within the mother's vessels during the latter months of pregnancy is increased beyond question, the same must be true of the amount circulating in the heart within a given period of time, since the heart is only a section of the general vascular system, unless it could be shown that the heart beats more quickly during the latter months of pregnancy than at other times. But the latter is not the fact; therefore it follows that at each contraction of the heart a larger amount than normal must be propelled from the ventricles, and at each diastole a larger amount must find its way to these cavities. And the same must be true of the auricles.

It follows, therefore, that all the chambers of the heart must be somewhat dilated during the latter months of pregnancy, though it would appear that the increase of muscular tissue is very greatly restricted to the left ventricle. There is therefore reason to believe that in the left ventricle, during pregnancy, we have as a physiological condition a certain

degree of increase in its capacity, associated with a variable amount of muscular hypertrophy in its walls; and in the other chambers we have the same slight dilatation, uncompensated by muscular hypertrophy. The application of this relationship under pathological conditions, however, will fall to be considered afterwards.

## CHAPTER III.

HISTORICAL CONSIDERATION AND CRITICISM OF THE EFFORTS WHICH HAVE HITHERTO BEEN MADE TO DISCOVER THE MUTUAL BEARINGS OF CARDIAC DISEASE, AND OF PREGNANCY, PARTURITION, AND CHILDBED UPON ONE ANOTHER.

FOREMOST among observers in this work must be placed Hecker. In 1860 he recorded two fatal cases of heart disease that occurred in the Midwifery Institution at Munich, and gives a short account of their post mortem examinations.

The one was a case of mitral stenosis of a moderate degree proving suddenly fatal from pulmonary œdema, and the other presented extreme mitral disease, with dilatation of the right ventricle. Tolerably lengthy abstracts of both are given in my collection of cases (see Cases X. and XIX.).

Hecker makes the following comments upon those cases: 'The danger to women who suffer from valvular lesions, which is determined by pregnancy and parturition, appears to me capable of developing itself specially in two directions: In the first place, during the latter months of the pregnancy, such a narrowing of the thoracic space is produced, that the lungs, already embarrassed in their functions by the cardiac lesion, become, at times quite acutely, functionally incapable through a serous effusion, and life is thereby brought to a standstill; or the heart is so used up by the exertions of the labour, that its already disturbed mechanism is brought

completely to a standstill, and thereby an end is made of life.'[1]

It is to be noticed in this connection that Hecker's statement about the narrowing of the thoracic space by pressure of the abdominal tumour is, as far as regards uncomplicated pregnancy, proved by scientific observation to be essentially incorrect. In a paper by Dohrn[2] upon this subject, which contains an elaborate series of cyrtometric observations on the chest made the week before and the week after parturition, he recounts the spirometric observations of Küchenmeister, of Fabius, and especially of Wintrich, which prove that the vital capacity of the lungs in the human female is constant throughout pregnancy; refers also to the observations of Gerhardt in reference to the changes in the height of the diaphragm; and is led to the conclusion that, while the perpendicular axis of the thoracic cavity is diminished during the latter months of pregnancy, and likewise its antero-posterior axis at the lower part of the cavity, the transverse diameter is at the same time much increased, so that as a whole the cavity is not at all, or very triflingly, diminished, except some pathological condition is present, such as hydramnios, ascites, etc. In a more recent paper (*Monatsschrift für Geburtskunde*, Bd. xxviii. S. 457), Dohrn records and tabulates spirometric observations made upon 100 patients during the last week of pregnancy, and again on their dismissal on the 12th or 14th day after delivery. Of these persons he says 60 per cent. exhibited an increase of the vital capacity of the lung at the date of their dismissal, whilst 14 per cent. showed no change, and 26 per cent. a diminution.

But these results, it appears to me, can hardly be said to be very convincing, as the proportion of cases exhibiting

[1] Hecker and Buhl, *Klinik der Geburtskunde*, Leipsig 1861, S. 173.
[2] *Monatsschrift für Geburtskunde*, Bd. xxiv. S. 414.

increase is not sufficiently great to shake our belief in the careful observations of Küchenmeister, Fabius, and Wintrich, already referred to. At any rate, we are warranted still in maintaining that the diminution in vital capacity of the lungs conditioned by normal pregnancy is very immaterial indeed.

Gusserow[1] also more lately applies these facts to help in the steadying of our views in regard to premature labour, when employed with the intention to benefit acute intra-thoracic inflammation. He holds that if hydramnios, ascites, etc., be present, then you may perform premature labour to facilitate the action of the chest; but that if it is simply to get rid of threatened suffocation from causes entirely restricted to the chest, such as acute pneumonia, then the certain harm which will result from the performance of the operation is far too great to warrant the extremely nugatory, if indeed not entirely illusory, good that the operation promises. His statistics of recorded cases certainly, so far as they go, support this view. These references are perhaps a little out of place here, but I mean to utilize them further on, and they help in keeping the real condition of matters clearly before us. It seems to me that they ought to be of material use in assisting us to weigh the propriety of inducing premature labour in connection with heart disease.

We next come to Spiegelberg's paper upon the relation of chronic heart disease to the pregnant and parturient conditions, which was published in 1871, and which is the most valuable contribution I know of on this subject.[2]

Of Hecker's remarks above quoted, Spiegelberg observes: 'The above propositions rather describe a correctly observed fact than explain it; also the cases in which the danger developed during the pregnancy is terminated by the labour;

[1] *Monatsschrift für Geburtskunde*, Bd. xxxii. S. 88.
[2] *Archiv für Gynäkologie*, Bd. ii. S. 236.

and those in which the danger only arises subsequently to the latter, are not elucidated by them.' Spiegelberg believes he has found the solution of the difficulty in the existence of suddenly diminished arterial pressure and increased venous pressure. He writes as follows: 'These unfortunate accidents are rather conditioned by this, that the degree of the compensation of the cardiac lesion being under ordinary circumstances enough to make the situation tolerable, is no longer sufficient for the altered, and especially for the suddenly altered, relations of pressure; in this respect, however, the compression of the thoracic space in pregnant women—in the absence of undue abdominal distension—being quite immaterial, if generally present, is not of the first importance, but the altered pressure, under which the heart acts during the pregnancy in comparison with the condition after the labour.'

Spiegelberg then goes on to argue that the introduction of the placental circulation imposes upon the heart the task of moving a greater amount of blood under a greater amount of tension, so far as concerns the general circulation, than in the non-pregnant state, and that this is rendered possible by the hypertrophy of the left ventricle, as demonstrated by the French authors I have already referred to. But he proceeds to maintain that, 'with the expulsion of the child and its membranes, these relations (of pressure) are suddenly changed, the placental circulation is eliminated, the numerous and wide utero-placental vessels are shut, the pressure in the aorta sinks; hereupon the pressure in the venous circulation increases; this results still further for the reason that, whilst the channels are in part (in the uterus) become impassable, the mass of blood is still relatively increased; for the hæmorrhage during the labour in normal cases is not so copious as to reduce the previously existing increased mass (of blood) immediately to the non-puerperal standard. In consequence

of the increased pressure arising in this manner in the veins, to which is added the greater mobility of the diaphragm, acting in the same direction, brought about by the emptying of the uterus, the blood after delivery flows more copiously into the thoracic cavity and to the lungs; the work for the right heart increases.

'It is now readily intelligible that these changes of pressure, quickly overcome and equalized by a sound heart, may act so as to bring into perturbation a mechanism disturbed by a valvular lesion. Where and how far they do this will essentially depend upon the situation of the lesion, and upon whether it is a compensated or a still relatively acute one, in which the compensation of the disturbance has not yet been established. At the same time, collateral circumstances, such as the degree of distension of the abdomen in the pregnancy, likewise that of the mobility of the diaphragm, the amount of hæmorrhage at and immediately after delivery, may still influence and modify the phenomena.' Setting out from these premises, Spiegelberg proceeds to maintain (he restricts his attention to lesions of the left side of the heart only) that in the case of aortic incompetency the serious symptoms of cardiac disturbance as a rule appear during the pregnancy, and usually about its second half, in consequence of the compensation being insufficient to meet the exalted tension in the arterial region. These functional disturbances, especially attacks of dyspnœa and extreme cardiac irregularity, are apt to lead to premature interruption of the pregnancy, attain the most threatening height during the labour, and if it is once completed, speedily remit, and often disappear entirely, as the aortic pressure now falls, and that in the veins suddenly increases.

On the other hand, he asserts that when the disturbances originate in insufficiency of the mitral valve and in stenosis of it, which is mostly coincident with incompetency, disten-

sion of the abdomen, the degree of mobility of the diaphragm, the condition of the lungs, and especially the presence or absence of compensation by hypertrophy, here of the right ventricle, are of much more importance than in those lesions that affect the aortic valves.

Such conditions, according to our author, present three possibilities, which we may briefly synopsize thus:—

*1st.* The lesion may occasion no disquieting symptoms, there being few if any complications, and the compensation being good.

*2d.* From over-distension of the pulmonary channels, symptoms of disturbed pulmonic circulation may gradually appear during the pregnancy, especially within the latter months, or suddenly, if from any cause the diaphragm is persistently kept in the position of expiration. Even under such conditions of mechanically obstructed pulmonary circulation, competency may be established by hypertrophy of the right ventricle, and the symptoms may subside rapidly after labour, or an aggravation of the symptoms, with dyspnœa, pulmonary catarrh, pulmonary œdema, general œdema, etc. etc. may result. All the distressing and dangerous symptoms attain alarming proportions during the labour, which is usually *premature*. After the labour they may remit or get worse.

*3d.* The symptoms of congestion may remain undeveloped during the pregnancy in mitral lesions of recent date, and alarming symptoms be postponed till after the confinement, due, according to Spiegelberg, to the fact that the right side of the heart is over-distended with blood under excessive pressure, whilst the hypertrophied left heart aggravates matters by pumping back the blood into the lungs.

Spiegelberg supports his views by the records of four cases, two of mitral and two of aortic lesions. I give abridged translations of these among my cases, and therefore abstain

from making any further observations upon them at present, except simply to say that most of his statements as to the results of the cardiac lesions are fully borne out by the facts found in the cases I have been able to collect.

Spiegelberg's paper was published about the middle of 1871; and in the March number of 1872 of *L'Union Médicale* is a lecture by M. Michel Peter, delivered at the Hôpital de la Charité, the 27th of November 1871, beginning in the following terms: 'I wish to speak to-day of facts which the authors of treatises on diseases of the heart have left in the shade, and which authors of treatises of obstetrics seem to me to have entirely forgotten: those facts are the pulmonary accidents to which pregnancy exposes women affected with disease of the heart.

'We have seen that pulmonary accidents are necessary at a certain period of cardiac affections. Well, that pregnancy renders those accidents both more abrupt, and more formidable on account of this abruptness even, is self-evident if only we reflect a little upon the condition of the circulation in this transitory physiological state. Only as it has not as yet been spoken of, however simple it may be, it is still necessary that I should speak to you about it; and you will presently see that we treat here on a point of practice of the highest interest.'

The author then proceeds to record two cases at length, in which organic heart disease gave rise to extremely threatening pulmonary symptoms, in the shape of suffocative congestion and œdema, about the sixth month of pregnancy. He merely refers to, but does not record at length, other two similar cases. There comes out a wonderful similarity between these cases—in regard to time of onset and general symptomatology. In the two first cases our author frankly confesses that he mistook completely the causation, as he overlooked the heart disease in both of

them, diagnosing simply pulmonary engorgement in the first case, and subacute phthisis in the second. Warned by his previous mistakes, he readily detected the chain of causation in the latter two cases. His treatment of the first case strikes one as specially heroic,—antimony and bloodletting by cupping and venesection,—yet it is so far justified by the fact that it succeeded in resuscitating the patient after she was believed to be actually in the jaws of death. The symptoms of suffocative catarrh and of extreme cardiac irregularity supervened in the first two cases during the fifth month, in the second two later on. Peter's cases *seem* (for the statement as to physical signs are painfully general) all to have been of mitral insufficiency, complicated in one instance with mitral contraction. He argues that there is double reason to expect the heart, when pre-existently diseased, to give rise to such serious complications during pregnancy,—first, because the total amount of blood is augmented in the pregnant female; second, because her left ventricle is hypertrophied. In all his cases, except one, he found the lesion restricted almost entirely to the smaller circulation. The systemic circulation was carried on tolerably well, the right heart showed no unhealthy symptoms, and the evil effects of the pregnancy expressed themselves chiefly to his mind as a consequence of the regurgitation, made more injurious during the pregnancy by the increased mass of blood and the increased power of the left ventricle. It only comes into play when the mass of blood needed to support the child, as well as the utero-placental circulation, become of sufficient extent to be of real importance, *i.e.*, about the fifth or sixth month. He recommends as a practical result,—first, the greatest care as to movements and exposure, supposing a patient with heart disease becomes pregnant; second, the avoidance of pregnancy in future; and third, the avoidance of lactation in all cases, inasmuch as the latter

process keeps up an abnormal amount of strain upon the heart, and prevents the left ventricle from returning to its ordinary size, and the consequent restoration of normal tension to the circulation.

This French author does not hold his subject so tightly in hand as his German confrères. But still his paper is a valuable and practical contribution to this difficult subject. I am inclined to believe that his first case was one of mitral obstruction, as well as of insufficiency, as one seldom hears a rasping mitral murmur from insufficiency alone; and though the author states that it occurred with the first 'time' of the heart, he does not define very accurately its relations to the first sound. At any rate, as we shall abundantly prove further on, symptoms of the kind that occurred in M. Peter's cases are very common in combination with stenosis of the mitral orifice.

Lebert contributes a very valuable article on this subject in Bd. iii. S. 38, *Archiv für Gynäkologie.* Its scope is more general than that of any of those I have as yet referred to. He agrees with Spiegelberg in regard to the important bearing which disturbed relationship of pressure has on cases of heart disease during pregnancy and parturition. But, as it appears to me with much wisdom, he insists that the problem has bearings wider than these, involving not only the alterations in the valves but also in the motor powers—the muscular tissue of the heart itself—and changes in the entire vascular system of the greater and lesser circulations. Lebert makes the following trenchant statement, which I cannot avoid translating *in extenso:* 'Before I communicate my observations, I observe that chronic endocarditis, with valvular lesions from the second childhood onwards, occurs not seldom in girls, and undoubtedly more frequently among them than among boys of the same age. Leaving out of account those more rare cases in which an acute articular

rheumatism or chorea are demonstrable as ætiological forces, valvular endocarditis developes itself for the most part stealthily, without any obvious cause. I have, indeed, seen it occasionally exist in girls for years with no observable disturbance; usually, however, shortness of breath appears sooner or later, and the subjective troubles make gradual advances. Puberty of itself has little influence upon the cause of the disease, yet in not a very few cases it is so far favourable, as menstruation at this time is habitually profuse, and thereby, for a time, a check is given to venous engorgements. However, one sees also, it is true, before and soon after puberty, some cases of this sort run a fatal course, and I have published several of such cases on different occasions. We have mostly to deal with diseases of the left orifices, with preponderating frequency of the mitral, more seldom with the tricuspid opening. Only seldom are these congenital lesions of the right heart. . . . If the compensation begins to be imperfect in young girls,—if the subjective disturbances have gradually increased,—then I *dissuade* from marriage, if I am consulted in the matter, because it is precisely those cases upon which pregnancy and parturition may operate very unfavourably. Altogether the fact is worthy of consideration, that not simply during the pregnancy the disturbances of compensation may come on much more readily, much more quickly, and much more intensely than apart from it; but that, also, truly the valvular endocarditis which developes itself during the second half of the pregnancy shares with puerperal endocarditis the tendency to decomposition and consecutive embolisms, and therefore is disproportionately more dangerous than plastic endocarditis, apart from pregnancy and the lying-in period, which advances more stealthily, and whose progress is usually less grave.'

Lebert accompanies and illustrates these valuable remarks

with the records of three cases of the very highest interest, and which are most elaborately reported. One only is logically connected with the subject of this paper, and it is given abridged in my selection of cases (see Case XXXI.).

Of the other two, the one is a case of severe puerperal fever, with breathlessness, ulcerative endocarditis, and embolism of the pulmonary artery; and the other of puerperal fever following abortion at the sixth month, and accompanied with embolism of the pulmonary artery, gangrene of the lower lobe of right lung, etc.

These cases are both extremely important, as bearing unmistakeable evidence of the manner in which pregnancy modifies the whole blood vascular system, so as to render it specially liable to become seriously affected in consequence of diseased conditions arising in connection with pregnancy.

But as in neither of them is there evidence from the records that the patients had been, previously to the delivery, the subjects of heart disease, and as I started with the determination to exclude anything approaching to a minute discussion of ulcerative endocarditis, I have forborne from including these among my cases.

The third, however, being that of a patient who had suffered from cardiac lesion for some months before becoming pregnant, is fairly entitled to come within my classification.

I have also to state here, that in the *Archiv für Gynäk.*, Bd. iv. S. 15, 1872, Ahlfeld records briefly two interesting cases of pregnancy complicated with chronic heart disease, abstracts of which are given further on. (See Cases XXI. and XXX.)

It is to Fritsch's recent papers in the *Archiv für Gynäkologie*, however, that we have to turn for the most recent and able efforts at the solution of this difficult problem.

This author denies the correctness of Spiegelberg's view

relative to diminished aortic tension. But he allows that the sudden accidents that arise from heart disease during childbed are due to the defective cardiac compensation being unable to meet the conditions introduced by the suddenly altered relative pressure, although he disagrees with Spiegelberg as to the manner in which it acts.

Fritsch predicates a slight degree of cardiac dilatation to meet the requirements of the greater amount of blood circulating in the heart during pregnancy at each beat. This dilatation, he argues, must involve both sides of the heart. The healthy heart is able to meet these extra requirements, but the diseased organ suffers from imperfect power of accommodation to these demands upon it, and as the pregnancy advances the effects of this defective power of accommodation increase. There is also reason to believe that the valvular lesions are apt to be altered for the worse during pregnancy. This is evidenced by the greater readiness with which pregnant women, whose hearts are unsound, suffer from puerperal endocarditis. If we estimate the effects of labour pains upon the circulation under normal circumstances, we find that during the pains, especially during the bearing-down pains, the pressure on it is increased, owing, first, to the condition of forced expiration in which the thorax is placed, and, second, to the greater obstruction presented to the passage of blood through the uterus.

The sudden deaths occurring during labour in cases of mitral disease, Fritsch would refer to the paralysing effect of the sudden flow of a strong stream of blood into the right heart, under the influence of the above causes that heighten vascular tension. It would appear that Panum has shown by experiment that a sudden jet of blood projected into the heart does paralyse it. The contractions of the uterus, and consequent lessening of the peripheral circulatory area, cannot of itself, as Spiegelberg believes, says our author,

diminish external pressure, but increase it. The real state of matters is probably as follows. The great amount of blood squeezed out of the uterus finds easy accommodation in the enormous abdominal venous plexuses, and in the expansible capillary plexuses of the peritonæum and broad ligaments. This it readily does, as the abdominal pressure after labour, though it need not be negative, is nevertheless much diminished. Provided the heart is healthy, all is soon restored to order. But suppose the heart is too large in connection with disease involving it, too little blood flows into it, it contracts strikingly irregularly, too little reaches the smaller circulation, and the blood throughout the body is defectively oxygenated. Vital force sinks in consequence, the ill-nourished cardiac muscle loses its force, and thus is established a vicious circle of the most dangerous kind.

On this depression of vitality fatal post-partum hæmorrhage and other clinical symptoms may supervene. Thus the emptying of the uterus causes dyspnœa,—the patient turns pale or cyanotic,—faints, or the cerebral anæmia may be sufficient to give rise to convulsions. The pulse is weak, small, and arrhythmic, and the patient dies from what, for lack of a better name, we call cardiac paralysis.

The risk manifestly must be greater in large hearts, yet well-compensated hearts may overcome the period of violence and pressure from the labours; and in anæmic and weakened persons possibly during the pregnancy the deterioration is so important that the labour is either the end or the beginning of the end.

Clinical experience leads Fritsch to believe that serious results are most likely to occur in cases of advanced mitral stenosis. It will be noticed as of considerable importance for our consideration of the question in its practical bearings, that the cases of mitral insufficiency that are complicated with stenosis are those which Spiegelberg has found most

fatal, although he differs from Fritsch *toto cælo* respecting the theoretical exposition of the chain of causation.

At page 306, Bd. ix., *Archiv für Gynäkologie*, 1876, Lahs of Marburg publishes an ill-tempered criticism of Fritsch's first paper, in which he angrily denies most of the positions taken up by Fritsch, and lays claim to the discovery of a mode of experiment, to prove that the introduction of a series of collateral tubes between an entrance and exit pipe does not diminish but increases the velocity of passage of fluid through the exit pipe, when constant pressure of fluid is maintained at the entrance pipe. This experiment he had not previously published, though it directs attention to an identical but more complete one by Dr. A. W. Volkmann, published twenty-five years ago. As this experiment is of great importance in helping us towards the solution of the problem as to whether the aortic tension is suddenly greatly decreased or not immediately after delivery, I will explain it presently, along with Volkmann's own observations. I shall only remark further in this reference, that Lahs believes the capacity of the thoracic cavity invariable, and consequently denies the possibility of either a sudden flow of blood to the heart, or of any marked deficiency of such flow, so long as the patient maintains the horizontal position, owing to the elastic force of the lungs being constant in quantity, and in reference to abdominal pressure being negative.

Fritsch, at S. 270, Bd. x., *Archiv für Gynäkologie*, publishes a supplement to his first paper, containing a rejoinder to Lahs's remarks, and, what is much more to our purpose, the record of a valuable set of cases of heart disease in connection with pregnancy and parturition, along with some extremely learned and interesting observations upon the subject. Abstracts of these cases are given by me further on. Besides objecting to the grotesque accusation of unfairness on the part of Lahs, and at the same time refusing

unreserved adhesion to the general application of Volkmann's experiment to the animal economy, he argues that the heart, from clinical observation, has unquestionably more work to perform during pregnancy than apart from that condition, and that the pains—more especially the down-bearing pains—aggravate the difficulties. He controverts Lahs's assertion that the whole lumen of the collective vessels flowing in the thorax is an unchangeable amount in relation always to the height of the elastic pressure of the lungs. He argues that if respiration alone regulated the heart's action by its aspirative power, then the latter must act synchronously with the former. But every one knows that such is not the case. He maintains that while the respiration regulates and controls the circulation, the heart's force maintains it. If any one of the important regulative forces of the circulation fails,—as, for example, the intra-abdominal pressure,—then defective blood supply to the heart and slowing of the circulation must be set up, and the blood must accumulate in the vena cava inferior and in the abdomen. He also explains certain hydro-dynamic experiments he had undertaken, which led him to the following conclusion,—that by the elongation of the channel of the stream the amount of fluid discharged must diminish, or the driving force must be increased in order to obtain an equal amount of fluid discharge; alternately widening and narrowing, especially in the additional portions of tube, had a retarding influence. He believes that the pregnant uterus would have a similar influence.

I have referred incidentally to a certain hydro-dynamic experiment of Volkmann's, which bears very importantly upon our views respecting the correctness or incorrectness of Spiegelberg's assumption, that we have sudden decrease of arterial pressure immediately after labour, and therefore I am under the necessity of making a some-

what fuller reference to it in this place. For the complete account, see *Hämodynamik*, S. 78, Leipsig, 1850. The experiment is briefly thus:—With the view of ascertaining the effects of the subdividing and subsequent reunion of the vessels in the animal economy, when conveying the blood from the heart to the tissues and back to the heart again, Volkmann constructed two apparatuses, a lesser and a greater, on the same model. In the former, simply one branching and reunion took place between the entrance and exit pipes, so that the fluid was made to move merely a part of the way in two streams. In the larger apparatus he introduced a second bifurcation, one upon each of the two primary branches, so that the fluid, in passing from the entrance tube to the outlet tube, became first subdivided into two streams, then into four, these again being collected, first into two, and subsequently into one, and afterwards discharged. The tubes were of the same diameter throughout, and the apparatuses were perfectly symmetrical in regard to their parts, and situated in one plane.

By calculation, the surface of adhesion between fluid and tubes in the smaller was to that in the larger as 4 : 10. The length of channel traversed by the fluid in the smaller was to that in the larger as 3 : 5.

On trial of these apparatuses with an equal amount of fluid pressure at the entrance tube of each, Volkmann found that the larger apparatus did not present more resistance than the smaller to the passage of fluid, and makes the following interesting comment on his experiment:—

'From this (experiment) the astonishing result is attained, that the larger apparatus opposes not more hindrance to the movement of the fluid than the smaller. The entire excess of retarding power, which it incontestably contains, is equilibriated by a favourable disposition of the collateral tubes, and rendered inoperative.

'If, now, we consider the two tubular apparatuses as the vascular systems of two animals of unequal size, and the height of fall as the power of the heart, there results that the same heart power may be sufficient to force the blood through the body of a large animal as a small one, provided only the collateral vessels are adapted to facilitate the flowing off of the blood. In reality, the static force of the heart in warm-blooded animals of the most different sizes appears to be subject to only slight variations,—a fact we shall recur to further on. Increase of blood channels, therefore, by the introduction of collateral vessels, can, under circumstances, set aside the disadvantage which increase of the surface of adhesion would present to the movement of the blood. What are these circumstances? We as yet do not know them, and it is questionable if even the higher mathematics would be in a position to explain them. Possibly it is that general forward movement is produced in the current, and the resistance arising out of the enlarged surface of cohesion is either entirely, or at least partially, annulled if the branching of the tubes is associated with enlargement of the channel of the current.'

It will be seen from this experiment that the sinking of aortic pressure immediately after labour, from the removal of the placental circulation, is not so self-evident an assumption as its learned author would have us to believe. At the same time, as Fritsch points out, such an experiment cannot be taken for our purposes in its entirety. We have not to deal in the living economy with tubes of equal calibre and rigid walls, but of varying size and with compressible walls, subjected to ever-changing amounts of pressure. Nervous influences and chemical changes constantly impress their peculiar effects upon the circulation in the living economy, and yet it is impossible to correctly imitate their influence by any arrangements in a hydro-dynamic experiment.

With a very brief reference to Löhlein's views on this head, I must close the historical part of this chapter.

This author takes considerable pains to point out how antagonistic to one another are the views advocated on this subject by Spiegelberg and by Fritsch respectively.

Löhlein grants the possibility of a slight increase of the work done by the ventricle on account of the increased quantity of blood circulating during the latter months; but he contends that, except the compensation in the heart is totally destroyed, the organ is able to accommodate itself to this increase, it is so slight in its amount and so slow in its onset.

Our author believes, further, that the placental circulation is capable of giving little obstruction to the general circulation. The slight increase in tension of the circulation is not capable, according to him, of producing serious disturbances during pregnancy, but it may co-operate injuriously along with other evil influences, such as compression of the chest, intercurrent bronchitis, pneumonia, pleurisy, etc., in aggravating the evil effects of a slightly dilated right ventricle.

Therefore, during pregnancy, Löhlein holds that the possible increase in arterial pressure must act within extremely narrow limits in disturbing the circulation. As to labour, *à priori*, he holds that if sudden disturbances of pressure were the main danger, most deaths from heart disease in relation to pregnancy ought to occur during delivery, which experience tells us is not the case.

Our author himself has treated fifteen cases of labour complicated with heart disease, and only one of the patients died during delivery, the cause of death being pulmonary œdema. This case was complicated, however, by recent pneumonia of the right lung, and there was also a pleuritic effusion on the same side to the amount of ten ounces; the right ventricle of the heart was evidently dilated.

Spiegelberg, he argues, has not proved his point as to the sinking of arterial pressure after delivery; and furthermore, physiological authors who have investigated such matters agree in stating that to contract a portion of the arterial system is to increase the tension in the rest of it. He thinks Spiegelberg's view, that danger in relation to delivery is to be dreaded on account of too copious amount of blood flowing into the heart, is well founded, and consequently declines to accept Fritsch's notion, that sudden death results from the paralysing influence of a sudden flow of blood into an empty heart, and the consequent irregular and imperfect action of the organ.

The greatly distended hyperæmic condition of the lungs in such cases he does not regard, with Fritsch, as a terminal symptom, but as a consequence of the imperfect compensation in the pulmonary circuit.

He records one case from W. B. Lublinski, Berlin, 1873, which he conceives confirms his views. It is one of sudden death on the eighth day from pulmonary œdema, in connection with mitral disease of long standing. It is interesting to notice, as I shall abundantly illustrate afterwards by examples, that this case led to premature labour, along with symptoms of serious disturbance of the respiration as well as of the circulation, even although the labour was not at all severe in itself. The delivery, as we shall prove further on is usual under such circumstances, was followed by a temporary improvement for a day or two, which ultimately, however, gave place to an aggravation of all the severe symptoms. It is difficult to make out whether this was a case of stenosis or of dilatation of the mitral, or of both, for it is very unsatisfactorily reported. But, so far as I can judge, there must have been a degree of stenosis present.

The gist of Löhlein's opinions on the influence of heart

disease upon pregnancy and parturition is given in the following sentences:—

'Of the alterations, then, that stand in direct relation with pregnancy and childbed, there remain, strictly taken, only two to which we grant, according to the present position of our science, a definite influence upon the already otherwise disturbed relations of circulation:

'1. The compression of the lungs by the upward distended diaphragm during pregnancy, especially in connection with greatly distended abdomen;

'2. The increased flow of blood to the pulmonary channels after delivery. To this has to be added the great variation in pressure during the activity of the pains.

'There is, however, also a further force introduced in addition to these, which by authors hitherto has either not been pointed out, or if so, only indicated in a passing observation,—a force which is equally able to greatly increase the existing mechanical obstructions and make their compensation impossible, as also to call forth completely new, sudden accidents, viz. the tendency to the recurrence of the inflammatory processes on the already chronically diseased portion of the vascular apparatus, which exists in pregnancy equally as well as in the lying-in period.'

The rest of the paper is chiefly devoted to the records of cases of chronic heart disease, with more or less serious acute exacerbation in connection with pregnancy and childbed, and it concludes with a few remarks on treatment.

Löhlein seems to me to place much too great importance upon the influence of upward pressure of the pregnant uterus upon the chest, and also to over-estimate the aspiratory effect of the lungs after delivery. The earlier paper of Dohrn, referred to at page 25, can scarcely be held to support him in either view; whilst his later contribution to the same subject, *Monat. für Geburtskunde,* Bd. xxviii. S. 457, at most

proves merely that in a majority of cases the vital capacity of the lungs is greater 12 to 14 days after delivery than in the latter weeks of pregnancy. It is nevertheless on Dohrn's observations that Löhlein chiefly supports his two main ideas in regard to the origin of disturbance during pregnancy and parturition respectively from cardiac disease. Therefore I regard his opinions on these points as needing further substantiation. His reference to the disastrous effects of the derangement in pressure during the activity of the pains seems to me abundantly proved by clinical experience.

That there is much truth in his third statement I am quite prepared to admit. But on this point I think he claims too much originality, for Lebert distinctly draws attention to it as one of the dangers to which a patient with chronic heart disease is liable, and it is inferentially deducible from the writings of almost every one who has directed attention to the subject.

I have thus traced as faithfully as possible the march of research upon these important conditions.

It will be seen that all the authors referred to are agreed that pregnancy is likely to introduce serious complications into the condition of a patient who suffers from chronic heart disease, except the lesion is of not very recent origin, and is well compensated, when there is a considerable probability that all may go well during the pregnancy, delivery, and childbed period.

It also appears that the symptoms of evil omen arise chiefly in the region of the lesser circulation, present themselves in the form of extreme dyspnœa, suffocative bronchial catarrh, œdema of the lungs, etc., are exceedingly apt to be complicated by the spontaneous onset of premature labour, and frequently end in death either during delivery or the childbed period.

There is likewise manifest agreement in the belief that

those urgent symptoms seldom occur till the first half of the pregnancy is past, and that they increase as the period of utero-gestation advances, provided premature labour does not come on; but that it by no means follows that they will either abate or disappear when the labour is past, although they are likely to attain a special intensity during the process of delivery, more especially during the second stage.

It will also be noticed that cardiac lesions of recent date are shown to be peculiarly dangerous, partly because, owing to the imperfect compensation attending them, they are apt to give rise to dangerous weakness of the right ventricle during pregnancy, and more especially during delivery; and partly because they are more apt to be associated with acute inflammatory changes in the endocardium, including ulcerative endocarditis. These conditions may appear either during the pregnancy or after delivery, even though the urgent symptoms during labour may have been recovered from.

It will be observed that Lebert believes that pregnancy and parturition are very liable to operate injuriously, not only upon the valves of the heart, but upon its muscular tissue, and upon the general vascular system. This view appears to be worthy of the most careful consideration, and seems to be supported by many clinical and pathological facts in relation to pregnancy, parturition, and the lying-in period.

As to the precise manner in which lesions of the heart lead to dangerous consequences in connection with labour, Spiegelberg has thrown out the ingenious theory that it is owing to suddenly diminished aortic pressure at the moment of delivery. The removal from the systemic circuit of the placental circulation, and the passage of the blood contained in the uterus before its final contraction into the venous circulation, according to him, have the effect of

lessening suddenly the arterial tension and increasing the venous, more especially in the right side of the heart. If, then, the heart is already defective, and its compensation inclining to give way, this final assault is apt to so completely perturb its action as to lead to sudden death.

Fritsch, on the other hand, believes that the danger under such circumstances is apt to arise rather from the dilated right heart being left too empty of blood, the large parametric venous plexuses, the other abdominal veins, etc., affording abundance of space to accommodate a great quantity of blood, especially after delivery, and when the abdominal pressure is not so great as it ought to be.

This condition of matters is apt, according to him, to lead to a sudden gush of blood from the abdomen into the empty right side of the heart, and to exert there a paralysing effect upon the organ, explained, as he believes, by Panum's experiment, which shows that a sudden gush of blood into the empty heart does paralyse that organ. The right heart then begins to contract upon the too small quantity of blood contained in it, its action is weak and irregular, too little blood gets to the pulmonary circulation, the blood is consequently imperfectly aërated. This, again, leads to imperfect cerebral nutrition, and so a vicious circle is established which ends in death.

Löhlein, again, denies both these views, and refers those accidents in general to imperfect cardiac compensation, aggravated by compression of the chest caused by the distended abdomen during pregnancy, and after delivery by excessive blood-supply to the lungs occasioned by the aspiratory effect of the over-distended thorax. In certain other cases, and more frequently, he thinks, than is usually suspected, he believes that acute inflammation of the diseased valves may lead to serious aggravation of symptoms both during pregnancy and childbed.

In Chapter V., when summing up the general results aimed at after full consideration of the cases of cardiac lesion contained in this contribution, there will occur another and more favourable opportunity to revert to the somewhat contradictory views entertained by these distinguished authors. At present my remarks must be very brief.

It may, however, be permitted at the present stage of our inquiry, to state that the evidence on which Spiegelberg predicates his sudden and great decrease in aortic tension is to my mind unsatisfactory, although it is an extremely beautiful and fascinating mode of explaining many of the difficulties connected with this subject. Indeed, there seems no well-established fact to support it, except that in cases of aortic insufficiency the danger usually disappears with the termination of the second stage. But it does not take any great amount of acuteness to discover that in this case it may not be the *diminution of arterial tension*, but the *absence of the extra tension* associated with the down-bearing effort, which removes the risks; and judging from the cases recorded by me, it is not even certain that these insufficiency cases possess the immunity from severe symptoms after delivery which Spiegelberg claims for them.

If, however, I refuse unreserved adhesion to Spiegelberg's special views, I see less reason to accept the explanation offered by Fritsch except in very rare cases of extraordinarily negative abdominal pressure. This theory, further, could only apply to accidents occurring after the conclusion of the second stage of labour. It appears to me that the true explanation of death during delivery in these cases, is rather to be sought for in the state of extreme exhaustion to which the enfeebled heart is reduced during the second stage than in either of these explanations.

That part of Löhlein's contribution to the literature of this subject is by far the most valuable, in which he deals

with the likelihood of acute exacerbation of the disease in the endocardium and its probable results.

We have to bear in mind, as shown in Chapter II., that there is evidence that cannot be contradicted, made up partly by the existence of an enlarged left ventricle, partly by the tendency to varicosity of the limbs, and partly by sphygmographic observations, to establish the existence of increased vascular tension in the latter months of pregnancy. Now, as this tension, according to my view, is mainly kept up by the hypertrophied ventricle, it must continue to exist for some time after delivery. This opinion is contrary to the views of Fritsch, but it corresponds with the results obtained by the sphygmograph, and explains to my mind how cardiac disturbances are so apt to continue, and even to become greatly aggravated, subsequently to delivery, when one would naturally expect the threatening symptoms to subside.

I may here also state incidentally, that in regard to the disputed question as to the slowing of the pulse in the newly-delivered, I have come to the conclusion, mainly on sphygmographic grounds, that the slow pulse rate is accompanied by increased tension, so that the puerperal pulse is no exception to the general law that pulses of high tension are slow.

The cardiac murmurs that occur in childbed independently of valvular disease seem to me to be due to mitral insufficiency in connection with the somewhat dilated heart. At any rate, they are usually heard most distinctly at the apex, and also a little to the left of the pulmonary area, exactly where murmurs of slight dilatation of the heart are found to occur. But they are in my experience not at all frequent; for, of a consecutive series of fifteen patients whom I have lately attended during their confinements, whose hearts were sound, although special note of this matter was taken in each case, in only three were murmurs audible.

## CHAPTER IV.

REPORT AND OBSERVATIONS UPON CASES OF CHRONIC DISEASE OF THE HEART, COMPLICATING PREGNANCY, PARTURITION, AND THE LYING-IN PERIOD. PART I.—CASES OF MITRAL OBSTRUCTION. PART II.—CASES OF MITRAL INSUFFICIENCY. PART III.—CASES OF AORTIC INSUFFICIENCY. PART IV. — CASES OF IRREGULAR LESIONS.

I SHALL now proceed to consider those cases of chronic heart disease, complicating pregnancy and parturition, which I have collected, the examination of which was to form the third great heading of our subject.

They amount in all to thirty-one, and embrace—

Under Part I., fourteen cases in which mitral stenosis was the leading lesion, with or without a certain amount of insufficiency. These include one case of tricuspid stenosis, which is well known to be a comparatively rare condition.

Under Part II., eight cases of mitral incompetency. Some of these combined a certain degree of stenosis coincidently with the insufficiency, but the latter was the leading defect in them all.

Under Part III., six cases of aortic insufficiency. Three of these will be found to have been complicated with well-marked mitral obstruction.

Under Part IV., one case of dilated weak heart, and two cases of endocarditis, the one of the plastic, and the other of the ulcerative type.

Thirteen of these cases I have myself watched more or less closely.

For the notes of two I am indebted to Dr. John Linton, and of one to Dr. Ziegler. One has been taken from Bennett's *Practice of Medicine*, and one from Ramsbotham's *Midwifery*.

The rest are abridged translations from the following German authors :—

From Hecker, two cases; from Spiegelberg, four cases; from Fritsch, four cases; from Ahlfeld, two cases; and from Lebert, one case.

## PART I.

CASE I.—*Mitral Stenosis—Hæmoptysis, Palpitation, and Dyspnœa, at end of Sixth Month of First Pregnancy—Pulmonary and General Œdema in the Second Pregnancy—Labour Supervening spontaneously about end of Eighth Month—Sudden Death Six Days after Delivery.*

Mrs. J. T. S., primipara, aged thirty-four, consulted me in the beginning of April 1870, for a cold accompanied with severe hæmoptysis, breathlessness, and palpitation. She had been married in July of 1869, and expected her confinement in July 1870. I examined her chest with care, and found nothing marked so far as the lungs were concerned; but there was detected a distinct præcordial thrill and a loud presystolic murmur at the left apex of the heart. No other murmur audible. I concluded that the hæmoptysis was due to congestion of the pulmonary vessels owing to the mitral obstruction, as I could not elicit evidence of any pulmonic lesion. I inquired into her history, but could discover no trace of a rheumatic seizure. Furthermore, the patient had never suffered from marked breathlessness till about the time she

sent for me. Digitalis and careful management succeeded in restoring equilibrium to the circulation, and the patient got on tolerably well for the rest of her pregnancy. There never was a recurrence of the hæmoptysis. She was confined on the 1st July. Her labour was easy and natural. She had chloroform during the latter part of it. She felt much better after her confinement, and suckled her child; and her health remained good till she became pregnant for the second time. Towards the middle of this pregnancy she became very markedly anæmic; her breathlessness, palpitation, and general discomfort returned, and her legs became enormously œdematous. About the end of the seventh month there was present general œdema, with cough and orthopnœa. I intended about this time to arrange for a consultation, with the view of deciding upon the propriety or otherwise of inducing premature labour, as Mrs. S. was now unable either to lie down or move herself in bed, so much pulmonary œdema and general anasarca were present. But on the 18th of May 1872, the patient, being near the end of the eighth month, fell in labour. The delivery was easy. I gave chloroform with some hesitation at first, but it was well borne, though the pulse was exceedingly weak and very irregular. But Mrs. S. did not improve much after the completion of labour. The anasarca and the dyspnœic symptoms continued, and she was scarcely able to take any food. Her condition remained much at a standstill, however, till the morning of the sixth day after her delivery, when she suddenly fell back in bed dead. There was no post-mortem.

This case occurred in my practice before my attention was markedly directed to the subject, so that I regret that the notes of it are not so perfect as I should have liked. Still it is a case of considerable interest, and presents many of the peculiarities of its class. It will be noticed that

this patient was not aware of having ever had rheumatic fever, and did not suffer from breathlessness, so far as she or her friends had observed, till she reached the sixth month of her first pregnancy. There is, to my mind, no doubt that Mrs. S. had either suffered from congenital stenosis of the mitral, or had been a victim of that stealthily advancing form of valvular endocarditis pointed out by Lebert (see page 32), most probably from her early girlhood. The lesion, however, remained mute till the condition of pregnancy put additional strain upon and injuriously modified the circulatory system, when hæmoptysis with breathlessness and palpitation supervened.

The employment of appropriate care and digitalis were sufficient to rehabilitate the heart for a time, and no distressing symptoms appeared at the period of delivery, or after it, till the middle of the second pregnancy, when the threatening symptoms returned with redoubled intensity.

The question of premature labour was solved, as it usually is in such cases, by the onset of spontaneous premature delivery. But, as happens not unfrequently, as we shall see, though the labour was got over in safety, immunity from danger was not thereby established. In less than a week death happened instantaneously, probably from asthenia,—the heart being no longer able to propel a quantity of blood sufficient to maintain life, probably also hurried on by the depressing effects of the imperfect aëration of the blood in the intensely œdematous lungs.

CASE II.—*Mitral and Tricuspid Stenosis with Insufficiency—Dropsical Symptoms in* 1873, *a Month after a Confinement—Embolism of Branch of Left Middle Cerebral Artery, with Hemiplegia of the Right Side Three Months afterwards—Recovery—Pregnancy again in* 1876, *with great Aggravation of Previous Symptoms of Dropsy and Dyspnœa—Labour*

*premature at Beginning of Ninth Month; Easy—Death from Exhaustion Fifteen Days afterwards—Enormous Dilatation of both Auricles—Contraction of both Auriculo-Ventricular Orifices—Enlargement of Left Ventricle, and Contraction of Right — Atrophy of Posterior Half of Left Corpus Striatum.*

(*Case reported by* Mr. F. R. RUSSELL, *my Dispensary Pupil.*)

Mrs. K., aged forty-six, residing at 17 St. James' Street, states that in August 1873, about a month after giving birth to her last child, she began to suffer from dropsy of the abdomen and legs, with loss of appetite. This became gradually worse till October, when she went to the Royal Infirmary, under Dr. Haldane's care. While there, about a month after admission, and the day before she was to have been dismissed as cured, she had a severe attack of hemiplegia of the right side, being unconscious for seven days. She was able to leave the hospital in January 1874, feeling tolerably well, but her right arm remained weak and semi-contracted. In the beginning of 1875 she suffered from difficulty of breathing, and had slight swelling in the ankles, —symptoms which have continued ever since, with only occasional short periods of intermission. She never in her youth suffered from rheumatic fever.

Her parents lived to about seventy, and had excellent health. A sister is still living. Patient has had five children, four of whom are still living. She has always been of strictly temperate habits.

*Condition on examination, 1st February* 1877.—The patient is rather ill nourished, and lies most comfortably on her left side or sitting propped up in bed. Perspires very much, especially at night.

Pulsation visible in the veins of the neck on the right side. No atheroma to be detected. Pulse 86, rather weak and

sometimes intermittent. She has suffered occasionally from palpitation, and from pain in the cardiac region, but never from syncope. The impulse of the apex beat is seen rather diffused between the sixth and seventh ribs, and can be best felt in the same intercostal space, about four inches to the left of the sternum. The heart's action is often intermittent, and varies much in strength. Slight pulsation can be felt in the auricular area, immediately outside the pulmonary area; it seems to precede the apex beat, and is followed by a marked systolic thrill. Vertical cardiac dulness, a little within the left mammary line, extends from the second rib to the lower border of the seventh, a distance of seven inches, where it is lost in the tympanitic sound of the stomach. Transverse dulness, on a level with the fourth rib, extends from three-quarters of an inch to the right of the sternum to nearly four inches to its left, being a distance of five and a quarter inches. In the mitral area there is a well-marked presystolic murmur, followed by a blowing systolic murmur, which partially replaces the first sound. The second sound is accentuated. In the tricuspid area both murmurs are equally well marked; if anything, the presystolic is scarcely so distinctly heard here. In the aortic area the murmurs can still be heard, but less clearly. In this area the second sound is accentuated and slightly impure. In the pulmonary area the blowing systolic is well marked; still better a little outside of it, over the auricle; second sound strongly accentuated.

Respirations 28 in a minute. Slight dyspnœa, but not severe. Expectoration thin and frothy, but very small in amount. Little cough. Some feeling of tightness on the right side. Patient used to be much troubled with hæmoptysis, having an attack nearly every time she went up stairs. Chest does not expand freely. The muscles of the neck are called into play in inspiration. Vocal fremitus normal;

similarly resonance. Percussion note somewhat impaired over the right side, more especially at base posteriorly. Over the left normal. Slight bubbling râles can be heard over right lung at various points. Respiratory sounds somewhat weak, and expiration prolonged. Urine increased in quantity, high coloured, sp. gr. 1020; contains no albumen at present, but did when tested in January. Copious urates.

Liver dulness not increased. Anorexia and dyspepsia great.

On the night of the 4th Feb. the patient began to have slight pains in the back, but did not think she was in labour, as she did not expect to be confined for three weeks. Next morning she was alarmed to find some blood in the bed, and at once sent for her medical attendant. On examination at 8 o'clock A.M., the os was found to be dilated to the size of a crown-piece, the head presenting in the left occipito-anterior position. The uterine contractions were not very strong, and almost painless; but the head being small, progress was rapid. During the labour the heart's action *increased in rapidity*, and became *more irregular;* the pulsation in the auricular area also became more marked. Pulse 105; respiration 34 per minute. The child was born at 8.40, and was followed by a gush of water and blood, but the bleeding ceased in about ten minutes. The placenta remained in the uterus about three-quarters of an hour after the child was born, and was not removed till 10 o'clock A.M. The patient felt very comfortable after the birth. The pulse fell to 90, and the respiration to 28. For the next three days she was much distressed with after-pains, but otherwise did very well. On the third day the baby began to refuse its food, and, becoming gradually weaker, died on the 15th. On the 9th the patient's breasts became engorged and painful, and this greatly increased her difficulty of breathing. On the 10th the dyspnœa had further increased, the respira-

tions being 38 and the pulse 100. The second sound became slightly reduplicated, and the pulsation in the auricular area more marked. The signs of œdema in the right lung were also gradually increased. On the 13th a distressing cough, accompanied with a large amount of expectoration, set in; the swelling in her ankles diminished, and her breasts almost ceased to trouble her, but the evidence of pulmonary œdema increased. She rallied a little on the 16th, but by the 18th was again much worse, her respirations being 48 and very laboured, and her pulse 106 and irregular. The presystolic murmur could no longer be detected, on account of the disturbances in the breathing. There was now marked dulness along the lower half of the right side posteriorly, with fine crepitation on expiration. These symptoms became gradually worse till the 22d Feb., when the patient sank. The treatment in this case was directed to support by wine, milk, beef-tea, etc., to meet the condition of the lungs with stimulating expectorants, and to aid the heart's action by well-sustained doses of digitalis. A post-mortem examination was allowed, and was made by Dr. Hamilton thirty-six hours after death. Appearances were as follow:—

Body poorly nourished. Left knee partially anchylosed and drawn up, with marks of previous sinuses. Mammæ not well developed. Cartilages of second pair of ribs ossified. Right pleural cavity contains about thirty-five ounces of serum; a small quantity also in the left. Pericardium contains about three ounces of straw-coloured serum. The heart is enlarged to about twice the natural size, extremely flabby, with slight deposits of fat on the surface. The left ventricle is distended with dark-coloured blood, its walls of normal thickness, but its tissue flabby and markedly fatty. The aortic valves are competent; the corpora arantii, more especially of one cusp,

hard and thickened; the bases of the valves are also hard and somewhat calcareous. The mitral valve admits the little finger; its anterior cusp is hard, and evidently in a calcareous condition; the posterior cusp is similarly affected; both segments are adherent at their edges to one another; the base of the valve is thickened and somewhat leathery, but not much contracted. The left auricle is much distended—to twice or thrice the natural size—and thickened. The auricular appendage is also enlarged. The tricuspid valve admits the fore-finger; all its divisions are thickened, and adherent to one another, especially towards their free edges. The right ventricle is extremely small, and its walls are somewhat thinned. A decolorized clot is seen in the pulmonary artery. The pulmonary valves are normal. The right auricle is occupied by a decolorized clot; its cavity, as well as that of the auricular appendage, is somewhat distended.

Left lung somewhat emphysematous, more especially at upper part and edge.

Right lung: Pleural surface covered with a recent and extremely thin deposit of inflammatory lymph. Old cicatrix in upper lobe, uniting this to the lower lobe. Anterior margins of lungs slightly emphysematous; substance very œdematous and congested. The cicatrix has the appearance of being the result of an old infarction. At a localized spot in the lower lobe, about the size of a small orange, is a densely infiltrated, sharply defined portion, deeply congested at some parts, lightly at others, in all probability a recent infarction; the surrounding substance is deeply congested.

Liver small, more especially its left lobe. A cicatrix on the surface of the right lobe. The organ is extremely congested, and somewhat atrophic, in some parts fatty. Spleen somewhat hypertrophied.

Kidneys healthy.

Uterus healthy, and involution well advanced. Breadth of fundus between the insertions of the Fallopian tubes, three and a half inches; length of body and cervix combined, four and a half inches.

Brain: Membranes somewhat anæmic. Posterior half of left corpus striatum entirely wasted, and in its situation a somewhat dense membrane of a slightly yellow colour. Optic thalamus of the same side irregular in shape, and its inner aspect somewhat flattened. The vessels appear healthy.

We had the satisfaction in this case of having the condition during life carefully noted, and also of checking the observations made then by an exhaustive post-mortem examination. The points of interest in this patient's condition are more numerous than in Case I. We have here, as in the other case, no history of rheumatism, so that the main cardiac lesions must have been either congenital or the result of slow and silent endocarditis in early life, unless, which is only probable, the disease originated as an acute endocarditis after the fifth confinement in 1873. The occurrence of embolism three months afterwards no doubt favours this view, as this accident more especially follows recent cases of endocarditis. It would appear, however, if the lesion dates from early life, that the compensatory arrangements were so good that tolerable health was maintained till the patient's system was repeatedly subjected to the special strain of pregnancy, but that after giving birth to her fifth child the previously mute lesion became manifest, leading first to dropsy and then to embolism in a branch of the left middle cerebral artery. Considering the great amount of structural change affecting the left corpus striatum and optic thalamus, extending, as such lesions are known to do, down the cord, it is extremely surprising that the extremities on the right side of the body should have possessed so considerable an amount

of motor power. A condition such as this lends strong support to the view that adjoining portions of the brain can vicariously discharge the function of other parts that have been destroyed by disease. But besides leading to cerebral embolism and dropsy, the effects of the backward congestion, resulting from the extreme stenosis of the mitral, are seen also in the numerous infarctions of both old and recent date, as well as in the bronchial congestion, the pulmonary œdema, and the pleuritic effusion. From the history of the physical signs, it is manifest, however, that the latter condition only came on a very short time before death. The amount of cardiac disease in this patient was so great, that her life could scarcely have been much prolonged even although pregnancy had not occurred. Still, that the latter condition seriously aggravated matters, and intensified the antecedent tendency to death, does not admit of doubt. For the symptoms of dyspnœa and pulmonary œdema, with premature onset of labour, are precisely those leading features that are found in Case I., and which we shall find, as we proceed to the other cases, almost invariably accompany severe cardiac disease when it is complicated with pregnancy. One would naturally have expected that in such a case as this the delivery would have led to remission of the chest symptoms, and indeed it is looked upon as an axiom among accoucheurs that premature labour is warranted under such circumstances. It will, however, be seen that in neither of these cases was premature delivery followed by any appreciable benefit, even when it occurred spontaneously, and consequently when it may be considered to have come on in the manner least likely to give rise to injurious effects. I may mention this by the way, as we shall see that these cases are not singular in this respect; and indeed, from what I have been able to gather, I should be inclined to dispute the propriety of ever inducing premature

labour to relieve pulmonary symptoms when the heart is diseased, except some abnormal abdominal pressure be present. The pathological condition of the heart is extremely interesting. It will be observed that the left auricle was greatly distended and thickened, which was obviously the result of the efforts of the left auricle to propel the blood passing through it into the left ventricle against the obstruction caused by the extremely stenosed mitral. The left ventricle was large, and its walls of natural size, whilst the right ventricle was extremely small, and its walls thinned. The right auricle was distended. We meet here with a rare condition of the right ventricle, occasioned doubtless by the long-continued tricuspid stenosis. This is the ordinary condition of the left ventricle in the case of mitral obstruction; but as tricuspid obstruction is very rare, this consequence of it must necessarily be rare also. I believe, however, that this condition must have tended towards diminishing the evil effects of this patient's heart disease so long as the right ventricle was able to propel sufficient blood to support existence. For the pulmonary vessels must necessarily have been subjected to a minimum amount of tension, when to the retro-dilatation inseparable from the severe stenosis of the mitral there was merely added so much contraction of a small and thin right ventricle as was competent to carry on the circulation through them, and to maintain life. In other words, I look upon the small right ventricle, in this patient's case, as a compensatory arrangement of a conservative nature, but which was liable to precipitate fatal results, when to the ordinary duties of the weakened right ventricle the propulsion of blood through an emphysematous and bronchitic lung was superadded. So soon as the right ventricle became unequal to its task of maintaining even the minimum of pulmonary circulation, then the destructive effects of retro-dilatation,

commencing at the mitral aperture, would make itself abundantly evident. The blood being imperfectly forced towards the lung by the feeble right ventricle, the obstruction resulting from the mitral valve during the contraction of the auricle, and the degree of regurgitation which would, and, as proved by auscultation, did take place through the mitral orifice during the contraction of the flabby but yet tolerably powerful left ventricle, form a concurrence of influences all tending to maintain the lungs in a state of persistent hyperæmia. Little can be inferred with certainty from the condition of a heart so very much diseased, but it is, as far as it goes, on the side of the view of the French authors, that the left ventricle hypertrophies during pregnancy, for there does not seem to have been any obstruction in the systemic circulation to warrant the relative difference in size of the two ventricles. Both cavities, no doubt, could only be partially filled, in consequence of the stenosis of their respective afferent orifices. But the resistance presented by the force necessary to propel the current of blood through the lungs, must have been to the *right* ventricle relatively greater than the force required to maintain the circulation of the blood throughout the body would be to the *left* ventricle, if we take the condition of pregnancy out of the reckoning. Still we find that, while the right ventricle became thinned and contracted, the left became dilated, and at any rate not thinned. So far, therefore, as this case may be depended upon, and I grant and have said that it is not much, it affords additional evidence in support of the opinions of those who maintain that there is a physiological hypertrophy of the left ventricle during pregnancy.

CASE III. — *Mitral Stenosis — Hemiplegia, with a certain degree of Aphasia, on 10th January 1877 (Embolic?)— Partial Recovery—Labour premature in consequence of*

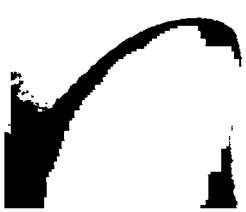
*Accidental Hæmorrhage, on 21st February 1877, being about Three Weeks too early—Urgent Symptoms during Delivery, which was ended by Forceps—Recovery.*

(*Reported by* Mr. FRANCIS R. RUSSELL, *my Dispensary Pupil.*)

Mrs. Conway, aged thirty-five, residing at 27 W. Norton Place, states that she always enjoyed good health, never having had rheumatic fever, nor any other serious illness that she can remember. Occasionally, when at work, she suddenly became giddy, which obliged her to rest for half an hour or so, and during these periods she was only partially conscious. This has occurred three or four times during the last year. She had an attack of childbed fever after the birth of her sixth child. She has had eleven children. The first is alive; the second was an abortion at the sixth month, after a fall in the street; the third is living; the fourth was premature, and born dead; the fifth was also born dead, but at the full time; the sixth is living; the seventh still-born at the full time; the eighth at the fourth month; the ninth was born at the full time, and is still living; the tenth was at the fifth month; and the eleventh at the full time, but dead.

On the 12th January 1877 she had a hemiplegic attack. She was unconscious for about twenty-four hours, and when she came to herself again she found she had paralysis of the right side. In a week she had recovered the use of her limbs, but her face was still drawn to the left, and her tongue protruded to the right. Her intellect remains slightly impaired, and she is still somewhat aphasic.

*Condition on examination, 20th Feb.* 1877. — No pulsation can be detected in the veins of the neck. Pulse 84, regular and full. She has never suffered from palpitation nor faintness. The heart's apex impulse can be faintly seen below the nipple; and on palpation it can



be felt between the fifth and sixth ribs two inches to the left of the sternum. Very slight pulsation can also be detected between the third and fourth ribs, an inch and a half to the left of the sternum, accompanied by a very faint thrill. Perpendicular dulness, in a line one inch to the left of the sternum, extends from the second rib to the upper border of the seventh, where it becomes tympanitic. At the level of the fourth rib transverse dulness extends from the centre of the sternum to 2¼ inches to the left of it. In the mitral area are audible—1st, a well-marked pure presystolic murmur; 2d, an accentuated first sound; and 3d, a slightly impure second sound, also accentuated. In the aortic and pulmonary areas a reduplicated second sound can sometimes be faintly heard; the pulmonary second sound is accentuated. Everything else normal. The sphygmographic tracing taken at the time, and which is given below, indicates the presence of great tension of the arterial system. Liver dulness is not increased. The patient has never had any general symptoms of obstruction to the circulation. Urine natural, and contains no albumen. Intelligence still markedly impaired. All other organs appear normal.

*Progress of Labour.*—On the afternoon of the 21st, a few drops of blood were noticed on the floor where the patient had been standing. She was at once put to bed, and soon after had a slight attack of hæmorrhage. She had been vomiting severely all day, and by night she had become pale and exhausted looking. At 7.15 P.M. her pulse was 108, regular, and of moderate strength. The external os was dilated to about the size of a penny piece, and filled up with a firm clot of blood. The pains returned at intervals of about twenty minutes. The uterus was continuously tense. The head presented.

8.30.—Pulse 115; decidedly feebler. Os dilated to the

size of half-a-crown. Patient had another attack of vomiting; still some bleeding.

9.30.—Pulse 100; extremely weak. Os about the same size. Hæmorrhage quite stopped. Breathing not at all affected.

10.15.—Pains now occur every five minutes, but are weak. Pulse so feeble that a glass of whisky and water was administered. Pulse immediately rose to 120, but fell in a few minutes to 94—much stronger; can occasionally be detected to be slightly irregular. Pulsation and thrill in auricular area greatly increased, where second sound is markedly accentuated. No cough. Os dilating very slowly; membranes very tense and strong.

10.45. — Pulse 120; irregular and weak. A tablespoonful of whisky reduced it to 72, and made it at the same time more powerful. Cervix about half dilated. Membranes were ruptured by the finger, and the waters evacuated.

11.30.—Pains weak, but uterus *constantly* tense. Pulse 100. Head ascertained to be in the right occipito-posterior position, and now well down in the pelvis.

12.30.—Pulse 108. The labour is now almost at a standstill. The occiput is more and more approaching the sacrum, and the anterior fontanelle is almost centrally placed. Accordingly it was deemed proper to effect delivery by forceps. The face continued forwards. The patient was much relieved immediately after delivery. The child was born at one o'clock A.M., and found to have been dead for some time. It was thought to be about three weeks before the full time. The placenta was found to be quite detached, and was expelled immediately after the child. It had the appearance of being channelled out by a blood-clot, hæmorrhage having apparently taken place into its centre, causing almost complete separation of it, and leading to considerable hæmorrhage between the membranes.

The auricular pulsation had nearly abated at 1.15.

2 P.M.—Uterus not firmly contracted; slight hæmorrhage. Four grains of ergotin were injected. Pulse 144.

4.30.—Hæmorrhage ceased. Uterus fairly contracted. Pulse 110.

3 o'clock.—Pulse fallen to 90; moderately strong and regular. Patient feels comfortable.

From this time onward the patient made a steady though very slow recovery.

This patient was seen and examined by me before her confinement, and also watched with great concern during it. From what I could ascertain by the most careful inquiry, I judge that her eleventh confinement had been complicated with placenta prævia. At any rate, there had been a very large amount of hæmorrhage before the birth of the child; and the patient's husband states that his wife never properly recovered from her last confinement. It is extremely interesting to note how, time after time, those parturient patients affected with chronic disease of the heart express themselves as unaware of ever having had rheumatic fever.

In the present instance, I am inclined to believe that the cardiac disease must have begun with endocarditis, arising in connection with her immediately preceding confinement. There is no history of cardiac disturbance of any kind in her case to warrant us in believing that the lesion had been congenital, or had begun in early life. Indeed it is, it seems to me, extremely improbable that such a lesion beginning in early life should have remained mute for so many years, and through so many pregnancies; and, besides, we have in relation with her eleventh pregnancy exactly the kind of condition that is wont to give rise to endocarditis. The stenosis in her case was pure, and still moderate in amount. The view of its being recent is rather confirmed by the

occurrence of embolism, which is more likely to follow from a recent case if the embolism is due to the escape of a valvular vegetation. But of course that is at best a probability, because, if embolism is due to the formation of auricular clots, it is more likely to occur in cases of old standing. There was in our patient's case only slight increase of cardiac dulness in the perpendicular direction, with unmistakeable increase in the transverse direction,—facts which indicate the commenced disruption of the compensatory arrangements that had been formed before the beginning of the pregnancy. The advance of the dulness to mid-sternum in a line with the nipple indicates considerable dilatation of the right auricle. Still the backward congestion was not as yet sufficiently great to cause any marked pulmonary symptoms, most probably because the right ventricle was still working powerfully, and was well nourished.

To the transmission of tension from the mitral valve backwards on the venous system, however, I am disposed to ascribe this accidental hæmorrhage, as it is called by obstetricians,—that is, the rupture of the placental vessels, which led to the premature delivery by effecting the curiously excavated separation of the placenta. This hæmorrhage, though slight in actual amount, introduced a complicating force, which renders the case, however carefully watched, of less value in aiding us to determine the mutual bearings of heart disease and of parturition upon one another than it otherwise would have been.

With a view to determine, if possible, the degree of the vascular tension, sphygmographic tracings were taken of this patient's pulse the day before the labour came on, and during delivery both during and in the interval between a pain. Specimens of these tracings are herewith annexed:—

No. 1.—Taken the day before delivery. Indicates great arterial tension.

No. 1.

No. 2.—Obtained from same patient's pulse after the hæmorrhage had taken place. Shows very great feebleness.

No. 2.

No. 3.—Tracing of same pulse during pain. Shows effects of pain on the pulse.

No. 3.

Before labour, it will be observed, on examining tracing No. 1, that the perpendicular range of the instrument was extensive and the pulse wave full, the tidal or predicrotic wave being especially strongly marked, while the dicrotic wave is only feebly indicated,—signs which indicate considerable tension of the systemic circulation. It will be observed that the high tension was accompanied with considerable irregularity. Every third wave is markedly more elevated than the two preceding. But during the labour the tension of the arterial system had sunk down very markedly, as is seen on inspecting tracing No. 2. The

pain did slightly increase the force of the pulse, as is shown by an inspection of the tracing No. 3, but only to a trifling extent. There had therefore taken place a manifest change in arterial tension between the previous day and the establishment of the labour. Whether to ascribe this alteration to the disturbing effect of the labour upon the circulation, or to the hæmorrhage which initiated the latter process, is a question of little difficulty, since, on the whole, by far the most obvious and probable cause seems to have been the hæmorrhage. But that the labour pains acted in a very peculiarly perturbing manner upon the action of the heart, is evident from the extraordinary changes in frequency that were seen to occur. It was not so much the irregularity of one pulsation compared with another, for there was not so much of that, as it was the extremely variable nature of the rate of pulsation for considerable periods. Thus the pulse would jump up to 120 at one time, and continue at that rate for a time with tolerable regularity; and then again it would come suddenly down to 94, and continue so for a considerable period,—these rapid alternations bearing no hard and fast ratio to the presence or absence of labour pains.

There is reason to believe, on general grounds, that during a pain arterial tension is increased. This undoubtedly must be the case during the second stage. But it does not appear to occur to a great degree during the first stage, to judge from the tracings which we have obtained. During the second stage, however, there is so much muscular tremor and restlessness as to render sphygmographic observations comparatively worthless.

In passing on to the further consideration of the case, this lowness of arterial tension and the coincident tendency to post-partum hæmorrhage seem also worthy of remark. We seldom see hæmorrhage post-partum when the pulse is

slow,—*i.e.*, as I hold, strong,—or rather when arterial tension is high.

It might be said that this patient, having had a series of abortions or dead children, was most probably constitutionally syphilitic, and that this condition led to the placental separation and premature delivery. I have studied with some care the subject of syphilitic placenta, and can vouch for its not being present in this patient's case. Moreover, after the most careful inquiries, we could elicit no fact that would warrant us in believing that the patient was constitutionally syphilitic.

Was the paralysis that occurred on the 7th of January due to embolism or to apoplexy? I think the presumption is that it was embolic. The amount and intensity of it was not so great as we should have expected in apoplexy. Moreover, the patient's blood vascular system, so far as we could judge of it by inspection, was quite normal, with the sole exception of the cardiac lesion. It is further well known that, supposing no vegetation had existed on the mitral valves, the blood stasis, arising behind such a constricted mitral, is apt to lead to the formation of clots in the auricular appendage. One of these clots may then have become loose and settled in some part of the left middle cerebral artery or its branches. But there is nothing improbable in the idea that a small vegetation had dropped off from the edge of the diseased mitral valves, and had got impacted in that part of the vascular system.

I think that it can fairly be regarded as probable from this case, that so soon as a mitral stenosis is sufficiently great to lead to dilatation of the right heart, it is liable so to be aggravated by the pregnancy as to transfer such an amount of pressure from the arterial system to the veins as may lead to rupture of the placental cells, and may in that way terminate the pregnancy prematurely. On one occasion I was

asked by Dr.—now Professor—Stephenson, of Aberdeen, to see a case with him, which bears out the view that undue vascular tension may lead to placental apoplexy, and hence to premature delivery. My note of this case runs as follows:—Mrs. C., aged twenty-seven, was in March 1875 about the seventh month of pregnancy. On Saturday, 27th March 1875, while calling at Dr. Stephenson's house, whom she had engaged to attend her in her approaching confinement, she felt an acute abdominal pain. She went home, and was next day delivered prematurely. There was considerable hæmorrhage before the os opened, and great prostration. These symptoms appearing serious, Dr. Stephenson asked me to see her with him. She quickly recovered, however, from her collapsed state. On examining into the cause of the miscarriage, and finding intense albuminuria, we thought the consequently heightened blood pressure might have led to separation of the placenta. On examining that structure, we found part of it occupied by a hard and firm clot, which had evidently been formed the day before. The urine was almost completely suppressed. There had also been vomiting during the day. We gave some chloral and ice, and orders to watch in case convulsions came on. One occurred that night, but not afterwards. This patient did well.

Then, when we have the mitral lesion complicated with even a comparatively small amount of hæmorrhage, we may expect a very alarming amount of feebleness and irregularity of the pulse, which, however, will in all probability disappear on the termination of the labour. Such cases manifestly require the free use of stimulants, notwithstanding the existence of hæmorrhage, and warrant us in hastening delivery by artificial means. Had I had digitalis by me during the time I watched this case, I certainly should have administered it freely; but the labour came on at midnight, and we were a considerable distance from a chemist's shop, so

that we were compelled to depend upon a general stimulant, without the advantage of the special cardiac one.

Some very interesting observations bearing upon this subject are to be found in a paper by Duroziez upon 'Pure Stenosis of the Mitral,' published in the *Archives Générales de Médecine*, Juillet 1877, p. 47. He points out that this lesion is apt to be accompanied with irregularity of menstruation, and with hæmorrhage during pregnancy and after delivery, as also with a special tendency to the occurrence of abortions and premature delivery. He finds that menstruation is often late in making its appearance in patients suffering from pure mitral stenosis; that it is apt to be irregular in its recurrence, and to be at certain times very profuse, at other times very scanty. He is inclined to regard the menstrual disturbances associated with the lesion as referable to the anæmia, which, from hæmoptysis, epistaxis, and otherwise, is so apt to complicate this cardiac lesion.

His experience leads him to the conclusion that death seldom follows from pregnancy when that is associated with pure stenosis, as the premature delivery, which is so apt to come on, removes, he thinks, the risks that would arise if the utero-gestation were continued to the end of the normal period.

This opinion is certainly not borne out by my experience of mitral stenosis; but few of my cases have been capable of being classified as pure. Perhaps this one might be regarded as nearly so; at least we find no evidence to the contrary, except the increased dulness at the base, which, however, was probably due to temporary causes.

CASE IV.—*Mitral Stenosis—Labour at Full Term—Threatening Symptoms of Weakness and Irregularity of the Pulse during Second Stage—Delivery by Forceps—Breathlessness, Cough, and Hæmoptysis during Seventh*

*Month of Second Pregnancy—Cardiac Pain, Faintness, and Irregular Pulse during Second Confinement—Delivery by Forceps—Recovery.*

(*Reported by* Dr. JOHN PLAYFAIR.)

Mrs. T., aged twenty-three, primipara, a slightly-built woman, under the average size, but fairly well developed, consulted me in the beginning of October 1876 for a pain in her left side, with occasional palpitation and breathlessness on exertion. On listening over the heart's apex, I heard a distinct presystolic murmur. The patient told me she expected to be confined in the second week of February, and I agreed to attend her. A belladonna plaster was ordered, and a mixture containing digitalis. I heard nothing more from her till I was called to attend her in labour on the 7th February 1877. On arriving at the house at twelve noon, I was told the pains had commenced at three A.M., and that the waters had come away at ten A.M. Patient also informed me that the pain in her side had not troubled her much after she had seen me in October, and that her health had been very good during her pregnancy, though always liable, on rapid movement or any excitement, to palpitation and slight dyspnœa. On vaginal examination, the os was found to be fully dilated, the membranes ruptured, and the vertex presenting. The head was already well down in the pelvis, with the occiput almost opposite the symphysis pubis, but inclined slightly to the left. Pulse was 104, soft but regular, and of fair strength. The pains were occurring about every four minutes, but were not strong, and of short duration. Patient showed no sign of exhaustion, and she had not as yet exhibited any tendency to faintness. During a down-bearing pain I now noticed that the pulse became imperceptible, and the patient somewhat cyanotic. Before, however, the sense of pulsation in the vessel was lost, the

beats were observed to quicken very considerably. As by two P.M. the case had made no progress whatever, I determined to apply the forceps. The patient was first put fully under the influence of chloroform, and now I observed that the pulse had become decidedly intermittent and irregular, and had risen to 120, and that the cyanosis was very marked. At one time the irregularity of the pulse was of such a character that a weak beat could be distinctly felt to follow a strong; and from the time the anæsthetic was first administered to the termination of the labour, the rhythm of the pulse varied much, but never became completely regular. The forceps were easily applied, but considerable force was necessary to bring the head through the bony outlet. The forceps were then removed, when delivery was speedily completed by the natural efforts. The child was born at 2.30. It was a strong, healthy-looking girl, and cried lustily immediately after its birth. The patient very soon came out of the chloroform. The uterus contracted well, and the placenta came away easily without any bleeding. A few minutes after the birth the pulse was 104, irregular; and when I left the house at 3.30 it was 96, but its character was not noted. At 9.30 P.M. the pulse was 96, slightly irregular, but not intermittent. Patient says she feels very comfortable, and inclined to sleep. Has had no sense of faintness. Had passed water.

Dr. Macdonald saw the patient with me next day, the 8th February, when the condition of the heart was made out to be as follows:—On palpation a diffused apex beat is felt, accompanied with the peculiar presystolic thrill, the pulsation being best felt, however, between the fifth and sixth ribs, about a quarter of an inch to the inner side of the vertical nipple line. On inspection, there is visible pulsation over an area about an inch and a half square, extending as high up as the transverse nipple line (the nipple being on the fourth

rib), and as far to the right as the left edge of the sternum. On auscultation, there is a distinct presystolic murmur heard over the pulsating area, the murmur ending abruptly with the first sound. At the base in both the pulmonary and aortic areas the second sound is markedly reduplicated, while in the pulmonary area it is also intensified. The presystolic murmur is heard, though faintly, in the pulmonary area. On percussion, the transverse dulness is not markedly increased, while the vertical dulness, beginning at the third intercostal space, is lost below in the dulness of the left lobe of the liver. There is no history of rheumatism. The patient has a slight cough, but there is no pulmonary disease detectable. To-day her pulse is 88, regular. She is doing very well.

22d February.—Patient has progressed very favourably since last report, and is now able to move about the house, though still rather feeble.

This patient again became pregnant in 1877, and in the month of October, being about the beginning of the sixth month, she began to suffer from breathlessness and severe cough, with repeated hæmoptysis,—the latter, however, in small amount. With careful treatment and strict abstinence from all exposure and work, as also along with the free use of digitalis, Mrs. T. improved considerably. At 1 P.M. of 2d February 1878 she again fell in labour. I saw her at 6.50 P.M., and found the os fully dilated, membranes protruding, and pains of an extremely feeble character recurring every five minutes. Pulse 124, irregular, intermittent, and extremely weak. The patient complained of pain in the region of the heart, and of a feeling of faintness. Being seriously concerned about Mrs. T.'s condition, I despatched a message for Dr. Macdonald. He arrived about 7.30, and we then conjointly made the following note of her condition:—Pulse 124, very weak, irregular, and intermittent. At the mitral area a well-marked presystolic murmur is audible, and along

with it a distinct systolic bruit. The latter is inaudible in the tricuspid area. At the base the second sound is heard markedly reduplicated and accentuated, especially in the pulmonary area. A marked precordial thrill is felt over the whole cardiac area, where also visible pulsation could be seen, more particularly over the auricular area. On percussion, the cardiac dulness is found to measure 5 inches in a transverse line on the level of the fourth rib, commencing with the right border of the sternum. The perpendicular dulness is also 5 inches. The apex beats with most force between the fifth and sixth ribs in the vertical nipple line. The veins in the neck are seen to pulsate and to fill from below when pressed upon. There is no cyanosis. As the pains were producing no effect upon the progress of the labour, it being clearly considerably retarded by the patient being entirely incapable of putting forth the slightest down-bearing effort, we decided to chloroform her, and deliver without further delay. Dr. Macdonald accordingly administered chloroform,—which steadied and strengthened the pulse,—and I applied instruments. The occiput was to the right and posterior, with low position of the anterior fontanelle, but the head rotated while in the grasp of the instruments, and delivery was easily and speedily effected.

The patient rallied greatly immediately afterwards. In less than an hour the pulse had fallen to 98, and was both stronger and steadier. The systolic murmur became inaudible, though diligently listened for, immediately after delivery. There was noticed a peculiar and unusual restlessness in the patient's demeanour throughout the whole of the delivery. There was no detectable pulmonary œdema. The accompanying tracing, No. 4, of Mrs. T.'s pulse was taken forty hours after her second delivery. It indicates some enfeeblement. Working pressure, 3 oz. Obliterating pressure, 15 oz.

3d February.—The patient felt greatly better, only she was much annoyed by a distressing short cough, for which no physical cause could be detected. For this she was

No. 4.

treated successfully by small doses of digitalis with chloral. From that time she recovered steadily. In spite of our strongest opposition, she persisted in nursing her child.

In this patient's case we meet with a most interesting example of a tolerably well compensated mitral lesion, which from its commencement would appear to have remained mute till the patient became far advanced in her first pregnancy. Had she not become pregnant, the fair presumption is that she would not have been aware of her heart disease for many years to come. Sphygmographic tracings obtained, one a fortnight, and another two and a half months after her first confinement, indicate considerable loss of arterial tension from the mitral obstruction. Still this patient could get on quite comfortably, the heart being equal to the ordinary requirements of a tolerably active life. During the latter periods of the pregnancy, however, the usual initial disturbances arising from such cardiac lesion, viz. palpitation and breathlessness, made themselves disagreeably prominent, but yet not to such an extent as to excite much concern in the patient's mind, or in Dr. Playfair's. Even during the first stage of labour the patient suffered little, if any, inconvenience from her heart disease. Nevertheless, when the second stage was reached, and proved to be difficult, the cardiac disturbance, in the shape of irregularity of the pulse and tendency to syncope, became positively alarming,

and led to the necessity of shortening the second stage. With the delivery it is to be noticed that the irregularity of the pulse abated, but did not entirely disappear for some time. Now this we observe in all such cases, except perhaps when the main lesion is aortic insufficiency. In that case, after delivery, the threatening symptoms usually subside rather suddenly on the successful conclusion of the second stage of labour.

It is evident from this case, as well as from several others, that during the first stage of labour there is not such an amount of extra pressure in the arterial system as makes any great demand upon the action of the heart. But so soon as the irritation due to the pressure of the uterine contraction has the down-bearing effort superadded to it, symptoms of an alarming nature are apt to arise.

It is easy to understand that a very little more irregularity and feebleness of pulse might have ended in fatal syncope during either of the two confinements.

The successful termination of her second confinement, notwithstanding the threatening symptoms occurring from the sixth month onwards, may fairly be in no small part ascribed, as it appears to me, to the careful management of the patient during the latter months. The patient's safety was also doubtless greatly secured by the prompt means that were adopted to abbreviate artificially the second stage of labour on both occasions.

It is also worthy of remark that the condition of this patient's heart, as ascertained by the increase of the transverse cardiac dulness, had been greatly altered for the worse between her first and second confinement. A peculiar circumstance is brought out in this and the following two cases, viz. that a systolic murmur is usually developed in those obstructive cases during the second stage of labour. The probable cause of the phenomenon is discussed under Case V.

How do patients who suffer from this form of mitral disease so frequently have an irregular weak pulse during the down-bearing efforts? That such is ordinarily the case, we may, I think, accept as a fact. At any rate, the present case and the last one abundantly prove its occasional occurrence, and in the sequel we shall find it a tolerably constant symptom. Let us attempt to analyse the physiological conditions connected with a down-bearing effort, with the view, if possible, of presenting a rational explanation of this occurrence. I am well aware of the difficulties attaching to all methods of explaining cardiac irregularity, and merely throw out this as a probable explanation in the special circumstances. 1st. We have the lungs tensely filled with air, and the glottis shut. 2d. The diaphragm is depressed as far as possible, and its muscular area contracted as tensely as it can be. 3d. The uterus is contracted so that the venous blood is squeezed out of it. 4th. The abdominal walls are powerfully contracted.

In consequence of the conjunction of the three latter influences, the blood existing in the venous system throughout the abdomen must be pushed forwards with considerable force into the right auricle, and thence into the right ventricle, so as to produce over-distension of these cavities. But the existence of a mitral obstruction for any length of time necessarily produces a dilated and weakened right heart, as well as a dilated, though not necessarily weakened, left auricle. We are therefore entitled to hold that in the present case such a condition of affairs existed to a certain amount. The lungs being distended with air under considerably increased tension during the down-bearing effort, would present further difficulty to the action of the right ventricle. The mitral stenosis maintaining a condition of continuous engorgement of the dilated left auricle, in its turn aggravates matters. Also, if it be true that during

pregnancy the left ventricle increases in thickness, whilst the other three chambers receive no addition to their muscular tissue, pregnancy would only make these chambers more liable to yield to the distending forces I have mentioned, and at the same time leave an hypertrophied left ventricle to act with its unusually great force in the same direction so far as the stenosis was accompanied by insufficiency; that is to say, the tendency of the powerful ventricle would be to still further aggravate the evil effects of the stenosis upon the left auricle. Add to all this the fact that we have within a given section of the vascular system an unusually large supply of blood, in consequence of the increased bulk of that fluid in circulation during the latter months of pregnancy, and we have during the down-bearing pains, it appears to me, sufficient conditions to account for the onset of very weak and irregular heart's action, with dyspnœa and cyanosis. In short, three chambers of the heart—viz. the right and left auricle and the right ventricle—are paralysed, not as Fritsch would have it, by a sudden gush of blood entering them when in a state of emptiness, but by over-distension. If the aortic pressure sinks, it is not from any removal of the placental circulation, for we assume that this as yet has not taken place, but from want of blood-supply from the left auricle or from general exhaustion. The blood not getting through the lungs is not aërated, cyanosis results, and general depression of cerebral action occurs. In many cases this latter condition is sufficient to occasion unconsciousness, the patient being in a condition approximating to chloroform narcosis. It is easy to see that though on a superficial examination this condition would appear to exclude the administration of chloroform, yet as this drug diminishes very markedly the down-bearing effort, on a closer examination of all the circumstances as I have put them, the advantage in stopping the *fons et origo mali* will be greater than any possible

disadvantage that might be expected from it. The latter belief is at any rate borne out by my experience of the use of this drug.

CASE V. — *Mitral Stenosis — Hæmoptysis, Palpitation, and Breathlessness on exertion during Latter Months of Sixth Pregnancy—Alarming Symptoms during Labour, which was easy—Recovery.*

(*Reported by* Dr. JOHN PLAYFAIR.)

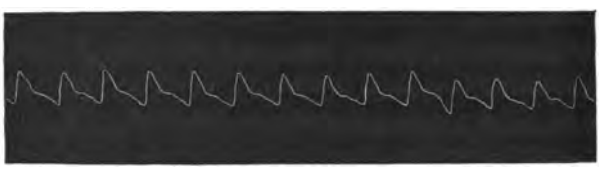
Mrs. C., aged thirty-five, residing at 17 Barony Street, when about ten years of age had rheumatic fever, but never any other illness of importance, having always enjoyed good health. On being seen on the 2d of October 1877, she was found to be in the ninth month of her sixth pregnancy. She has had no miscarriages. Two of her children died from scarlet fever, and two from acute tubercular meningitis. She never suffered from chest symptoms till about two years ago, when, during the illness of one of her children, she was much exhausted with nursing. She then began to feel pain in the left side, and suffered from breathlessness and palpitation. In the summer of 1876 she consulted Dr. Macdonald about indigestion. He then detected mitral stenosis. Within the last twelve months she has occasionally expectorated small quantities of blood. She has, however, very little cough, and no mucous expectoration, but suffers from breathlessness and palpitation on exertion, though she does not think these symptoms have become worse since she became pregnant. Her appetite is fair, and her digestion good. On inspection, the veins over the front of the chest are seen to be somewhat engorged. After exertion, pulsation is observed over the cardiac area and in the neck; but after lying quiet for a little, pulsation is only visible between the fifth and sixth ribs, inside the perpendicular nipple line. It

does not subside, however, in the neck. On palpation, diffused pulsation and a slight thrill are felt after exertion, but on lying quiet these symptoms also almost entirely disappear. Transverse cardiac dulness measures 3¼ inches, and the perpendicular, 4 inches. At mitral area a loud rough grating murmur is heard to precede the first sound, and run up to it. The latter is perceived to be clear, but sharply accentuated. Traced towards the axilla, the murmur gradually disappears, and only the accentuated first sound is heard. In the tricuspid area the presystolic murmur is still audible, but not so clearly marked. In the auricular area it is also audible, but not so loudly. At the pulmonary and aortic areas, the first sound is rather harsh and accentuated, the second sound is very remarkably reduplicated. Posteriorly, within the lower angle of the scapula, the first sound is heard unaccompanied by a murmur.

The patient fell in labour early on the morning of the 28th October. The first stage was slow, and unaccompanied by any special heart symptoms. At 9.15 P.M. the membranes were ruptured, and thereafter the pains became decidedly down-bearing in character. The pulse now rose to 100, and became distinctly irregular. The patient herself complained of palpitation and faintness during the pain, feeling, she so expressed it, as if her 'heart were being torn from her.' Her face was much flushed, and she was altogether greatly distressed. I now was on the point of putting the patient under chloroform, and sending for assistance, being alarmed in case the down-bearing effort should precipitate a serious issue, and thought I ought to shorten the second stage with instruments. But, on examining, I discovered that meanwhile the head had descended very rapidly, and was already distending the perineum. Pressing on the forehead from the rectum, I got the head safely and easily over the perineum and through the vulvar opening, and by 9.30 the child was

born. It was a fine healthy boy of full size. The patient never fainted, and expressed herself as much relieved after the birth. The placenta was spontaneously separated, and expelled about ten minutes after with very little hæmorrhage. The pulse immediately after delivery was noticed to be 80 beats per minute, of good strength, irregular, but not intermittent. The præcordial thrill was felt to be now wonderfully lessened. On auscultation, the presystolic murmur was heard at the apex, along with a distinct systolic murmur. At the base, the presystolic murmur was also heard, but faintly, and the systolic not at all. Second sound, especially in the pulmonary area, was observed to be intensified and reduplicated.

29th October.—Pulse 76, and feels almost regular. The



No. 5.

systolic murmur heard last night at the apex can no longer be detected. The patient slept occasionally during the night, and feels very comfortable. From this time onwards she continued to improve uninterruptedly.

This patient presents a case of tolerably well-marked but not severe mitral stenosis. The accompanying pulse tracing, taken during the eighth month of this pregnancy, exhibits great regularity in the radial pulse, although it also shows evidence of somewhat enfeebled cardiac action. It is difficult to determine whether the cardiac disease had originated with the attack of rheumatism or not. Had it done so, the amount of involvement of the valves must have been slight, and of such a nature as to give rise to no objective symptoms.

I myself attended Mrs. C. through several confinements before she passed under the care of Dr. Playfair. During these I never detected any cardiac lesion, though it is my custom almost invariably to examine the state of that organ before I administer chloroform. At any rate, if the lesion existed then it was perfectly mute, for until the present delivery Mrs. C. showed no symptoms of irregular action of the heart during any of the stages of labour.

It is worthy of remark how sharply the second stage of labour was defined from the first, in so far as seriousness of symptoms was concerned. All went well during the first stage. But apparently, so soon as the additional pressure upon the vascular system called forth by the down-bearing pains became operative, serious symptoms supervened. The pulse became quick and irregular, the patient felt excessively distressed; and had it not been that this stage was very short, it is beyond question that the patient's life would have rapidly been in great jeopardy. This case, therefore, illustrates the very great effect of the down-bearing pains in straining the weakened powers of an already enfeebled diseased heart, and supports to the full, as I believe, my view that it is not any sudden diminution of arterial pressure, arising, as Spiegelberg suggests, from the removal of the placental circulation, that we have to fear as the cause of sudden death during labour, but rather the presence of the *extra pressure* arising out of the down-bearing efforts. According to my view, the danger from disturbed pressure is past so soon as the child is expelled, and not only beginning then, as Spiegelberg would have us to believe.

The fact of a systolic murmur appearing in connection with this delivery, as was observed by Dr. Playfair, is extremely interesting. The same phenomenon was seen in Case IV.; and when we come to Case VI., it will be observed that a like murmur was developed in it also. How

did it occur? At what orifice was it produced? On this point we consulted Dr. G. W. Balfour, and he expresses himself as strongly inclined to believe that it was tricuspid in origin. In that case, the explanation would be that the resistance arising in the contracted mitral, when added to the difficulty which the blood experienced in passing through the lungs during the down-bearing efforts, was able to cause such an amount of retro-dilatation of the right side of the heart as produced rapid dilatation of the right auricle and ventricle, and consequent incompetency of the tricuspid. If this were so,—and it certainly seems the most rational explanation of the phenomenon, for it is difficult to understand how such a murmur should have been temporarily developed in the mitral, whilst it could not be detected in either Case IV. or in the present one before the delivery nor since,—it would help to afford an explanation of some of the very serious results which labour brings with it in cardiac cases. If the right heart could so suddenly dilate, as in this case, that it led to insufficiency of the tricuspid in the course of half an hour, what evil consequences might not be expected to follow from the same strain continued over many hours?

The case shows also the advantage that results from abbreviation of the second stage in removing threatening risks from such patients, and it ought to warrant us in abbreviating such a case by art, provided there were present anything approaching to difficulty or delay in delivery.

CASE VI.—*Mitral Obstruction, with Slight Insufficiency—Patient ill for two years with Recurrent Attacks of Palpitation and Pain in the Chest—Sufferings aggravated during Sixth Month of Seventh Pregnancy—Delivery easy—Recovery.*

(*Reported by* MR. WEIR, *my Dispensary Pupil.*)

Mrs. Moodie, aged twenty-six, has had six children and

two miscarriages. All her children are dead, four of them from jaundice. The patient enjoyed tolerably good health till two years ago, when she began to suffer from occasional attacks of palpitation, with pain in the chest, giddiness, breathlessness, and a sense of suffocation. These attacks are wont to come on suddenly with a feeling of languor, last for one, two, or three days, and then go off as suddenly as they appeared. She also suffers from a distressing dry cough. So far as she knows, the patient never had rheumatic fever. In October 1877, the patient was for some time under treatment in the Edinburgh Royal Infirmary, under the care of Dr. Muirhead. She got better after a short residence, but, becoming worse, returned again in the end of November. On the 27th of November she was transferred to Ward XVI., under the care of Dr. Macdonald, where she remained a week. The following is the condition of her circulatory system at that date:—

No regurgitation in the veins of the neck; distinct pulsation in the epigastric region, also in the præcordial region. Apex beat diffused and intensified. Præcordial thrill very evident; auricular thrill only very indistinctly perceptible. Transverse dulness from mid-sternum, 3¼ to 3½ inches; perpendicular dulness normal.

At mitral area, a rough, prolonged murmur precedes and runs up to the first sound. The latter is somewhat intensified, but not entirely clear. The second sound at this space is only indistinctly audible, and follows very closely upon the first sound. At the tricuspid area the presystolic sound is only feebly audible, but the second sound is well heard, and is accentuated; the first sound is less distinct. At the pulmonary area the first sound is somewhat impure, the second accentuated, and frequently reduplicated. At the aortic area the first sound appears pure, the second sound is accentuated, and often reduplicated.

At the auricular area the presystolic murmur is audible, but not so loudly as at the mitral area. The patient has also a very troublesome cough, but examination of the lungs reveals nothing abnormal. At this period the patient believed herself to be in the seventh month of pregnancy. The fœtal heart's beat could be distinctly heard. The patient is pale and somewhat anæmic, and is also of a highly nervous disposition. The treatment adopted was digitaline granules, dialysed iron, rest and care, with a dose of bromide of potassium at night.

It is to be noted that in the record of her case taken in October, the second sound in the aortic area was observed to be feeble, and the transverse dulness towards the right very slightly increased.

She left the Infirmary in the beginning of December, after which the cough and dyspnœa got rather worse. The sputum was noticed to be streaked with blood on the 15th December. On the 7th of January 1878 she felt very poorly, in consequence of some extra exertion. She complained of recurrent attacks of syncope, and there were moist sounds heard over the base of the right lung. There were some pains in the abdomen and back, but no evidence of the commencement of true labour pains.

On 31st of January she sent for me at 4.30 P.M., believing herself in labour. The cervical cavity was found to be obliterated, but there were scarcely any labour pains. The patient was suffering much from dyspnœa, giddiness, headache, and pain in the cardiac region. Dr. Macdonald and Dr. Playfair saw her at 11 P.M., and the following note of her condition was then made :—

Slight venous pulsation in the neck. Præcordial thrill well marked, vertical dulness natural, transverse four inches.

At the mitral area a soft systolic murmur is audible, but very little of the presystolic murmur can be heard. In the

tricuspid area the first sound is impure, at the base the pulmonary second sound is accentuated, and so is the aortic second sound. The pulse is 96.

The patient was ordered a belladonna plaster to be applied over the region of the heart, ten minims of tincture of digitalis every six hours, and also a mixture of aromatic spirits of ammonia and of spirits of chloroform as a stimulant.

At 4 A.M., 1st February, Mrs. M. was delivered of a healthy female child. The labour was very easy and rapid, and was finished before I arrived. The placenta was expelled by the natural contractions immediately afterwards. The patient felt greatly relieved after delivery, and made upon the whole a good recovery.

This patient's case is clearly one of mitral stenosis, but not of great severity. A considerable number of the disagreeable feelings experienced by her must be explained by reference to her highly nervous temperament. Still, notwithstanding these facts, one notices the evil effects of the pregnancy in disturbing the cardiac equilibrium, and we are able to watch with tolerable precision the march of its influence by following the history of her physical condition. In October the ward book gives the transverse dulness at the base as very slightly increased to the right; by the end of November it had travelled quite to mid-sternum, and measured $3\frac{1}{4}$ to $3\frac{1}{2}$ inches, whilst at the confinement it had made a further increase to the right, so as to give a transverse dulness of 4 inches. It is also observed that there was at the confinement visible venous pulsation in the neck. Changes indicative of greater tension were coincidently observed in the systemic circulation, for in October the aortic second sound was noted as feeble; in the end of November it had become markedly accentuated, so that the vascular tension had been gradually increasing both in the lesser and in the greater circulation. Co-ordinately symptoms of difficulty of breath-

ing, palpitation, and cough, and of pulmonary œdema became more and more manifest. Evidence of high tension, on the evening when the labour commenced, is seen in the accompanying tracing, No. 6.

No. 6.

We tried several working pressures, but obtained the best results with one of 7 ounces. It required the exertion of a strain of 18 ounces on the spring of the sphygmograph to entirely obliterate the radial pulsations. Independently of these considerations, the tracing itself exhibits characters indicative of considerable tension. The up-stroke is quite perpendicular; the tidal or predicrotic wave, though clearly not so well pronounced as it would have been had the pulse not been so quick (the rate is stated in the report to be 96), is nevertheless in several of the parts of the tracing well developed. The special prominence of the dicrotic wave is most probably to be chiefly accounted for by the rapidity of the pulsations.

The tracing No. 7, obtained the day after her delivery,

No. 7.

presents some peculiar characteristics, and therefore I have included it also. The best working pressure in this instance was found to be 5 ounces, and the obliterating pressure 15 ounces. It will be noticed that the up-stroke is of astonish-

ing length. In this respect the tracing bears considerable likeness to those which we obtained in making some experiments on the effect of the Turkish bath upon pulse tension. Undoubtedly the Turkish bath must quickly and effectively diminish the resistance to capillary circulation, very especially in the skin, but also throughout the body. At the same time, the application of the heat must act as a stimulant to the heart. In this patient's case we have the presence of a high up-stroke with still considerable cardiac force, seeing that the best tracing was obtained by a pressure of 5 ounces, and the obliterating pressure was as high as 15 ounces. It would thus appear that the delivery had in some way brought about very decided diminution in the capillary resistance, without at the same time very markedly weakening the force of the cardiac muscle. This coincidence of character in Mrs. M.'s sphygmographic tracing with that of the tracing of a healthy pulse in a Turkish bath, is extremely interesting, and seems to me to lend important support to the opinion expressed by Dr. Mahomed, that the height of the percussion wave in a sphygmographic tracing is very largely conditioned by the amount of resistance offered by the capillary circulation to the action of the heart.

In comparing such a tracing with that obtained in the Turkish bath, it is always to be remembered that there is introduced a disturbing element, whose effect it is difficult to estimate,—I mean the loss of elasticity sustained by the spring of the instrument, as a result of the high temperature at which it is working. That element would, of course, act in the direction of exaggerating the length of the up-stroke. We did not attempt to estimate the pulse tension in the bath, because the results would have been worthless, and would only have introduced a fresh source of fallacy. Besides, it was next to impossible to work with the metallic parts of the sphygmograph in the great heat of the bath.

But what was the cause of the diminished capillary resistance in the present instance? It seems to me to have probably been partly due to nervous causes, for the patient's nervous system was specially peculiar, but essentially referable to the changes in the circulation which were brought about by the delivery. It bears out, but in an exaggerated degree, the opinion that we arrived at as a result of the facts considered and compared in Chapter II., viz. that delivery does diminish to a certain amount the general arterial tension, without, however, reducing the action of the heart to the normal standard. I do not quite see why the presystolic murmur should have disappeared in this patient's case during the labour, as, though it occasionally vanished during the pregnancy, it was tolerably steadily present. It is well known, however, that this murmur is apt to come and go. Its disappearance could not be due to loss of cardiac force, for the pulse tension was good. It seems rather to have been due to some change in the nervous supply to the heart. Its disappearance, along with the persistence of good force in the pulse, would seem to support the view that there was only a limited degree of stenosis in this patient's mitral.

Again, the presence of the systolic murmur during the delivery could hardly be considered entirely due in Mrs. M.'s case to dilatation of the right ventricle, and consequent tricuspid insufficiency; for the murmur was very imperfectly heard over the tricuspid area, and much better over the mitral. The first sound, throughout the period during which the patient was under observation, was never quite pure. As, however, we have evidence of rather acute dilatation of the right side of the heart, it is possible that a certain amount of this soft systolic murmur may have originated at the tricuspid orifice.

Though the labour was got over without much discomfort, the symptoms from which the patient was suffering on the

night previously clearly demonstrate that had there been any unusual, or even had there been the usual difficulty during the second stage, the consequences might have been serious.

I think, also, some of the immunity from danger which fell to the lot of this patient, as well as of Mrs. T., Case IV., and of Mrs. C., Case V., was due to the careful measures taken to avoid all possible sources of danger to the patients. These measures were observed, because we had, before they came to be requiring treatment, greatly improved our knowledge of the possible risks they ran, as well as of the necessary precautions to be observed in their management.

These good results encourage us to believe that judicious and skilful management of similar cases, both throughout the pregnancy and at delivery, might greatly lessen the risks associated with lesions of this nature.

CASE VII.—*Mitral Stenosis—Miscarriage about Fifth Month, and Death thirty-six hours afterwards.*

(*Communicated to me by* Dr. JOHN LINTON.)

I was first called to see Mrs. I., six months previous to her marriage. She was then about twenty-one years of age, was suffering from a dry cough and breathlessness, and was unable to walk for any distance, even on a level pavement, without feeling more or less out of breath. The breathlessness was sensibly increased on ascending an incline, and very markedly so on going up a stair. On examination, I found her suffering from mitral disease — presystolic. Being a lady of good personal appearance and affable manners, I inquired whether there was a prospect of marriage, and being answered in the affirmative, I considered it my duty to warn the parents and the gentleman to whom she was engaged, that the consequences might be disastrous. The announcement of the nature of the disease took the

friends somewhat by surprise, and I suggested that the late Dr. J. W. Begbie should see her. He confirmed my diagnosis, and gave advice very similar to mine, saying that, though not necessarily certain of a fatal termination, still there was a very great risk involved in the proposed matrimony. As usual under similar circumstances, the physician's advice was not followed, and the marriage took place. For two months or so the patient was well, but on becoming pregnant symptoms of increased breathlessness now manifested themselves. These continued to get gradually worse as pregnancy advanced, until after the fourth month, when she could lie down neither day nor night, and seemed in the greatest possible distress. Miscarriage then took place, and death followed in about thirty-six hours afterwards.

We have in Dr. Linton's case evidence of mitral stenosis in which the compensation had begun to be very distinctly interrupted before the commencement of pregnancy. But the introduction of that condition, with its disturbing influences, upon the condition of the heart and circulation, speedily aggravated matters, and led to an interruption of the pregnancy and a termination of life about the middle of the ordinary period of utero-gestation. It will be noticed that the severe symptoms came on about the middle of pregnancy. This peculiarity was pointed out by M. Peter and others, and is an undeniable fact. Whether or not the true explanation of the occurrence of these symptoms at this period of pregnancy, is to be found in the assertion that the increased vascular tension due to the physiologically enlarging ventricle is the main cause of the disturbances, cannot as yet be held to be fully established. In favour of this view being the correct one, it may be said that it affords at least a rational explanation of the disastrous influences introduced by utero-gestation into a case complicated with heart lesion of a severe form. If we assume (which we are usually correct

in doing in such cases) that we had here a degree of incompetency of the mitral as well as severe stenosis of it, it is quite clear that an hypertrophied left ventricle could only aggravate matters, already sufficiently bad, by pumping back with increased force upon the pulmonary circuit a portion of the blood that each time struggled through the narrow mitral orifice. If, then, that narrowing of the orifice of itself kept the left auricle and the pulmonary veins distended with blood, and if by dilatation and weakness of the right heart, the latter not being co-ordinately strengthened by hypertrophy during pregnancy, the blood was imperfectly pushed forwards from behind, it is easy to see that a disastrous degree of pulmonary congestion, leading up to the gravest results, might be produced. In point of fact, suffocative œdema of the lungs, congestive bronchitis with or without actual pulmonary hæmorrhage, extreme dyspnœa, and cardiac irregularity, culminating in abortion and death, either during or shortly after delivery, form the sad circle of phenomena we are bound to expect in a pregnancy complicated with a badly compensated mitral stenosis. These are facts established beyond dispute, and I think, for the credit of the profession and the good of humanity, they cannot be too strongly emphasized. I can scarcely too highly estimate the wisdom, conscientiousness, and sense of honour exhibited by Dr. Linton, in endeavouring to interfere with the mournful sequence of events at a time when, if his advice had been followed, he would have benefited his patient, and saved this poor lady, and especially her friends, a great deal of trouble and sorrow. It is painful to think that, though duly warned against it, the imprudent step was taken. I think it is to be regretted that Dr. J. W. Begbie did not more decidedly support Dr. Linton in the case; and the hesitancy exhibited in his opinion seems to me to establish the necessity of laying definite and well-matured views in regard to this

subject before the members of our branch of medical science and practice. Had Dr. J. W. Begbie known that this cardiac lesion was certain to be so very fatal when it arose in connection with pregnancy, he would have given a decided and emphatic negative to the proposal of marriage, and thus might have led to its being put off, at any rate, and have saved the friends from being parties to this young lady shortening her days.

The account of the physical signs is somewhat brief in this record, so that it is difficult to tell what was the direct cause of death. But, from what we know of other cases, I do not hesitate to say it was most probably due to suffocative pulmonary œdema. And here, as remarked already, one of the most curious points is, that though the labour was completed without very much distress, the danger to life was not thereby removed. This, it seems to me, leaves little ground for recommending premature labour in cases in which there exists no abnormal abdominal distension.

CASE VIII.—*Mitral Stenosis—Breathlessness from age of Fifteen—Symptoms much worse after Marriage—Miscarriage about Fourth Month of Pregnancy, and Death immediately after—Post-mortem—Great Mitral Contraction—Both Auricles and Right Ventricle dilated and hypertrophied—Small Aorta.*

(*Extracted from Bennett's 'Clinical Medicine,' Case* CXXII. *p.* 585, *edition* v.)

Ann L——, at age of fifteen, began to be breathless on exertion, and to suffer from a troublesome cough with hæmoptysis. At age of nineteen she was admitted into the Royal Infirmary, Edinburgh, in May 1859, when she was found to suffer from symptoms of mitral stenosis and bronchial catarrh. The presystolic murmur was loud. She got better, and married in December 1859, but, in conse-

quence of cardiac and bronchial symptoms, was readmitted on 13th February 1860. She improved somewhat, except that she had severe hæmoptysis (℥ xxxij) on one occasion, till the 5th of May, when she miscarried of a four months' fœtus, and died immediately thereafter.

Post-mortem examination showed the right heart much enlarged, both auricles and the right ventricle dilated and hypertrophied. Left ventricle normal and thinned. Mitral orifice admitted only the tip of the little finger. Aorta smaller in calibre than the pulmonary artery, which was somewhat dilated. Uterus quite healthy.

We have here an example of cardiac disturbance commencing at the age of fifteen,—that is, it appears to me, becoming obvious at the time when menstruation became established. It is fair to suppose that up to this time the lesion, though most probably present, was so in a slight degree, and was entirely mute. There is in this case no history of an acute rheumatic attack, although evidence of a rheumatic diathesis is shown in the progress of the case, but, to economize space, I have not noticed it in my abstract of the record.

We therefore would seem in this patient's case to have to deal with a cardiac lesion originating in early life, if it had not been congenital, becoming worse, and that very manifestly, with the establishment of menstruation, and finally leading to the most disastrous consequences about the middle of pregnancy. The small left ventricle and the dilated auricles, with narrow aorta and widened pulmonary artery, are very characteristic of this special cardiac lesion. It would appear that here excessive action of a powerful and hypertrophied right ventricle, spending its strength against an extremely narrow mitral orifice, had more to do with the resulting pulmonary engorgement than any regurgitant action of the left ventricle, which, moreover, was very small

and thin. Such a case abundantly proves the risk that is incurred by pregnancy under such conditions, and also another risk that I have not distinctly alluded to, although it is abundantly insisted upon by M. Peter,—I mean the danger of exposure to cold. If such patients will get married whether we consent or not, then they ought to be kept as quiet as possible during pregnancy, and should be advised to avoid every influence that is likely either to embarrass or disturb the circulation directly or indirectly. The onset of bronchial catarrh is one of the most dangerous indirect influences in such cases, and therefore its exciting causes especially ought to be avoided.

Under no conditions is good nursing more needed, or likely to be better rewarded, than in warding off the exciting causes of pulmonary disturbances in connection with pregnancy complicated with various cardiac lesions.

CASE IX.—*Extreme Stenosis of Mitral—Violent Palpitation and Severe Breathlessness for Three or Four Years—Death from Pulmonary Engorgement immediately after an Easy Labour, being the Patient's Fourth—On Post-mortem Examination, Lungs found healthy, but gorged with Blood—Mitral Valve only admitted tip of Little Finger—Heart small.*

(*Extracted from Ramsbotham's 'Midwifery,' p.* 509, *footnote.*)

I was requested by an old pupil to assist him in investigating the cause of death in a patient aged twenty-eight, who suddenly expired immediately after having given birth to her fourth child. She had been for three or four years subject to violent palpitations, and much difficulty of breathing on the least exertion or when walking up stairs. She had constant cough, and occasionally expectorated small quantities of blood. My friend was not called until the os

uteri was entirely dilated. The labour was unusually easy. The child was born an hour after he entered the room, and the same pain which expelled the breech also threw off the placenta. She appeared not to have suffered much from fatigue, and inquired concerning the sex of the child. While, however, her attendant was tying the funis, he observed that she was attacked with a slight convulsion, and before he could get round to the side of the bed near which her head lay, she had ceased to breathe. The uterus was firmly contracted, and contained a very small quantity of coagula. The viscera of the abdomen were remarkably healthy. The lungs were healthy in structure, but gorged with blood. The heart was small and very flaccid. The mitral valve was much thickened, and the communication between the left auricle and ventricle would only just admit the end of the little finger. There were about five ounces of serum in the pericardium.

The statements in this case are too brief to prove anything further than that the patient had extreme mitral stenosis, from the evil effects of which on her pulmonary circulation she had suffered for three or four years, and finally died suddenly after an easy labour. It is to be regretted that the brain and lungs were not examined for embolism or apoplexy, although there is little reason to believe that either condition would have been found.

But from the report we are very far from being able to answer the question why this patient suddenly died. I would here remark that, from what we know of the invariable sequence of a prolonged mitral stenosis, the auricles, more especially the left, must have been dilated, whatever was the thickness of their walls. The pericardial effusion alone would scarcely account for the suddenness of the fatal issue, though it doubtless partly contributed thereto. If the mere stenosis was so intense as to give rise to death

by backward congestion of the lungs, then one would have expected more threatening symptoms during the labour, when such congestion could scarcely fail to have been aggravated by the increased vascular tension in the thorax during each pain, and especially during each down-bearing pain, and not after the labour had been terminated, and apparently with so much ease. There is one possibility which our knowledge of what occurs occasionally in such labours suggests, and which the absence of very strict statements regarding the case does not exclude, and this is, that during this patient's delivery partial unconsciousness on her part may have led to the serious symptoms being unobserved by her attendants. It may also have been the case that the heart was so fattily degenerated or otherwise enfeebled that it was entirely exhausted with the extra efforts of an easy labour, and suddenly ceased to beat, like a worn-out horse which carries its master out of the reach of danger to his own door, and then drops down dead. But of this we have here no exact information, in consequence of the meagreness of the record. In such cases Spiegelberg's idea of suddenly disturbed relative pressures would apply, if we could only be satisfied of its correctness. It is just possible that here the cause of death was due to such an amount of fulness of the left auricle and of the right heart, that the weakened right heart was overpowered, and death followed in the condition of diastole. But the reporter has not told us whether the heart did or did not contain much or any blood. We are merely told that the lungs were gorged with blood, and that the heart was small and very flaccid, which certainly is not what one would expect, except in so far as the left ventricle is concerned, for it is well known that this lesion leads usually to diminution of the left ventricle, with dilatation of the other chambers. It would appear, also, that the patient lived some little time after the removal of the

placental circulation, the period when, according to Spiegelberg's views, the sudden death ought to take place; for it is stated that the patient inquired respecting the sex of the child, whilst it had been stated above that the placenta was expelled along with the breech of the child. Thus it appears to me that, even granting that at that moment sudden diminution of aortic pressure did take place, which, as already stated, I hold has not been proved to occur, it could scarcely have been operative in the present instance. Pericardial effusion and over-distension of the right heart, and practical cardiac paralysis therefrom, with consequent pulmonary engorgement, would appear, therefore, to have been the main elements that hurried on the death. It is possible, however, that this death may have resulted from embolism of the cardiac arteries, which is occasionally the cause of sudden termination of life in connection with mitral stenosis.

CASE X.—*Stenosis of the Mitral of moderate amount—Severe Chest Symptoms (Œdema and Congestion of Lungs) during the Latter Months of Second Pregnancy—Premature Labour suggested, but came on spontaneously before arrangements could be completed, and Patient died during Confinement of Suffocative Œdema of Lungs—Post-mortem proved Lungs intensely Œdematous — Heart ordinary size—Aorta small—Pulmonary Artery dilated—Mitral Valve contracted.*

(*Abridged translation from 'von Hecker und Buhl,' S.* 173. *Leipsig,* 1861.)

Patient, twenty-four years old, admitted in the ninth month of her second pregnancy. Previous labour easy. Had suffered from breathlessness for some months, and spat blood three weeks previously to admission. On examination, lips cyanotic, respirations 64 per minute, and very difficult; pulse 120. No pulmonary dulness, but loud, sonorous râles

everywhere audible. A weak presystolic murmur audible with heart's sounds. Acute œdema of lungs diagnosed, and arrangements made to effect delivery by artificial means so soon as practicable. Patient slept well on night after admission, and seemed better in the morning; pulse 100, respiration 40. But the following evening, at 6.30 P.M., there was a frightful exacerbation of all the symptoms of dyspnœa, the liquor amnii escaped, and the patient was carried into the delivery room, but died in half an hour of suffocative œdema of the lungs, the pulse keeping as low as 100, and the convulsive efforts at breathing being extremely violent, whilst copious quantities of frothy mucus escaped from the mouth. Cæsarean section was performed two minutes after death, and a dead, nearly mature child extracted.

Post-mortem examination revealed intense congestive œdema of the lungs everywhere, partial consolidation at base of right lung, which sank in water. Heart ordinary size; aorta strikingly small; pulmonary artery somewhat dilated. The left auriculo-ventricular valve was narrowed into a circular opening, which still allowed a man's finger to pass. On its auricular aspect a number of fine warty vegetations were found.

This case is one of those in which the disturbance due to labour pains and the resistance offered by bronchitic obstruction to the circulation, throw a heart with a badly compensated mitral obstructive lesion into such perturbation, that a fatal result is precipitated from acute pulmonary suffocative œdema of the lungs. Such is a very common result in cases of labour complicated with cardiac disease. It is again to be noticed how commonly in these cases labour comes on prematurely. This case is of especial interest, as it is one of the earliest well-described cases, and was largely the means of directing the attention of obstetricians to this important

subject, as I have already pointed out in speaking of Hecker's original contributions to its literature.

CASE XI.—*Mitral Stenosis with Insufficiency—Dilatation of the Right Side of the Heart—Severe Chest Symptoms (Œdema of Lungs with Bronchial Congestion)—Symptoms during Labour very critical—Venesection—Liquor Amnii excessive—Recovery.*

(*Abridged translation from Spiegelberg, 'Archiv für Gynäkologie,' Bd.* ii. *S.* 241.)

This patient was thirty years old, and had previously been pregnant, when she was delivered naturally, and had enjoyed good health till the middle of the present pregnancy. On admission she believed herself near the termination of her second pregnancy. Her sufferings included a tormenting cough and great breathlessness. Her appearance was cyanotic; pulse small, soft, and rapid. Œdema of the lower limbs and of the abdominal walls was present; uterus also was much distended with liquor amnii. This the patient had noticed to increase of late very rapidly, and there was a little ascites, but no albuminuria. Examination demonstrated the liver to be pushed up to the level of the fifth rib, widespread sonorous and sibilant râles over the chest, emphysema of border of lungs, and insufficiency with stenosis of the mitral valves, with slight hypertrophy and manifest dilatation of the right heart. Small doses of digitalis and stimulants were administered, with relief of the subjective symptoms. Labour supervened some days afterwards, and along with it the chest symptoms attained a most critical height, so as to necessitate venesection. With the discharge of the excessive liquor amnii, however, they abated quickly, and after the labour they completely remitted, and the lying-in period was passed without further disturbance.

We have here a case of mitral stenosis, not pure, but, as

usually happens, combined with insufficiency of the valve. It has most of the usual peculiarities. However the lesion had arisen, it did not manifest itself till the usual period, viz. till past the middle of the pregnancy. The usual routine symptoms of cough, dyspnœa, bronchitic râles, cyanosis, and palpitations were present in increasing intensity as the pregnancy advanced; and as the over-distended uterus pressed more and more upon the thorax, they attained a maximum of threatening intensity on the occurrence of labour. Such a case is exactly of the kind likely to be benefited by premature labour. In support of this opinion, it is only necessary to notice the very marked abatement of the symptoms that followed upon the escape of the excessive liquor amnii.

It is interesting to notice here that Spiegelberg places greatest reliance in a somewhat heroic treatment, including venesection and purgatives, and that by blood-letting and antimony M. Peter rescued his famous case after the other physicians had given the lady up for dead.

In this patient's case there is reason to believe that, though the compensating arrangements in the heart were seriously interfered with, and were very unequal to the extra demands upon the organ consequent upon pregnancy, and particularly with a uterus in a condition approximating hydramnios, yet they were sufficiently good to support a tolerably regular circulation when once these two disturbing forces were removed. It forms also a very confirming proof of the injurious effects which a pregnancy introduces into the condition of a patient who suffers from chronic cardiac disease, even when the lesion is nearly mute under ordinary circumstances.

CASE XII.—*Mitral Stenosis with Insufficiency—No Special Trouble before the Sixth Month—Then Breathlessness,*

*Palpitation, Cough, Expectoration of Rusty Sputum, and occasionally of Blood—Labour Premature during Seventh Month, with Unconsciousness and extremely Alarming Symptoms—After this, Remission of urgent Symptoms—Death Nine Months afterwards with Bright's Disease—On Post-mortem Examination: Pericardium distended with Fluid—Heart twice the ordinary size—Both Auricles and Right Ventricle greatly dilated; Left Ventricle natural size—Left Auriculo-Ventricular Opening admitted only one Finger—Lungs partly collapsed, partly emphysematous and œdematous.*

(*Abridged translation from Fritsch,* '*Archiv für Gynäkologie,*' *Bd.* x. *S.* 272.)

Patient twenty-four years old. Chronic invalid. Experienced no extra trouble from her pregnancy till the *sixth* month, when her cough became excessive, expectoration purulent, and every kind of exertion set up palpitation. She also suffered from gastric catarrh, loss of appetite, sleeplessness, and dyspnœa. On admission, 4th October 1871, Professor Olshausen examined her, she being then in the seventh month of her pregnancy. He found slight cyanosis of the lips and eyelids. Cardiac region somewhat arched forwards from the second rib downwards. Cardiac impulse visible, and causing tremor of almost the whole mamma. Cardiac dulness begins on left at lower border of second left rib; is absolute over the third. Inferiorly it is impossible to ascertain its amount, owing to the mamma. In the third intercostal space, the transverse dulness reaches from the edge of the sternum to the mammillary line, and measures 3½ to 4 inches. Præcordial thrill present everywhere, weak but distinct. Apex beats most distinctly in the fifth intercostal space, a finger-breadth outside the mammillary line. At the apex a prolonged rasping murmur and a weakened heart sound. Over the aorta

both hearts' sounds audible, the pulmonary second sound intensified.

The diagnosis made was—insufficiency and stenosis of the mitral, with hæmorrhagic infarction of the lungs. There were coarse moist râles over the whole breast, and a limited dull area inside the left scapula, with weak breathing there and crepitation. The radial pulse was feeble, but only quite seldom was there a smaller or missed beat. Œdema of both legs.

The patient, with good nursing, got on tolerably well till her confinement. Only at times the cough and expectoration increased so as to require treatment with morphia and expectorants. The sputum was frequently rusty, and often contained slight admixture of pure blood.

On the 9th of October an attack of limited pneumonia of the right lung came on, which was treated with digitalis, and the patient improved a little. In the night of the following day violent cough with shortness of breath set in, and the labour commenced. The patient's countenance was strikingly pale and cyanotic. The pains were weak, yet the labour progressed with surprising rapidity. Notwithstanding, the patient was now on the point of death. The pulse was slow, small, extremely irregular, and intermittent. Unconsciousness came on. The forceps were then applied, and the labour easily terminated. The placenta was expelled by the natural efforts. Immediately after delivery the patient got very much better; she awoke as if from deep chloroform narcosis. The pulse became quicker. There was no post-partum hæmorrhage. The patient improved, and was dismissed on the 25th October. But shortly after that she again grew worse, partly owing to her circumstances in life, which were very wretched and depressing. The œdema increased and extended to the whole body. Albumen appeared in the urine, and the patient died of Bright's disease on the 11th July 1871.

The *sectio* revealed the pericardium greatly distended, reaching transversely from 1 to 1½ inch on the right of the sternum to the axillary line on the left, and perpendicularly from the second to the ninth rib. Its cavity contained a large amount of clear serous exudation.

The heart was at least doubled in all its dimensions, tensely filled with dark blood.

The left auriculo-ventricular opening admitted one finger; both cusps remarkably diminished, their free edges thickened, swollen, tolerably smooth, the chordæ tendineæ very much shortened and yellow. The other valves completely free. The muscular tissue in the right ventricle somewhat thickened, generally pale; in the left ventricle plainly spotted yellowish. The cavity of the left ventricle of normal size. Both auricles and the right ventricle greatly dilated.

Left lung of normal size, somewhat dense, its upper lobe slightly emphysematous; in the lower lobe there is a considerable portion almost completely empty of air, loosely infiltrated, of a slightly granular palish red, moist section. In the bronchi, frothy fluid. The right lung was slightly adherent; its upper lobe very voluminous, dense, almost completely empty of air; its cut surface brownish-red; distinctly granular. The infiltration involves the upper part of the middle and lower lobes, which latter appear principally filled with air, but œdematous.

Both kidneys were hardened and slightly injected; cortical substance increased; yellowish coloured; shining (not amyloid); much fatty degeneration of the tubuli uriniferi.

In this particular case we find the presence of heart disease accompanied with most of its baneful effects upon pregnancy. We have nearly the same story in all these cases. The patient gets on well till the fifth or sixth month; then comes breathlessness, palpitation, pulmonary hæmorrhage, and so forth, in ever-increasing amount, till spontaneous

interruption of the pregnancy occurs. But the delivery in this patient's case proved more favourable than it is wont to be, on account, no doubt, of the fact that the stenosis, though serious, was to a considerable amount compensated by a powerful right ventricle, which was able to maintain the circulation through the lungs notwithstanding the difficulty which the blood current experienced in passing the block on the left side; but it would appear that, so soon as the obstruction to the systemic circulation owing to the renal disease was supperadded to that in the pulmonic circuit arising from the stenosed mitral, the heart was no longer able to maintain the balance of the circulation. It is not impossible that the renal condition arose in consequence of the cardiac disease, as so frequently happens. This is particularly likely to occur when cardiac disease complicates pregnancy, owing to the general predisposition to irregular vascular changes associated with pregnancy.

It is to be noticed that in this case and in others observed by Fritsch there occurred an astonishing degree of unconsciousness during labour. This I have not been able to notice except in the case of Mrs. C. (Case III.), and then it was present only to a small amount; but as several of our cases had chloroform during the difficult part of the labour, it is possible that this condition may have occurred oftener in the cases observed by me and my friends than we have been able to record.

It is surprising how easy the labours are in those cases of severe cardiac complication, and yet how extremely irregular the pulse is apt to become during it. The condition of both auricles in this patient's case is worthy of notice, as being characteristic of the special lesion under consideration.

CASE XIII.—*Mitral Stenosis—Slight Attack of Acute Rheumatism after Second Confinement, followed by Severe*

*Illness, involving Heart and Kidneys—Admitted in Seventh Month of Third Pregnancy, with Symptoms referable to Mitral Stenosis with Retro-dilatation and Venous Congestion — Induction of Premature Delivery thought of towards end of Eighth Month, but Labour supervened spontaneously, Patient being in a state of Unconsciousness—Death three months afterwards — Post-mortem: Great Dilatation of Right Heart, also of Left Auricle—Slight Enlargement of Left Ventricle — The Mitral Opening much contracted—Valves thickened—Evidence of Recent and Old-standing Pulmonary Infarction—Hydrothorax.*

(*Abridged translation from Fritsch,* '*Archiv für Gynäkologie,*' *Bd.* x. *S.* 277.)

This case came under treatment in the seventh month of the third pregnancy. Patient, after her second confinement, had an attack of acute articular rheumatism, confined, however, to the shoulder joints, but had not been kept in bed by it. Fourteen days after this seizure, she had to take to bed for disease of the heart and kidneys. All her present symptoms point towards a mitral stenosis, with considerable retro-dilatation and venous congestion. Both legs are enormously œdematous; the vulva is so swollen that the patient can only lie with the legs widely separated; the œdema has extended up to the hypogastric region; the jugular veins are considerably enlarged and undulate violently. The patient is so short of breath that she can only sit in bed and speak in broken sentences. The sputa are frothy, without blood and pus, but blood has been expectorated from time to time. The patient complains of nausea, frequent vomiting, tormenting headache, and of attacks of unconsciousness and giddiness. Over the entire lungs small and large moist sounds are audible; bronchial breathing is heard at the lower edges of the lungs, before and behind; cardiac dulness begins on the left with the third rib, passes outwards a little beyond the

nipple line, and extends to the right two finger-breadths beyond the right edge of the sternum; the cardiac impulse consists in an irregular heaving tremulous motion of the lower half of the left thorax; the undulation extends beyond the xiphoid process on to the abdomen; both sounds of the heart are changed into loud grating murmurs; the second pulmonary sound is intensified; much albumen and pus corpuscles, but no tube casts are present in the urine.

The patient seemed so ill that premature labour was thought of and discussed. Suddenly, however, the membranes broke, and a large quantity of water escaped. On examination immediately afterwards, the cervix was found still persistent; but in the course of the night labour came on when the patient was alone and in a state of unconsciousness, and the child was born without assistance from any one. The child was fully four weeks before the full time. The patient got slightly better for a few days; thereafter disease of the kidneys appeared; the œdema, cough, and dyspnœa increased; hydrothorax, diarrhœa, and finally discharge of blood from the bowels came on. Her death occurred about three months after delivery.

Post-mortem examination, twenty-four hours after death, revealed the following condition: Œdema of the whole lower half of the body present. In the abdominal cavity there is 1½ litres of ascitic fluid. The pericardium lies widely exposed, reaches on the left to the nipple line, and on the right at least 1⅜ of an inch beyond the right edge of the sternum. It contained a large amount of serous fluid. The heart is without apex, globular, and double the ordinary size. The left auricle is decidedly dilated, the mitral valve contracted; its orifice does not admit one finger. The segments are much smaller than ordinary, thickened and knotty, and the chordæ tendineæ shortened. The musculi papillares are thick and short. Left ventricle slightly enlarged; greatest

thickness of the ventricular cone 1·2 cent. Muscular tissue soft, brittle, anæmic, and fatty. Right auricle and ventricle enormously dilated. The thickness of the latter measures not much more than two millimetres (0·079 of an inch), and both the venæ cavæ are greatly distended. The tricuspid valve is unaffected. Both kidneys are small and pale. Cortical substance diminished. On the surface of the kidneys small extravasations of blood visible. The renal tissue is strikingly hard.

All the bronchi are intensely reddened. There are in the lungs apoplexies, infarctions, and cicatrices of old standing, and recent infiltrations. Some portions are compressed. Hydrothorax is present.

In this interesting case we have again serious symptoms becoming more pronounced after the middle of pregnancy. There is the usual thinning and dilatation of both auricles, with great dilatation and thinning of the right ventricle. That the left ventricle was rather enlarged than diminished in size, may be due either to the natural tendency towards hypertrophy of that cavity of the heart during pregnancy having overcome the tendency of the special cardiac lesion present to lead to diminution in the size of the left ventricle, or it may have been because the case was a comparatively acute one, in which the chronic effects of stenosis of the mitral had not had time to become so pronounced as they would have been had the patient lived longer with the lesion operative. This case, unlike most of the examples of stenosis with which we have had to deal, presents an unmistakeable history of acute rheumatism coming on after the second delivery. It also illustrates the greater tendency for pregnancy to act injuriously upon a heart which suffers from a comparatively recent lesion, in which the compensation has been but very imperfectly established before the commencement of the pregnancy.

We have also premature labour coming on spontaneously, but with little, and that too only transitory, relief to symptoms,—a result which, as we have on several occasions observed already, throws a considerable amount of doubt upon the propriety of inducing labour under such circumstances; for if, when the delivery comes on naturally, the prognosis for the mother is scarcely if at all bettered thereby, it follows that the irritation and annoyance necessarily associated with every method of inducing labour artificially must be expected to increase the mother's risks. Still, the excessive amount of liquor amnii would, it appears to me, have justified the induction of labour in this particular instance. I am inclined to think that, in some of those cases, the premature labour is referable, as in our Case III., to retro-dilatation of the vessels leading to separation of the placenta; but this can only be applicable to a few of them. It would rather appear that, as a general rule, labour supervened co-ordinately with, and as a result of, imperfect nutrition of the brain and cord, resulting from the imperfect aëration of the blood. The latter results would arise from the great amount of the pulmonary congestion and œdema present. If it be true, as is rendered more than probable by the investigations of Frankenhaüser and Obernier, that the inhibitory centres of the uterus are situated in the cerebro-spinal nerve system, we have merely to assume that the cerebral changes in those severe cases attained such a height as to paralyse the inhibitory centres of the uterus, and thereby lead to the onset of labour prematurely.

That nervous disturbances have much to do with the onset of premature labour in connection with severe heart disease, is, I think, rendered further probable by the occurrence of more or less complete unconsciousness during the delivery in several of the cases.

CASE XIV.—*Mitral Stenosis and Insufficiency with Aortic Insufficiency—Enlargement of the Left Side of Heart, consequent upon an Acute Attack following the Tenth Pregnancy; which was a Placenta Prævia—Aggravation of Symptoms during the latter half of the Eleventh Pregnancy—Spontaneous Premature Labour in the Eighth Month, whilst Patient was in a state of Unconsciousness—Cardiac Symptoms much aggravated therewith, and Death forty-eight hours afterwards—Post-mortem: Enlargement of Left Heart—Left Auriculo-ventricular Valve thickened and nodulated—Opening much contracted—The Right Heart only moderately dilated.*

(*Abridged translation from Fritsch,* '*Archiv für Gynäkologie,*' *Bd.* x. *S.* 280.)

Patient, aged thirty-nine, had given birth to nine children living, at full term. Tenth pregnancy was complicated with placenta prævia, and delivery was completed by *accouchement forcé*, accompanied with much hæmorrhage. All went well for some days. On evening of 5th patient had a rigor, and was seized with a stitch in the side and pain in breathing. Examination next day detected pleurisy involving the lower part of the left lung posteriorly, as also pericardial friction and peri- and para-metritis. In the right calf and left thigh a phlebothrombosis and periphlebitis were developed. Pulse was 100, and temperature 105·8°. Under treatment with hydropathic packs, digitalis, quinine, and purgatives, all the symptoms except the cardiac subsided; yet the temperature continued at 104°, and the pulse remained rapid. At base and apex of the heart a distinct murmur was heard instead of the first sound, which also somewhat masked the second sound. This was regarded by Professor Olshausen as endocardial in origin, whilst Dr. Fritsch was inclined to look upon it as anæmic. The patient gradually got better, but the heart murmur persisted, and she began to suffer from

palpitation and dyspnœa. In the summer of 1874 the collective symptoms of a badly compensated mitral lesion were found present. Heart's perpendicular dulness reached from the second to the seventh rib on left side, and the transverse from the nipple line to quite beyond the right border of the sternum. Both cardiac sounds changed into murmurs, especially the loud, grating murmur of the first sound, were audible everywhere. Pulmonary second sound not essentially intensified. Slight œdema of the ankles. Thus evidence of undoubted stenosis of the mitral with insufficiency was established. The clinical history pointed to this being the result of the acute rheumatism that followed the tenth confinement, rather than that it was due to the lighting up of some antecedent chronic lesion. But whether it arose as part of the pleurisy and pericarditis, or resulted from the phlebitic disease, and was thus of the nature of ulcerative endocarditis, it was impossible to determine with certainty.

Treatment was directed towards mitigating the most urgent symptoms. These were dyspnœa and oppression.

In the summer of 1875 the patient was again seen. She was now in the fifth or sixth month of pregnancy. During the first three months she had felt better than during the last year. But now her condition had become very bad, and the palpitation and dyspnœa had greatly increased. The medicines prescribed were of little use; the condition got constantly worse. The œdema, however, did not pass above the lower extremities. After another six weeks the patient's husband brought Dr. Fritsch a fœtus of about eight months, which had been born dead during the previous night. He reported that his wife had become suddenly restless during the night, and when spoken to had returned no answer; that he himself then fell asleep again; that after some time his wife had uttered a loud groan, and when he looked at her, she had

become unconscious; that a female neighbour, on being called in, had raised the bed-clothes to rub the patient's legs, and discovered the dead child in the bed. He had seen no blood. His wife had gradually come to herself again. Fritsch, on arriving at the patient's house, found her now on the point of death. The pulse was not to be counted. Most of the beats were quite small, but suddenly there would occur a largish pulse-wave; at times the pulse stopped entirely for three or four seconds. The heart hammered against the breast wall with extreme irregularity. The patient was quite conscious, and believed she was going to die. Her lips were cyanotic, the visible skin and mucous membranes were pale. Urine discharged clear in colour, and large in quantity. Loose stools were passed unconsciously. The patient died forty-eight hours after labour.

Post-mortem showed œdema of both legs. Little serous effusion in either abdominal, pleural, or pericardial cavities. The heart was manifestly enlarged. The enlargement was confined almost exclusively to the left side of the heart. The heart extended on the left beyond the nipple line, and perpendicularly from the second to the seventh rib. On the right it terminated about the right border of the sternum. The vasa propria of the heart were distended to the size of the little finger. The left auriculo-ventricular opening was contracted and insufficient, but one finger could still pass it. The cusps were nodulated at their edges; otherwise smooth, and formed into a cartilaginous ring. Pericardium in the neighbourhood was reddened in its deeper layers. The aortic valve was incompetent, all three cusps being altered. Left auricle and ventricle were enlarged to twice the natural size. The wall of the ventricle not thinned—thickest part 1·4 cent. Right heart only moderately dilated.

The kidneys were large and congested; the cortical substance increased.

We gather from the history of this case, that acute endocarditis supervened upon a severe labour complicated with placenta prævia and much hæmorrhage. No better example could be found of the deteriorating effects of pregnancy upon the course of chronic cardiac disease than this record presents. It will be noticed that, notwithstanding many other serious complications, the patient recovered tolerably well, and for nearly twelve months she was decidedly improving. But having the misfortune to become again pregnant, the evil effects of the pregnancy upon the heart disease began to develop themselves in the shape of palpitation and dyspnœa of a most distressing kind.

In its turn the cardiac disease led to interruption of the pregnancy, the labour having come on during the eighth month, and while the patient was in a state of complete unconsciousness. The history of this delivery, as given by the husband, is extremely odd and very interesting. But the usual sequence followed. The patient got no real relief from the delivery, but succumbed within two days, with great œdema of both lungs. In consequence of the aortic insufficiency present in this case, it is easy to explain the great amount of enlargement affecting the left ventricle. The dilatation of the left auricle was no doubt partly due to the stenosis of the mitral, and partly referable to the regurgitation from the left ventricle. The causation of the dilatation of the right heart is too obvious to need demonstration.

The physical signs in this case are given with a somewhat regrettable degree of looseness, so that it is difficult to know exactly what is meant to be implied in them, or how they are to be squared with the post-mortem appearances. After all, however, the history of the case is as interesting as it is curious.

We have thus collected and commented upon a series of

fourteen cases of chronic cardiac disease, in which mitral stenosis, if not always the only lesion, was at least the leading one. The results are now to be grouped together in as little compass as practicable, so that we may form as correct an idea as possible of the effect of this complication.

CASE I.—Patient well till latter end of first pregnancy; in good health after delivery till middle of second pregnancy, which terminated spontaneously at end of eighth month. Mother died suddenly, after six days, of exhaustion and pulmonary œdema; baby, a month afterwards.

CASE II.—Complicated with tricuspid lesion. The patient was seen by us only during her sixth pregnancy. Premature labour came on in the beginning of the ninth month. Death ensued fifteen days after delivery, from pulmonary œdema, hydrothorax, and exhaustion. This patient suffered also from embolic right hemiplegia for seven years.

CASE III.—Safe delivery after most threatening symptoms. Patient hemiplegic and aphasic. As in Case II., it is impossible to fix the precise date of the commencement of the lesion in this patient, though it probably only dates from the immediately preceding confinement.

CASE IV.—Escaped with nothing worse than extreme irregularity of the pulse and cyanosis during the latter stage of a first and a second labour.

CASE V.—Was safely tided over the sixth confinement, though dangerous symptoms supervened during it.

CASE VI.—Presented threatening symptoms during the latter part of pregnancy, but got safely through an easy labour.

CASE VII.—Ended in abortion at the fifth month of the first pregnancy, with death thirty-six hours afterwards.

CASE VIII.—Also ended in abortion in the fourth month of the first pregnancy, and death immediately afterwards.

CASE IX.—Ended in sudden death immediately after an

easy labour. It is not stated whether or not the full term had been reached in this instance.

CASE X.—Patient died during delivery, at about full time, of suffocative œdema of the lungs.

CASE XI.—Presented the most threatening symptoms of breathlessness and palpitation during delivery at full time, but ultimately recovered so far as to be able to leave the hospital.

CASE XII.—Led to supervention of labour at seventh month, and its being finished whilst the patient was quite unconscious, accompanied with the most threatening symptoms; slight relief followed, but the patient died of exhaustion and Bright's disease nine months afterwards.

CASE XIII. and CASE XIV. both ended in premature delivery in the eighth month, and were followed, the former by death three months afterwards, and the latter by death thirty-six hours after delivery, both being accompanied with pulmonary œdema and congestive bronchitis.

We have thus nine cases out of the fourteen, or 64·4 per cent., fatal, which indicates a tendency to death in the combination of mitral stenosis with pregnancy which is surely sufficiently grave, and more especially seeing that there was in none of the cases any purely obstetrical reason, such as pelvic deformity, likely to add additional risk to the delivery.

Of these fourteen cases three were primiparæ, viz. the seventh, eighth, and twelfth, and all of these died.

Four were pregnant for the second time, viz. first, fourth, tenth, and eleventh, and of these four two died; two were pregnant for the sixth time, and of these one died.

The other five were respectively, at the time they were under observation, confined for the third, fourth, seventh, eleventh, and twelfth time. Of these, three died. Of the two that lived, one was a peculiarly mild case, and had a

specially easy labour—her sixth; the other, who was confined for the twelfth time, appears to me to have suffered only a comparatively short time from the cardiac lesion, which at any rate was not of an advanced type.

It will be observed that the deaths occurred usually either suddenly during the labour, or within a few days or weeks afterwards. In one case, life, though thrown into extreme jeopardy during delivery, was prolonged for nine months afterwards. The next longest period after delivery when death occurred was three months, after which we come down to three weeks or a few days. We see also that there is no proved instance of death from embolism among the cases. A very special tendency to abortion or premature labour is clearly demonstrated in connection with all these patients. Very few indeed of them are found to carry their children to the full time. But after delivery there is not a corresponding improvement of symptoms, the reason being, in my opinion, that in addition to the shock and exhaustion inflicted upon the deranged heart during delivery, the evil results of the original disturbed relations of compensation are nearly as powerful during the childbed week as during the latter period of pregnancy.

The frequent occurrence of congestive bronchitis, pulmonary œdema, apoplexy of the lungs, and dyspnœa, is worthy of very serious consideration; but as these symptoms are not special to this form of cardiac lesion, I pass on, in Part II. of this chapter, to the consideration of the other great form of mitral disease.

## PART II.

### CASES OF MITRAL INSUFFICIENCY.

I NOW proceed to the consideration of the following eight cases of mitral insufficiency :—

CASE XV.—*Mitral Insufficiency of slight degree watched through Five Pregnancies.*

Mrs. B., now aged thirty-two, was married on 25th June 1867, and has been five times confined since that period. 1st, on 23d June 1868; 2d, on 19th December 1869; 3d, on 19th March 1872; 4th, on 10th April 1874; 5th, on 23d March 1876. She never had rheumatic fever. When I was first asked to attend her, I found she had a soft systolic murmur at the left apex, which still persists. This was then accompanied by occasional palpitation, slight increase of cardiac dulness, and much pain in that portion of the upper dorsal and lower cervical parts of the spine called by some authors the cilio-cardiac centre. But since that period Mrs. B. has improved greatly in general health, and her pregnancies and labours, with the exception of the last, have been natural, and have been got over without any inconvenience. On the last occasion, Mrs. B. was very sleepless after her confinement, and became on the fourth day delirious; her pulse rose suddenly to about 130, whilst her temperature mounted up to 105°, but apparently entirely independently of the cardiac complication. Matters, however, returned to the normal condition under the use of chloral with bromide of potassium to subdue the nervous symptoms and secure sleep, and of quinine to combat the fever. There was no evidence whatever of septicæmia, nor could any inflammatory lesion be

detected. It is to be added, that the whole of Mrs. B.'s children have been remarkably small, and that in consequence the second stage of labour has been both easy and short in all her confinements.

Mrs. B.'s condition on 1st March 1877 was as follows:—There is only very slight increase in cardiac dulness in any direction. There is still a soft systolic murmur audible at both the mitral and the tricuspid area. The second sound in the pulmonary area is intensified, and the impulse of the heart is somewhat thumping. There is apt to be breathlessness on exertion, and occasional palpitation. But, on the whole, Mrs. B. enjoys good health.

CASE XVI.—*Mitral Insufficiency—Considerable Hypertrophy of Left Ventricle—Watched through Three Confinements.*

Mrs. M. enjoyed good health till she was twenty-seven years of age, when she suffered from a severe attack of acute rheumatism. The fever had two relapses, and she was compelled to keep her bed for upwards of three months. Ever after this illness she was breathless and liable to palpitation on the slightest exertion. In the year 1870 she consulted me on account of these symptoms. I found a loud systolic murmur covering the first sound of the heart at the apex. There was little increase of dulness. The other orifices seemed to be perfectly healthy, and there were none of the other general symptoms of defective cardiac action, such as œdema and cough. The patient was, however, somewhat anæmic, and she profited greatly by the use of iron and digitalis. Before her marriage, which took place on the 8th of December 1872, she consulted me as to the propriety of taking this step. I gave my sanction to the marriage. My judgment rested upon the grounds—1st, that the patient suffered from a purely mitral lesion of insufficiency, which at present was nearly mute and likely to remain so for a long time;

2d, that her intended husband was in such comfortable circumstances that her married life was likely to be an easier one than the life she was leading in her unmarried state. She was accordingly married in December 1872, at the age of thirty-five. She became pregnant, and fell in labour at full term on 24th November 1873. Her pregnancy had been unusually free from discomfort. Her labour was easy, till towards the end of the second stage, when the bearing-down efforts became so severe, that to avoid injury to her heart I completed delivery with short forceps. Both mother and child did well, the child being a fine healthy boy. She was again confined on 12th of November 1875. On this occasion the second stage was prolonged, and I had again to assist instrumentally, with a like good result to mother and child. I examined Mrs. M.'s heart on 1st March 1877, and found its condition as follows:—Cardiac perpendicular dulness 5 inches, transverse 4–4½ inches. A loud soft systolic murmur replaces the first sound at mitral area, but it is heard, though faintly, at each of the other three areas, and also loudly on moving the stethoscope towards the axilla. The second sound is clear, somewhat intensified, but not reduplicated in the pulmonary area. Patient is very thin, but there are no chest symptoms except breathlessness on exertion. No enlargement of liver or spleen. Systolic murmur audible between scapula and spine on left side behind. I was called to attend Mrs. M. during her third confinement on the morning of 14th January 1878, being fully a fortnight earlier than the expected period of delivery. I had not seen her except once since my last report, and was then glad to find her much improved in health. The cardiac action was more powerful, and her general health better, but otherwise the patient was much as on the 1st March 1877. Mrs. M. underwent a particularly easy confinement, without any special inconvenience. There was some irregularity of the

pulse during the down-bearing pains, but this was removed by the steadying influence of chloroform.

The labour was completely natural, and was followed by an entirely normal lying-in period. The patient is, however, still thin, breathless, and easily worn out on the slightest exertion.

CASE XVII.—*Mitral Insufficiency—Hæmoptysis—Congestion of Right Pulmonary Apex—Heart hypertrophied—Sudden Death eleven days after the Second Confinement from Phthisis, aggravated by the Cardiac Disease.*

Mrs. J. A. suffered from rheumatic fever first when eleven years old, and again when eighteen. Since the latter attack she always felt rather breathless on exertion. Having been considerably exhausted while nursing her mother during the last illness of the latter, she went to Italy to recruit, and passed the winter of 1873 at Como. While there she had several attacks of hæmoptysis, once in October of that year, and again in the April and the summer of 1874. She was married in April 1875, at the age of twenty-three. I saw her for the first time in November 1875, when she stated to me that she expected to be confined in the first week of February 1876. On her making some inquiries concerning the propriety of her having chloroform during her approaching accouchement, I was led to examine her heart, and discovered a very loud soft systolic mitral murmur. Her labour occurred on the morning of 27th January 1876. It was normal, as well as easy in every respect, the only peculiarity being that her pulse rose very high, more particularly during the second stage. It was not, however, irregular, so far at least as the finger could make out. It ought to be noticed that there was no hæmoptysis during the pregnancy or during the labour. The patient had chloroform during the greater part of the second

stage. She suckled her child for six months, but as I then found that it was telling seriously upon her general health, I advised her to cease nursing. Soon after this Mrs. A. went to reside for a month in Arran, and returned to town in the end of August considerably improved in health. Shortly afterwards, however, the hæmoptysis recurred. I examined her chest and heart on the 4th September, and could find no pulmonary lesion sufficient to account for the hæmoptysis. There was still the loud systolic mitral murmur audible everywhere over the cardiac region, but no evidence of any lesion affecting the other cardiac orifices. I again examined my patient on the 7th March 1877, when I made the following note of her state:—The apex of the heart beats most perceptibly between the fifth and sixth ribs, a little inside the perpendicular nipple line. Heart's impulse intensified. The perpendicular dulness begins at the upper border of the third rib, extends to the sixth rib, and measures 5 inches. The transverse dulness on a level with the nipple amounts to fully 3½ inches. There is no evidence of extension of cardiac dulness towards the right, and there is no pulsation in the jugular veins. A loud murmur at the apex replaces the first sound in the mitral area. In the tricuspid area the systolic murmur is heard to be harsher and apparently louder than in the mitral area. The murmur is audible over the whole of the cardiac region, and also posteriorly between the lower part of the left scapula and the spine. Second sound seems clear throughout, and is not markedly intensified in the pulmonary area. There is comparative dulness at the apex of the right lung anteriorly, with increased vocal resonance and thrill, and occasional crepitation. The patient is very thin. Appetite is good. Since this was written, the worst anticipations of this case have been realized. Mrs. A. became pregnant, and had rather a severe hæmoptysis in Dalkeith in the summer of 1877. She returned to town, and was con-

fined at 8½ months on the 1st October 1877. The labour was very easy, but the patient had, during the early stage of it, a strange feeling of faintness and sense of impending death. When I saw her first at 9.20 A.M., her pulse was extremely weak and somewhat irregular. I administered an inhalation of 1½ parts of chloroform in 3½ of absolute ether, which had the effect of steadying and strengthening the pulse. The pulsations were readily felt—96 per minute; the respirations were 60. The labour was speedily terminated by natural efforts, and then the pulse fell to 90, and the respirations to 30.

After the delivery the patient felt much better, but the radials were full and corded, and the cough was very distressing.

The accompanying tracing was taken eight hours after labour, the pulsations being 100 per minute. It exhibits a very long line of ascent and considerable irregularity, though at this time the irregularity could scarcely be felt by the finger.

No. 8.

Mrs. A. never improved, however. The pulse and breath-rate increased, pulmonary œdema set in; there was a fine crackle to be heard with inspiration all over the lungs, but no detectable dulness except at the right apex. I endeavoured to support the patient's strength by nutrients, champagne, and digitalis, but all proved unavailing. Mrs. A. died suddenly on the 11th October, apparently from sudden syncope, the result of the exhaustion induced by the phthisis, and no doubt aggravated by the cardiac disease. A post-mortem examination was not obtained.

The three foregoing cases of mitral insufficiency may be dismissed with very few observations.

Case XV. is one of very slight incompetency, well compensated, and occurring in a patient whose circumstances are tolerably easy, and whose labours have been specially so, in consequence of her children being unusually small. Consequently, although the patient has been carefully watched since June 1867 through five pregnancies, the condition of her heart, and indeed of her general health, is better now than at the date of her marriage. The only evidence of cardiac disturbance she experiences is occasional palpitation, and considerable breathlessness on exertion. I ought to observe that Mrs. B. has never nursed her children, which may also account partially for her continued good health.

Case XVI., on the other hand, shows that Mrs. M. is manifestly much worse in health now than when I allowed her to get married. She is thin and worn-out looking; the murmur of insufficiency is louder and more diffused. There is very manifest increase of size in the heart, although the symptoms do not give evidence of increased power. It is to be noticed that without my knowledge she foolishly suckled her second child for over a year,—a proceeding which in her case could only be very injurious. Still she passed through her third pregnancy and confinement with surprisingly little inconvenience, and her condition at present is tolerable. If she is fortunate enough to escape becoming pregnant again, which at her age of forty-one years is not improbable, she may hope to enjoy a life of many years with a fair measure of health and comfort.

The condition of the patient, of which Case XVII. is the history, has proved much more serious than that of either of the other two. Mrs. A. suffered from a lesion of the heart, due to rheumatism occurring a *short* period before marriage,

and consequently the injurious effects of the pregnancy upon the condition of the heart have, as usual, been more pronounced. Though she was able to get through her first pregnancy with comparatively little trouble, the deteriorating effects of it, and of the consequent lactation, though only continued for a few months, were seen in the repeated attacks of hæmoptysis, with breathlessness, palpitation, and general weakness to which Mrs. A. became liable.

Increase of the incompetency was also evidenced by the greater loudness and wider distribution of the cardiac murmur, while the absence of the usual accentuation in the pulmonary area indicated enfeebled action of the ventricles, right as well as left. The dulness at the right apex, though at first of doubtful character, gradually proved itself to be of a tubercular nature. The springs of being were thus sapped at once through the lungs and the heart. A second pregnancy gave rise, as we had feared, to the most unfavourable symptoms, and, under the combined influence of cardiac and pulmonic disease, the patient succumbed.

Such a case strongly supports the view that we ought not to give our sanction to the marriage of a patient who has recently suffered from endocarditis, even although the acute symptoms have disappeared, and the compensatory arrangements of the circulation seem in a fair way of being consolidated. Pregnancy is certain to lead to derangement of the compensation, as well as to prevent the new conditions on which the circulation is to be carried on from being effected quickly and safely.

CASE XVIII.—*Mitral Insufficiency—Premature Labour at Seventh Month—Death three days after.*

(*Communicated to me by* Dr. JOHN LINTON.)

Mrs. S., aged thirty-two, was, for about two years previous to her marriage, noticed to be breathless on going up steep

inclines and on ascending stairs, on which occasions she would place her hand over the region of the heart. There was nothing further observable about her condition to indicate that there was disease of any kind, except a slight occasional dry cough, which was always increased by exposure to night air. She menstruated once after marriage. When about two months pregnant, the cough and breathlessness increased considerably, and I was accordingly consulted regarding her. I found her suffering from slight bronchitis, as also from mitral disease of the heart—systolic. I ordered a stimulant expectorant, which gave her great relief, and enjoined her to keep the house. The cough abated considerably, but the breathing steadily became worse. On going out during the month of January (being the second time for a month or two), and walking quietly for about five hundred yards, the patient became so breathless that she had almost to be carried home, and managed, with the greatest difficulty, to ascend two flights of steps which led from the street to her house. About a month afterwards she was seized with labour pains, and gave birth with comparative ease to a living seven months' child. For three days she continued well, her breathing being quite easy. About 10 A.M. on the third day the nurse's attention was attracted to the fact that she was getting much worse, by observing great coldness of her patient's extremities. Simultaneously with this the patient complained of weakness, and the breathlessness became much increased; she was at the same time tormented by severe cough, and her lips became cyanotic, so that she could not lie down, but had to sit upright, in which condition she died at 7 P.M. on the same evening.

CASE XIX. — *Mitral Insufficiency — Severe Breathlessness during Last Month—Labour short and easy—Patient got*

*worse after Confinement—Had Irregular Pulse, Breathlessness, Bloody Sputa, and died on twenty-third day—Post-mortem: Œdema of Lungs—The Right Ventricle greatly enlarged—The Mitral Valves much thickened and cartilaginous—Kidneys diseased.*

(*Abridged translation from 'von Hecker und Buhl,' S.* 165, *Leipsig*, 1861.)

Patient was twenty-four years old, and had been pregnant four years previously. The former pregnancy had been natural. She had suffered occasionally from breathlessness on exertion, but not severely till three weeks before admission. She believed herself at full term, and her labour had begun when she came into hospital. It was short and easy. The child was a female, weighed 5 lbs., and measured 17⅛ inches. Still, the heart was extremely perturbed. The contractions were so quick and irregular, that complete arrest of cardiac action was feared. Examination after delivery evolved evidence of greatly increased transverse cardiac dulness, and a systolic murmur at the apex, with intensification of the pulmonary second sound. Liver dulness increased considerably. Mitral insufficiency was diagnosed. The patient did not improve much for the first week; the pulse continued at 160–180, imperceptible at the wrist, along with troublesome vomiting, bloody sputa, and great sleeplessness. On the eighth day a change for the better set in, the pulse came down to 96, and could be felt at the wrist; but still the vomiting, breathlessness, and bloody sputa continued. A relapse speedily occurred, and the patient succumbed on the twenty-third day. On section, the lungs were found very œdematous. The heart was greatly enlarged transversely, chiefly owing to increase in the *right ventricle;* its walls appear dilated and fattily degenerated. Microscopical examination of the heart's substance showed general granular infiltration. The borders of the mitral valve were

much thickened, at certain points almost cartilaginous in consistence. Kidneys enlarged; cortical substance pale, and showing the characters of parenchymatous nephritis in the second stage. Liver greatly enlarged, nutmeg-coloured.

CASE XX.—*Mitral Insufficiency—Shortness of Breath and Palpitation during the Latter Months—Delivery easy and quick—After Delivery, serious Chest and Cardiac Symptoms —Recovery.*

(*Abridged translation from* SPIEGELBERG, '*Archiv für Gynäkologie*,' *Bd.* ii. *S.* 243.)

Patient well-built but dusky-looking; had, with good nursing and careful management, got through her pregnancy, and had arrived at the full term, when she was delivered safely and speedily, with nothing further of a disagreeable nature during the latter months than shortness of breath on exertion, and palpitation. Shortly after delivery, her pulse, which was not frequent, became strikingly irregular, and intermitted at variable intervals; but still the arteries were moderately full and tense. After a few hours, oppression of the chest and breathlessness set in, which increased momentarily to orthopnœa. Even at a distance mucous râles were audible. The radial arteries became small and slightly tense, the pulse-wave weak and enormously frequent (140–150), and the face cyanotic. Examination of the thorax showed considerable congestion of the pulmonary vessels, and consequent serous transudation into the bronchi, abnormal extension of cardiac dulness to the right, the apex beat in its normal situation, a loud blowing murmur following the impulse at the apex, which, however, gradually became less distinct as it was followed upwards. Abdomen free; urine scanty and albuminous. Second sound at apex markedly weak, becoming stronger at mid-sternum and towards the right. Over pulmonary area both sounds clear, the second neither intensified nor accentuated. Under the tormenting

addition of a dry cough, during next two days repeated attacks of dyspnœa came on, which disappeared under the continued use of small doses of digitalis, with morphia and saline purgatives. Symptoms disappeared, and patient was discharged on the tenth day.

Cases XVIII., XIX., and XX., which we have now recorded, are examples of the serious results that may follow severe cases of mitral insufficiency when complicated with pregnancy. In Dr. Linton's case (XVIII.), the starting-point for the serious symptoms was a trifling attack of bronchitis during the third month of her first and only pregnancy. From this, however, as the record informs us, she got greatly better, and yet the breathlessness continued to torment her, becoming gradually worse as the pregnancy advanced, notwithstanding very careful management. The exposure to the external air on a single occasion in January, with the exertion of a walk of a few hundred yards, was sufficient to bring on such an amount of dyspnœa that the patient had almost to be carried home. As usual in such cases, premature labour came on, occurring in this instance at the seventh month. The labour, as is also usual, was easy. A short period of deceptive improvement followed delivery. But, only three days afterwards, symptoms of extreme dyspnœa and weakness set in suddenly, no doubt due to suffocative pulmonary œdema, and death took place after a hard struggle of nine hours' duration. Besides showing the extreme danger that may arise in connection with a badly-compensated mitral lesion during pregnancy, the record of this case teaches that the risks cannot be expected to terminate with delivery, but are equally liable to occur during the lying-in period.

Case XIX. emphasizes the same observation. We have in its history the record of a severe mitral lesion, of incom-

petency giving rise to very aggravated cough and breathlessness during the last month of a second pregnancy. From the length and weight of the child, however, I cannot help thinking this patient must have gone wrong in her calculation of the probable day of delivery, and that the confinement must have occurred prematurely, as it usually does in such cases. These symptoms attained a maximum of intensity during delivery, which, however, was short and easy. The usual observation was made, that during the labour the heart became extremely irregular, so much so, indeed, as to seriously threaten the patient's life. Delivery was, however, not followed by any real improvement of the symptoms. Congestive bronchial catarrh, with bloody sputum, and symptoms of pulmonary œdema, with enlarged liver, and an accentuated pulmonary second sound, all bore evidence of congestion and increased tension within the pulmonary circuit. An illusory indication of betterness on the eighth day was followed by a speedy relapse, under which the patient succumbed on the twenty-third day after delivery.

In this case post-mortem examination was able to test the accuracy of the observations and diagnosis during life, and it showed the lungs to be extremely œdematous, and the heart enlarged greatly, the main source of enlargement being the right ventricle, which clearly in this case was ultimately unable to hold its own against the regurgitation from the left ventricle, and hence the pulmonary troubles. The left ventricle was not only enlarged and dilated, but its muscular tissue had begun to be structurally degenerated. Though we have not, in Dr. Linton's case, the evidence of post-mortem examination to go by, the symptoms of the two cases are so very much alike, that it appears to me we might fairly assume that in all probability the right heart here also had been dilated and weak. It seems to me that if we grant that the left ventricle does hypertrophy during

pregnancy, we have in this fact an important help towards an explanation of the great tendency that cases of this kind exhibit to go wrong in the latter months. We know that dilatation is, as a general rule, found to commence first in cardiac cases with the right auricle and ventricle. If, now, it is true, as the observations of Larcher and others show, that the left ventricle during the latter months of pregnancy hypertrophies, whilst the walls of the other three chambers do not, and if before the pregnancy the right ventricle has, owing to the presence of the mitral incompetency, already become barely able to perform its usual function, it appears obvious that if the latter organ is called upon, without obtaining any fresh strength, to propel an increased amount of blood through the lungs, in the face of a regurgitant stream through the left auriculo-ventricular orifice, both increased in absolute amount and under the increased tension it acquires from the contractions of the hypertrophied left ventricle, the right heart, as the pregnancy advances, must come to suffer more and more, and to be less and less competent to maintain the lesser circulation. From this disastrous state of matters arise the œdema of the lungs, the pulmonary infarctions, and the congestive form, as also the persistent nature, of the bronchitic catarrh, which we find associated with pregnancy when complicated with chronic heart disease.

Further, by assuming a tolerably constant hypertrophy of the left ventricle, and a dilatation and consequent weakening of the other chambers, we find an explanation of why delivery does not give that amount of relief in mitral lesions which we would, on a superficial consideration of the case, expect; for it must take a considerable time for the left ventricle of the heart to become reduced in bulk to its normal size, and meanwhile the dilatation of the right side is maintained by its regurgitant action, and the pulmonary lesions

become thereby more and more aggravated, till by and by the patient's condition is irreparable. When we think of the slight changes that an easy labour impress upon the circulation, when the bearing-down efforts which chiefly affect it are weak, we can easily understand how such cases get quietly through the process of delivery, though they gradually succumb to the continuously disturbed condition of compensation operating after parturition is completed. When the labour pains are at all severe, we find their effects evinced by profound irregularity of the heart's action. Those cases that are decidedly relieved by the completion of labour are no doubt such as have still a powerfully-acting right ventricle, with possibly also a minor degree of incompetency of the mitral, for we can seldom with certainty determine by signs during life the exact amount of insufficiency of a valve. It is in cases of this kind that Spiegelberg's theory of suddenly disturbed relative pressure at the moment of delivery, seems to me most inapplicable as an explanation of the untoward results that are apt to arise.

Case XX. is so nearly a counterpart of Case XIX., that I refrain from commenting on it further than to point out—1st, that there was demonstrated a great amount of increased cardiac dulness to the right, which proves great retro-dilatation of the right side of the heart; and 2d, that Spiegelberg's treatment by saline purges, opium, and digitalis appears to have given this patient some relief.

CASE XXI.—*Insufficiency and Stenosis of the Mitral—Embolus of the Artery of the Sylvian Fissure—Labour without any very serious Symptoms, except on the part of the Respiration—Renewed Embolism—Ultimate partial Recovery.*

(*Abridged translation from* AHLFELD, '*Archiv für Gynäkologie*,' *Bd.* iv. *S.* 159.)

Patient thirty-eight years old; primipara; exerted herself

exceedingly when at work, once during the third month, and again during the seventh month of utero-gestation, and in consequence suffered much from violent pains in the hypochondrium. Otherwise she was well during her pregnancy. She had suffered from palpitation and severe headaches six months before the commencement of her pregnancy, but had had no other bad symptoms. During the period of utero-gestation the palpitation had diminished. In the beginning of the ninth month (2d March 1868) she was suddenly seized with giddiness, and became unconscious and hemiplegic on the right side. Her speech was inarticulate, and the extremities on the right side were swollen. A few days in hospital were followed by marked improvement in all the symptoms. The movements on the right side were very defective, but sensation had returned to it completely on the 17th March, when she was taken to the Midwifery Institution at Leipsig to await her confinement. On examination at this time the lungs were found normal. Transverse cardiac dulness $3\frac{3}{4}$ inches from left border of sternum, perpendicular reached from upper edge of third left rib to upper edge of seventh; heart's impulse in normal situation, but diffused; præcordial thrill present; auscultation over the left ventricle reveals a distinct systolic murmur, replacing the first sound; the second sound clear; the sounds of the right heart and of the aorta are normal; the pulmonary second sound is strongly accentuated. On 20th of March, at 6 o'clock P.M., the respiration rose to 48 per minute. There were troublesome symptoms of abdominal distension, for which a dose of castor oil was administered. At 8 P.M. labour began. The pains were at first powerful, so that the cervix was fully developed and the outer os dilated within twelve hours. In the second stage the pains slackened so much that labour had to be completed by traction on a foot, which presented. Immediately after

delivery the frequency of respiration sank, and the patient went into a tolerably quiet sleep. On the following day the patient had a renewed attack of embolism, but ultimately so far recovered as to be able to leave the hospital on 7th July.

We find in Case XXI. another of the leading dangers that chronic disease of the heart is apt to bring in its train, more especially when it occurs in connection with pregnancy and parturition,—I mean embolism. We have already noticed its occurrence in two of our cases—the second and third. In the one under consideration it occurred twice, and yet its results were so far recovered from that the patient was able to leave the hospital in less than four months after delivery. The history of this patient's condition is peculiar, in as far as it declares that the cardiac symptoms decreased with the occurrence of pregnancy. From finding the reverse so constant in our other cases, one is almost tempted to believe that this patient had made an inaccurate statement to Ahlfeld.

It is surprising to find that embolism has occurred so seldom in the cases before us, when the conditions that ordinarily give rise to it have been so specially prominent, as there is no doubt of its relatively increased frequency of occurrence in connection with cardiac lesions. There is as little doubt, however, that it is when the endocarditis, which gives rise to the cardiac lesion complicating the pregnancy, is of recent origin, or when the endocarditis is of the ulcerative form, that embolisms are most apt to occur. In so speaking, I do not include septic embolisms, which are too well proved to appear in connection with puerperal phlebitis of a pyæmic character, and to be the medium of spreading the lethal disorder to distant parts of the system at the same time. I refer merely to simple non-septic plugging of arteries, such as arose in the present case. I have been astonished that it

has not occurred oftener among the cases which we have collected. I have not been able to find a single symptom in any of these cases, if we except the ninth case, to indicate thrombosis or embolism of the pulmonary artery. Yet the conditions under consideration are very many of them such as one would have expected to be accompanied by thrombosis of the latter vessel, if the accident were as common a probability as some authors believe. I have serious doubts, however, respecting the accuracy of the views urged on that subject, and am far from being convinced that the appearance of a phlegmasia dolens some days after or coincident with symptoms of embolism, or, if you like, thrombosis of the pulmonary artery, proves that they were due to a common cause acting at once on the pulmonary artery and in the veins of the limb affected. I am much more inclined to believe that in an obscure pelvic phlebitis a common origin for both conditions would usually be found. At any rate, we have so many possible sources of plugging of the pulmonary artery, such as the existence of a dilated right auricular appendage, or the presence of a clot in a varicose vein in some part of the body, that I am loth to accept the probability of spontaneous coagulation of blood in the pulmonary artery even in hæmorrhagic cases, except on irrefragable post-mortem evidence.

The only case among those that I have recorded in which death might probably have arisen from embolism of the pulmonary artery is the seventh one, which I extracted from Ramsbotham's *Midwifery*. It is, however, to be noticed that though it is to be presumed the lungs were examined with great care, as the condition of the heart, which does not seem to have been suspected before death, is noted with considerable accuracy, there is no mention made of any plugging or of any large clot in connection with the pulmonary organs. It is merely stated that

the lungs were healthy in character and gorged with blood.

In fact, severe embolism, as well as thrombosis of the pulmonary artery, would seem to me to have been considerably overrated in regard to frequency of occurrence in connection with delivery. They belong to the class of loud-sounding terms that are apt to get fashionable, and to be used as a satisfactory explanation in cases of sudden death, even when the presumption is that some other cause may have quite as likely led to the fatal issue. The frequent occurrence, however, of small emboli in the pulmonic circuit, in connection especially with cardiac disease, which give rise to more or less disturbance in the lungs, no person who for a moment considers the conditions that may give rise to embolism can doubt. It is difficult to determine how many of the pulmonary infarctions constantly found in connection with cardiac disease are due to embolic causes.

It is to be remarked that the physical signs, as given by Ahlfeld, are somewhat defective. Except on the score of præcordial thrill alone, it is difficult to see how he satisfied himself that this case had stenosis as well as insufficiency of the mitral. Certainly the rhythm of the murmur given by him is not that of the auriculo-systolic murmur. It is also to be noticed how exaggerated the cardiac and pulmonary irregularity became during the labour. But yet the heart was able, after the conclusion of the extra work associated with delivery, to regain its regularity of action.

CASE XXII.—*Mitral Insufficiency, with slight Stenosis and slightly-contracted Pelvis, observed by Professor Olshausen and Dr. Fritsch through Three Pregnancies—Patient ill from Childhood—Special Distress from Pregnancy appeared about the Sixth Month—First Labour finished by Forceps on account of Pelvic Narrowing—During Lying-in*

*Period repeated attacks of Dyspnœa—Pulse extremely irregular, without apparent cause—Similar History in Second Pregnancy—Delivery by Turning—During Third Pregnancy all bad symptoms aggravated, reaching a climax during Labour—Delivery by Turning—Death ten days afterwards—Great Cardiac Hypertrophy—Mitral Valves slightly contracted, but extremely incompetent — Right Auricle and the Venæ Cavæ considerably dilated—Lungs healthy.*

(*Abridged translation from* FRITSCH, '*Archiv für Gynäkologie*,' *Bd.* x. *S.* 274.)

This patient was watched by Professor Olshausen and Dr. Fritsch through three confinements.

She came into hospital at the age of twenty-one, being in the eighth month of her first pregnancy, on 26th September 1871. She had suffered from palpitation and expectoration of blood ever since her childhood; still, she had been able to do hard work as a servant. She experienced no special distress till the sixth month of her pregnancy, when, on walking quickly or exerting herself or bending down, she was apt to suffer from giddiness, pulsating feelings in her head, muscæ volitantes, tinnitus aurium, and very distressing palpitations. Still, on admission she looked in tolerably good health.

Examination of her condition then gave the following results:—Patient well nourished, nowhere any trace of œdema, slight cyanosis of the lips and eyelids, paleness of the mucous membranes. Cardiac dulness begins at the third rib, extends only quite slightly to the right beyond the left edge of the sternum, and is altogether 4–4½ inches in breadth. Heart's impulse not strikingly powerful, to be felt in the sixth intercostal space, somewhat outside the line of the nipple. At the apex of the heart the first sound is changed into a double weak murmur, the second sound is very weak;

the second pulmonary sound is markedly intensified. The radial pulse rate is 92 ; many beats, small, and almost absent. The pulse is generally small, soft, and irregular.

Labour supervened on the 17th October. It had been preceded by dolores præsagientes for several days. A moderate degree of narrowing in the antero-posterior diameter of the pelvis was diagnosed, and a bad prognosis formed. After rupture of the membranes, the head was found to present the brow at the brim for a considerable time. After the head got pressed down into the pelvis under the influence of good pains, the down-bearing efforts began, and during these the patient was neither cyanotic nor did she suffer from dyspnœa. Delivery was completed by forceps. Throughout the whole labour the cardiac lesion had produced no symptom, and the *early* days of the lying-in period passed in a completely normal manner, except that the patient complained of attacks of distress and palpitation that came on suddenly, without any apparent cause. During these seizures the pulse was found extremely irregular, and 160 per minute. The last attack occurred on the tenth day after the confinement. The patient was then discharged.

On the 14th of June 1873 the patient presented herself at the Maternity for a second time to be confined. Again during the latter months the symptoms had become worse, the palpitation especially having been so distressing since the eighth month that the patient had often been awakened by it during the night. The most troublesome subjective symptom was the shortness of breath, which had scarcely existed in the previous pregnancy.

Pains began on the 9th of July. The head presented with a loop of the cord pulsating in front of it. Professor Olshausen turned before the waters were ruptured, and delivered quickly and safely, the child being born alive.

Some considerable bleeding followed, which was quickly put a stop to. The patient recovered well.

In March 1875 she sent for the aid of Dr. Fritsch, and asked him to attend her at her home during her approaching confinement. She then presented slight œdema of the eyelids and ankles, which, however, was not constant, but came and went. There was no albumen. Her condition was only endurable with complete quietness, and even then she occasionally had attacks of palpitation and shortness of breath, which would wake her in a fright during sleep. Morphia, digitalis, and purgatives afforded her some relief.

Cardiac dulness begins at the second rib, and extends to the lower border of the sixth, towards the left considerably beyond the nipple line, and to the right a little beyond the right edge of the sternum. Præcordial thrill perceptible from the fourth rib downwards. Heart's impulse to be seen and felt over a wide area. Both sounds at apex and base changed into a murmur of a rasping character. Second aortic and pulmonary sounds accentuated. Pulse 92, intermittent, many beats strikingly powerful. On the slightest exertion the heart makes the thorax vibrate.

On the 9th of April pains began, and advanced tolerably rapidly; the patient's condition got markedly worse at this period. The pulse became intermittent and irregular. The patient was at times unconscious, and presented very much the appearance of one partially under the influence of chloroform. The child was turned, as the patient's life seemed now to be in extreme jeopardy, and as turning had previously given good results. Immediately before the operation some strong pains came on, during which the respiration was extremely quick, the pulse could not be counted, the heart hammered against the thorax, and the patient, after uttering a loud scream, became unconscious. The turning was easy, and was followed by a sudden

improvement in the most alarming symptoms. After the free use of wine, the pulse and respiration became quieter. The heart still beat violently, the pupils were dilated, the expression of the patient's face indicated consciousness, but she could not speak. She improved somewhat, but never made any steady advance, and died on the 19th of April.

*Sectio* twenty-four hours after death. Nowhere any trace of œdema. On opening the thorax, both lungs are found retracted. The pericardium lies widely exposed, but contains only a small quantity of pericardial fluid. The heart itself is increased to at least double its size, is horizontally placed, almost globular, and without an apex. Greatest length of the heart, six inches; the left ventricular cone measures somewhat over four inches. The heart contains fluid and coagulated blood, is not specially flabby, but rather hard and firm. The cardiac muscle is healthy (even on microscopic examination). The aortic valve is competent, and free from pathological alteration. The mitral is *slightly contracted*, but *extremely insufficient*. The chordæ tendineæ are greatly shortened; the special valvular cusps are non-existent; on the edges of the valves are flattish thickenings, along with nodules. Greatest thickness of the left ventricular wall, 1·3 cent. Right chamber enlarged in a somewhat less degree than the left—its wall 0·7 cent.; the right auricle and the venæ cavæ considerably dilated. The lungs are anæmic, and free from every pathological change.

I have abstracted this case at considerable length, though by no means in full, on account of its great value as a record of interesting facts. It contrasts in almost every detail, in my opinion, very favourably with cases of severe stenosis. Had we had as pronounced stenosis as we had insufficiency in this case, I am confident that the patient would not have been got so safely over her two first confinements, the former

of which was by no means easy. It is positively astonishing to find so little pulmonary disturbance, and especially to find œdema and hæmorrhagic infarctions absent. These would all have been decidedly present if the stenosis had been to such a degree as to tell upon the lesser circulation. It is interesting to notice how this patient, whose heart was even to the last well nourished, its muscular tissue being healthy, got on tolerably well till the second half of her pregnancy was more or less advanced. Then the usual train of distressing symptoms began to develop themselves, in the form of attacks of dyspnœa, palpitation, and cardiac irregularity. However, during the first two pregnancies the compensation was good, and the deliveries, though the first especially was difficult, were got over with surprisingly little disturbance on the part of the circulation, and were followed by a disappearance of the distressing symptoms. It is only just to observe that the management of this patient's case was throughout conducted with such care and forethought as gave her the very best chance. The timely use of forceps shortening the second stage of labour in her first confinement, doubtless warded off serious risks from over-action of the diseased heart during the second stage. At the next delivery, turning before the membranes ruptured prevented the patient from putting that dangerous strain upon her circulatory apparatus that is inseparable from the down-bearing efforts. Prompt delivery by version in the third confinement was equally judicious, and, had it not been that the heart was by this time hopelessly diseased, ought to have been followed by equally good results as on the former occasion.

Still the fell disease was advancing, as is seen when the great amount of cardiac dulness found by Fritsch in March 1875 is compared with that given by Olshausen in September 1871; and the heart was consequently not able to

recover from the condition of perturbation into which it was thrown by the third confinement.

It is curious to notice the frequency with which Fritsch found the patients unconscious coincidently with the existence of alarming symptoms during delivery. I have already expressed the view that this is most probably the result of imperfectly aërated blood being sent to the brain in consequence of the imperfect circulation in the lungs, the latter arising out of the defective cardiac action during the delivery.

The extremely distressing symptoms affecting the respiration are the more curious, seeing that on post-mortem section the lungs were found so clearly sound, that they are stated to have been free from every pathological change. It would appear most probable that the disturbance of circulation within the lungs had never been of a very permanent character, and that the vessels of these organs had had sufficient time between the attacks of congestion to recover their normal contractility. Still, as we find in the history that the patient had suffered from palpitation and *expectoration of blood* ever since her childhood, it is extremely difficult to believe that the lungs were free from all pathological changes. I fear there must have been evidences of old infarctions found, provided the lungs had been subjected to a searching examination. Still, such must have been of slight amount, or else they would have arrested attention to themselves at the post-mortem examination. Some influence in the direction of limiting the extravasation might perhaps be attributable to the sound condition of this patient's vascular system in general, which, as evidenced by the condition of the cardiac muscles, was good to the last, the lesion being essentially restricted to the left auriculo-ventricular valvular opening. The evil effects of this lesion had begun latterly to tell upon the right auricle, the starting-point for cardiac failure; but even at the time of

death the right ventricle was in good condition. The case illustrates well how a very great amount of incompetency of the mitral, if its effects are well compensated, may be borne, without producing distressing symptoms of a permanent character, provided no continuously acting disturbing force is introduced into the circulation. That force here was the repeated pregnancies, and, according to my judgment, a better illustration of their injurious tendencies could scarcely be found. The pregnancy, at the same time, presented increased difficulty for the heart, and tended to induce further hypertrophy of the left ventricle, and thus to destroy the established equilibrium of the circulation. The labour and lying-in period aggravated all these existing evil conditions, but on the conclusion of these processes all went well till after the middle of the following pregnancy, and so on till the fatal result. I would wish to draw special attention to the fact noted in connection with the first confinement, that the attacks of palpitation were absent during the delivery, but came on during the lying-in period. This fact seems to me to tend in the direction of proving that the condition of the circulation for some time after delivery is such as to continue the risks that threatened before delivery, and helps to explain thereby how so many of those cases go wrong, not during the confinement, but some days or weeks afterwards. The extreme irregularity of the heart's action was probably due to the imperfect manner in which the filling of the large left ventricle was effected in the face of the extreme incompetency of the mitral. It would appear that this chamber only occasionally retained such a supply of blood as stimulated it to powerful contraction.

The following are the results of the above eight cases grouped in abstract:—

CASE XV.—Lesion slight. Patient, after five pregnancies followed by easy labours, scattered over nine years, is now in as good health as at her marriage.

CASE XVI.—Has been three times pregnant. Condition precarious.

CASE XVII.—Patient twice pregnant. Suffered from hæmoptysis and chronic catarrhal pneumonia, with severe palpitation and breathlessness. The right lung ultimately broke down at its upper part. Patient died from phthisis and exhaustion eleven days after her second delivery.

CASE XVIII.—Patient taken severely ill during her first pregnancy. Premature labour supervened spontaneously at seventh month. Patient died three days afterwards.

CASE XIX.—This patient was pregnant for the second time, when distressing symptoms arose. Death from exhaustion and pulmonary œdema twenty-third day after delivery. Labour happened at full term.

CASE XX.—Appears to be that of a primipara, but it is not clearly stated. Serious symptoms supervened during the last month, then became much worse after delivery, which was an easy one ; but patient ultimately recovered. Labour was at term.

CASE XXI.—This refers to an elderly primipara. Embolism supervened in the middle of the ninth month. Labour was not very difficult. Patient was able to leave the hospital after three months.

CASE XXII.—This patient was watched through three pregnancies, and the evil effects of utero-gestation on the heart disease observed by comparison with the patient's condition in the periods between these pregnancies. Death occurred ten days after the third confinement.

In these eight cases, therefore, we find four deaths and four recoveries, or a percentage of 50 fatal cases.

When we remember that our cases of mitral stenosis gave us a fatality of 64·4 per cent., we are pointed to the conclusion that mitral insufficiency must be a less dangerous complication of pregnancy than mitral stenosis.

But Case XVII. can hardly be recorded as a death from heart disease, inasmuch as the phthisis had quite as much to do in precipitating the fatal issue. If we leave it out of account, as it appears to me we are entitled to do, we would then have three deaths and four recoveries, or a mortality of 42·5 per cent. These percentages show a distinct difference between the mortality resulting from incompetency and that from insufficiency of the mitral, and appear to demonstrate that the obstructive lesion is the more dangerous of the two.

We also observe a distinctly less tendency to premature interruption of pregnancy than in the other mitral lesion; for whereas the other cases seldom went the full time (nine out of fourteen being premature), of these eight cases of mitral insufficiency, only one led to premature delivery. It is to be noted, however, that in Case XXII. it is impossible to make out whether the patient had reached the full term or not, owing to imperfection in the record. It has also to be stated that the cases embraced under this head refer to pregnancies of low number, as they include two, if not three, primiparæ, two cases of two pregnancies, two of three pregnancies, and one of five.

For many considerations, it is hazardous to attempt drawing too strict a comparison between the evil results to be expected from the two common mitral lesions. But, on the whole, it seems pretty obvious that a more favourable prognosis may be risked in cases of mitral insufficiency.

## PART III.

### CASES OF AORTIC INSUFFICIENCY.

IN this part of Chapter IV. we proceed to the consideration of our collection of six cases of aortic incompetency.

CASE XXIII.—*Aortic Insufficiency, with Mitral Obstruction —Pulse extremely irregular during Labour—Great Tendency to Faint—Delivery by Forceps—Abortion at 4½ Months, with Threatening Symptoms at time—Recovery.*

Mrs. F., primipara, aged twenty-three, confined on the 20th August 1876.

Patient had an attack of rheumatic fever when fifteen years of age. At that period the physician in charge stated that her heart was somewhat affected. Ever since she has been liable to occasional feelings of breathlessness on going up hill, but has, on the whole, enjoyed fairly good health, and has been particularly well since her pregnancy began. I saw and examined Mrs. F. for the first time in June 1876, with the following results:—Patient is well nourished, and looks healthy. Does not complain of breathlessness. Heart's impulse intensified. Apex beats between sixth and seventh ribs. Dulness increased; the transverse measures 3½ inches, and the perpendicular 5½. Auscultation over mitral area reveals a loud, rough murmur, which precedes and leads up to the first sound. The latter is accentuated, and is immediately followed by a distinct blowing sound. This blowing sound is found at the aortic area to immediately follow the second sound, and it is audible also at left side of the xiphoid cartilage, where it is heard very loud but soft. At the pulmonary area both the presystolic and the diastolic sounds are audible,

—the former only feebly. The pulmonary second sound is clear and accentuated. The presystolic sound is not audible in the aortic area. The radial pulse is jerky, regular as to time, but not so corded as one would expect with apparently such a considerable amount of aortic insufficiency. Mrs. F. fell in labour about 2 A.M. on the morning of Sunday, 20th August. I was sent for at 7 A.M. I then found the head well down in the pelvis, the vertex presenting in the right occipito-anterior position, the cervix fully dilated, but the waters unruptured. The pulse was markedly irregular and feeble. The patient looked *pale*, the face not being congested as is usual at this stage of labour, and complained that she felt ready to faint with every pain. I ruptured the membranes, and administered a tablespoonful of brandy, which latter had the effect of steadying the pulse for a time. The second stage proceeded slowly, as the head was large, and bearing-down efforts on the part of the patient were discouraged by me, because I noticed that during each pain the pulse became extremely irregular, and the patient felt faint. At first I hesitated to administer chloroform, but as the patient was anxious to have it, I ultimately gave it cautiously. I found that under its employment the pulse became stronger and steadier, instead of feebler and more irregular, as I had feared it might. After waiting for several hours, I sent for my friend Dr. Affleck, who came, and administered chloroform, whilst I completed the delivery slowly and safely by the use of forceps. This was about 12 noon The child was a large male. Immediately after the delivery of the child, the after-birth was spontaneously expelled. The hæmorrhage accompanying this process was rather profuse, but was quickly brought under control by kneading the uterus. So soon as the uterus was emptied, the pulse became again steady, and assumed the usual character of the aortic pulse. Both mother and child did as well as could be wished.

On the 25th of September 1877, Mrs. F. aborted, being about 4½ months pregnant. The fœtus had been dead for some time. The same threatening symptoms of irregularity of pulse and tendency to faint were reproduced as on the occasion of her first confinement, but in a somewhat less degree. She, however, revived very quickly so soon as the expulsive pains had completed their task. There was no extra amount of bleeding this time. She recovered uninterruptedly well, only that the pulse continued irregular and weak for some days.

This was the first case of serious insufficiency of the aorta complicating pregnancy and parturition which I had to deal with personally. As the rules of management of such cases given in text-books of midwifery are not very decided, I felt myself working a good deal in the dark, and was consequently greatly concerned as to how the labour would terminate, and watched its progress with much anxiety. It will be noticed that there is a certain amount of obstruction of the mitral as well. But that condition, though sufficient to produce a perfectly audible murmur, and to modify the quality of the pulse, is clearly not as yet very great. It is also to be observed that the lesion was at the time of the delivery of eight years' standing, and that the compensation was good, and indicated no tendency to become disrupted except during the delivery.

So soon as severe labour pains were established, and particularly on the commencement of the down-bearing efforts, alarming symptoms arose, in the shape of great irregularity and weakness of the pulse along with a tendency to faint. To combat these symptoms, brandy was used in considerable quantity, and with advantage. I felt in great difficulty as to whether I ought or ought not to administer chloroform in this patient's case. I reasoned with myself that, as the down-bearing effort was the condition that deter-

mined the greatest amount of irregularity of the pulse, and produced in my patient the feeling of faintness, anything that would diminish it was likely to do good. Besides, I felt assured that, by carefully moderating the dose, I could maintain a constant amount of stimulation to the circulation by means of the chloroform. The results fully satisfied my anticipations. The pulse became stronger, much more regular, and the patient's pallor of countenance less pronounced from the commencement of its administration. It is sufficiently obvious that such a case ought to be allowed to remain as short a time as possible in the second stage. I should have effected delivery earlier had it not happened that Dr. Affleck, for whom I sent to help me, was at the time on duty in a distant part of the city, and fully an hour was lost before he could arrive.

It is to be observed that so soon as delivery was completed the serious symptoms subsided. This is found to be particularly the case with disturbances referable to the effects of labour upon aortic lesions. It will be noticed that the same observation holds good regarding the other cases, except Case XXV., which was so hopelessly complicated that temporary improvement even was impossible, for reasons well established on post-mortem examination. In respect to the abortion that occurred in September 1877, I have little to remark, except that on a smaller scale it was a tolerably perfect counterpart of the confinement in August 1876.

It is difficult to determine whether in a case of this kind the cardiac disease bears any causal connection with the abortion.

Premature interruption to pregnancy is such a common result from causes which we are unable to explain, that a cautious physician does not feel at liberty to dogmatize in a matter of this kind. Still one cannot help thinking that the presence of very pronounced mitral stenosis, along with

aortic insufficiency, may have so deranged the placental circulation in this patient's case as to have led to the death of the fœtus.

CASE XXIV.—*Case of extreme Aortic Insufficiency, with great Mitral Obstruction—Hæmoptysis frequent—Cough, Dyspnœa, and Vomiting—Premature Labour coming on spontaneously at the end of the Eighth Month—Dyspnœa and repeated Attacks of Syncope during the Labour—Death three weeks afterwards from Suffocative Congestion, and Œdema of the Lungs—Post-mortem: Mitral Orifice only admitted the Little Finger to pass—Cusps of Aortic Valve reduced to mere Stumps—Heart dilated and hypertrophied—Lungs œdematous and congested.*

(*Communicated to me by* Dr. ZIEGLER.)

J. M'G., aged twenty-four, primipara, was admitted into the Royal Maternity Hospital, 29th January 1877, having been sent thither from the Royal Infirmary.

Patient had rheumatic fever three years ago. She never was strong, and suffered from shortness of breath and palpitation on the slightest exertion since that illness. In July 1876 she caught cold, and was confined to bed for a fortnight in consequence of an attack of rheumatism, which, however, was less severe than the first attack. Last October, on a cold, damp morning, she was seized with a violent fit of coughing, and expectorated a considerable quantity of blood. She was admitted on the 11th January to the Royal Infirmary, under Professor Simpson's care, where she remained for three weeks, during which time she frequently coughed up blood.

From the Royal Infirmary she was next transferred to the Royal Maternity Hospital, where the following note of her condition on the 1st Feb. 1877 was taken:—Heart's apex beats between the fifth and sixth ribs, 4 inches from mid-

sternum. Vertical dulness commences at the upper border of the third rib, and extends downwards till it meets the hepatic dulness. Transverse dulness at the level of the fifth rib commences at the right edge of the sternum, and extends 4 inches. The first sound is loud and thumping in the mitral area, and a presystolic bruit is heard on auscultation. This varies greatly; sometimes it is well marked, at others almost inaudible. A loud double blowing murmur is heard at the base. A systolic bruit is plainly heard over the carotids. Over the lungs the percussion note is fair. On auscultation, breathing harsh over base of right lung posteriorly. Pulse is weak and irregular, but not jerking. Patient is greatly prostrated, and vomits almost incessantly. Temperature is normal.

Urine.—Sp. gr. 1021. No albumen.

2d Feb.—Vomiting ceased. Patient feels better.

3d Feb.—Felt well during the day. No vomiting. About 9 o'clock P.M. the patient began again to feel breathless, with great pain over the apex of the heart. Slight cough. Some moist rhonchi are heard over the right lung, both anteriorly and posteriorly. Patient takes iron and digitalis.

4th and 5th Feb.—Patient continuing better.

6th Feb.—Premature labour came on. She complained of uneasiness during the day, and by 6 o'clock she felt slight pains. At the same time the os was found to be the size of a shilling, and the vertex presenting. Patient had taken little food during the day, and appeared very anxious and weak. An egg and brandy beaten up was administered and retained, though thrice previously the same nutriment had been vomited. At 7 o'clock the pains were more regular and more frequent. She now, after showing some signs of dyspnœa, fainted. Small quantities of brandy were frequently administered, but, notwithstanding, she twice fainted during the following two hours, although the pulse

kept tolerably good. Thrice she suffered from sharp attacks of dyspnœa. During this time it was difficult to keep her in bed, although, when told in a decided tone to lie down, she could do so without any worse attack coming on. She had an enema at 8.30 P.M., which seemed to relieve her, for she remained tolerably quiet for an hour afterwards, the pains being regular. At 10 o'clock, Dr. Ziegler, who was summoned to see her, ruptured the membranes, with great relief to the patient, who afterwards breathed quietly. The labour terminated satisfactorily at 11.15 P.M. The child, a female, weighed 5 lb. 5½ oz., and was healthy. Its length was 18 inches. Temperature after delivery, 98·9°. Pulse, 92.

7th Feb.—Patient very sick during the night, but was relieved after taking some brandy. Morning temperature, 98·6°; pulse, 80. Evening temperature, 99·4°; pulse, 78.

8th Feb.—Breathing much improved. The iron and digitalis mixture has again been commenced.

9th Feb.—Passed a bad night. Rheumatic pains in the elbows, wrists, fingers, and right hip and knee. Quantity of urine for the last few days small. A little albumen present. Morning temperature, 100°; pulse, 84. Evening temperature, 101·1°; pulse, 100. Patient taking acetate of potash and salicylic acid.

14th Feb.—Presystolic mitral murmur well developed, and thrill felt over the mitral area.

15th Feb.—Face swollen and puffy. Abdomen tympanitic. Rheumatic pains are now confined to the right hip and knee.

16th Feb.—Patient has had four attacks of dyspnœa. Moist râles heard over the chest.

24th Feb.—Patient's condition varies. Rheumatic pains in hip and hands. The attacks of dyspnœa are more frequent. For two days she managed to sit up in bed, the rheumatic pains which had previously prevented this having disap-

peared. There is also occasional cough, with expectoration of frothy mucus tinged with blood. Average temperature for the last ten days,—morning, 99·6°; evening, 100·8°. Twice in the evening the temperature reached 101·8°. Average pulse for the same period,—morning, 94; evening, 108.

25th Feb.—Patient suffering much from dyspnœa, and unable to lie down.

26th Feb.—Patient very weak, and has suffered all night from extreme dyspnœa. At 2 P.M. she appeared somewhat better, and asked for a little tea, after which she slept for half an hour. At 3 P.M. she had another severe attack of dyspnœa, and though afterwards she seemed slightly easier, she gradually became weaker, and sank at 4.45 P.M.

Post-mortem examination on 28th February, conducted by Dr. Littlejohn.—Pericardium thickened and roughened in some places, and contained about six ounces of serous fluid and a few flakes of lymph. Heart considerably enlarged from hypertrophy and dilatation. Great mitral stenosis, the orifice roughened, and scarcely admitting the little finger. Aortic valve thoroughly incompetent, its cusps being reduced to mere stumps. Cavities containing clots; from the right ventricle a decolorized clot was removed, together with one passing into the pulmonary artery and its branches.

Lungs.—Old pleuritic adhesions posteriorly; tissue œdematous and congested, also somewhat consolidated, but small cut portions did not sink in water.

Liver enlarged, fatty, and much congested.

Kidneys fatty and much congested.

Uterus.—Its inner surface shows the late attachment of the placenta. Externally there are some adhesions between it and the surrounding structures.

It is extremely interesting to notice in this valuable case the points of similarity of lesions and symptoms existing between it and Case XXIII. Thus we had well-marked aortic

lesion, coinciding with constriction of the mitral orifice, and during delivery a pronounced tendency to syncope in both cases. Dr. Ziegler's case, however, was altogether a more serious one than mine, and presented many more of the usual disagreeable concomitants of the conjunction of heart disease with pregnancy. Thus we had not only a tendency to faint, but actually repeated attacks of syncope, notwithstanding the administration of considerable doses of brandy. Besides this, there were pulmonary œdema, and hæmorrhagic infarction of the lungs leading to hæmoptysis, frequent attacks of severe dyspnœa, and ultimately to premature delivery about the end of the eighth month, followed finally by death three weeks afterwards. The exhaustion of the labour, though it appears to have been an easy one, was no doubt a serious shock to the enfeebled and extremely diseased heart; and though this was partially recovered from, the unfortunate onset of an attack of subacute rheumatism, accompanied by pulmonary congestion and œdema, proved too much for the patient's recuperative powers.

In this instance the aortic insufficiency as well as the mitral obstruction was of a very extreme type. It is indeed strange that with such an amount of cardiac disease the patient could have lived so long, and more especially have survived her delivery. It is of importance to observe the very great relief afforded by evacuation of the liquor amnii. It is not stated whether it was greater in quantity than normal. But from what we know of the bearing of abdominal distension upon cardiac disease, it seems to me we are entitled to assume that it really had been present in excess. That being granted, it is quite in conformity with what we know of the pathology of pregnancy, that the removal of the liquor amnii, by giving more room in the thoracic cavity, would render the circulation less laboured within that space, and tend to relieve the urgent symptoms

on the part of the heart and lungs, whilst it at the same time strengthened the expulsive power of the uterus.

An interesting question is suggested by this case, viz., What effect would the extreme mitral obstruction have upon the condition of matters? would it be beneficial or otherwise in its bearing upon the aortic lesion? To solve this point, we have to consider how danger is specially apt to arise in connection with this lesion. 1. Fatty degeneration is apt to occur in the enlarged ventricle in consequence of defective nutrition of the heart, the coronary arteries not being filled sufficiently, because, owing to the incompetency of the aortic valves, there is never a sufficient tension in the blood in the sinuses of Valsalva to thoroughly flush these arteries. 2. Danger is apt to arise from the hydraulic pressure of the column of blood, which regurgitates at each diastole with a force proportional to the perpendicular height of the highest part of the vascular system above the apex of the left ventricle. 3. Danger is apt to arise, also, in consequence of the hypertrophy of the left ventricle leading to mitral incompetency, and thus setting free an hypertrophied ventricle to pump with a force, corresponding to its thickness, a stream of blood into the venous radicles of the lungs, and thus to directly oppose the normal action of the right ventricle and induce destructive congestion of the lungs.

The extreme mitral obstruction would have no effect upon the first tendency to disaster; but, by diminishing the velocity of regurgitation from the ventricle, it seems to me quite possible that, within certain limits, it may have prevented in some degree the indirect evil effects of the aortic lesion in the third tendency to death mentioned. Upon the question whether it could modify the serious effects of the backward column from the aorta, by limiting the distension of the ventricle before each systole, I really feel unable to venture a positive opinion, although, from the way in which the

pulse was modified in the cases I have watched, it seems not altogether improbable. If these ideas are correct, the mitral lesion might have acted rather beneficially in these cases, in the way of lessening the evil tendencies indicated under heads second and third. But then if the mitral obstruction really modified in any way favourably the aortic lesion pure and simple, it in its turn introduced fresh troubles, in the form of dyspnœa, continuous pulmonary engorgement, and so forth, so that I fear, on the whole, the complication can scarcely be looked upon as a beneficial one.

There is no doubt but it did harm in Dr. Ziegler's case, by precipitating the fatal issue after delivery; and there is reason to fear that, even if it may possibly be an advantage during the delivery, it is a complication of evil import during the lying-in period.

CASE XXV.—*Aortic Insufficiency, with Mitral Stenosis, referable to an attack of Rheumatic Fever five years previously—About end of Third Month of First Pregnancy, severe Cold caught from Exposure—About the Sixth Month, severe symptoms supervene—Pain in Left Chest—Palpitation—Tendency to Faint—Hæmoptysis—Nephritis—Convulsions—Accouchement Forcé—Death—Post-mortem: Aortic Valve much diseased and acutely inflamed—Mitral much stenosed.*

M. S., aged twenty-six, residing at High Street, Edinburgh, was admitted to the Royal Infirmary, bed 8, Ward XI., on 8th April 1877. She complained of severe cough and hoarseness, also of pain in the left side, with palpitation and shortness of breath.

Four months ago the cough and hoarseness came on after exposure to wet and cold. At this time she applied at the New Town Dispensary for medical attendance. This she obtained, and was considerably relieved, but about the

beginning of April she again became worse, her cough returned, and her voice was almost entirely lost. The pain in her left chest now also began, its first onset being sudden and apparently of a spasmodic character. Five years ago palpitation began, and has gradually got worse till now. She had a severe attack of rheumatic fever at the age of nineteen, with which she was confined to bed fourteen weeks. She had no ill health after the rheumatic attack, till the palpitation began as above stated. She has never been overworked, and has had a fairly comfortable home.

Her father died suddenly of paralysis. Her mother is still alive.

The patient's condition at the above date was noted to be as follows :—She is of average height, but her muscular system is very ill-developed. For the last six months she appears to have been losing flesh to a considerable amount. Her face is flushed, and her temperature is 98·2°. She perspires a good deal. So far as can be made out, the patient is about the end of the seventh month of pregnancy. The fœtal heart and uterine souffle are both to be heard.

Circulatory System.—Pulse 110, irregular, bounding and receding rapidly from under the finger. Severe pain in the præcordia, with palpitation and frequent syncope on exertion. On placing the hand over the cardiac area, the apex beat is felt to be bounding, irregular, and diffused, but most perceptible between the sixth and seventh ribs. There is feeble pulsation in the epigastrium, and between the cartilages of the fourth and fifth ribs on the left side, where a slight thrill can also be felt. On percussion, perpendicular dulness is found to begin at the third rib, and extend downwards to the upper border of the seventh. Transverse dulness at the level of the fourth rib extends 4 inches outwards from the median line. Listening in the mitral area, a rough blowing murmur is heard to replace

the first sound, and it is propagated upwards towards the axilla, its rough character being gradually lost as the axilla is approached. A well-marked diastolic murmur exists also in the mitral area. A murmur probably presystolic can be detected here also, but, from the rapid action of the heart and the diastolic murmur, its rhythm cannot be determined with certainty. In the aortic area the systolic murmur is loud and rough, and propagated up the carotid, while the diastolic murmur is also heard, but not loudly. The second sound is barely audible in the aortic, but can be heard in the pulmonary area.

Respiratory System.—Cough was severe a fortnight ago, but is much better now. Respirations vary between thirty and forty in the minute. Three weeks ago, patient expectorated three or four mouthfuls of blood, but never spat blood previously, and has not done so since. At present there is little expectoration of any kind. Vocal thrill is felt more distinctly on the right side than on the left, and at the right apex the percussion note is impaired, and the respiration is heard to be prolonged, but there are no accompaniments. Her appetite is bad, and her thirst great. Patient vomits almost everything she takes. Micturition is frequent, and accompanied with pain. Treatment consisted in the administration of digitalis and an antispasmodic mixture, with mustard poultices applied occasionally to the chest. She was also put upon full diet, with wine.

The patient's condition continued much as above described till 24th May. Her pulse occasionally rose a few beats, but her temperature was never above the normal. On the 20th and 21st of April it is noted that she spat a few mouthfuls of blood, and on the 20th of May that her urine contained about one-fourth albumen. On the 23d of May it was noticed that she had slight œdema of the feet, and on again testing the urine it was found to be albu-

minous. At 1.30 A.M. on the 24th May, the patient had a convulsion, after which she was delirious. Professor Simpson was sent for at 4 A.M., and, after examining the patient, ordered her to be taken to the Maternity Hospital.

Mr. James A. J. Smith, house surgeon at the Royal Maternity Hospital, reports as follows on the 25th of May: —'Patient was sent down here from the Royal Infirmary at 6 o'clock A.M. She was unconscious on admission, but started up in delirium every five or six minutes, and had to be constantly watched and held down by two nurses. Pupils not much dilated. Pulse was full and bounding, rate 144 per minute; temperature, 101·1°; respiration, 48. Her urine was drawn off shortly after admission, and on being tested was found to be highly albuminous, sp. gr. 1010. Dr. Keiller saw her at 11 A.M., and induced premature labour by means of the india-rubber bags. At 12 o'clock he turned, and delivered her of a seven months' fœtus, which had evidently been dead for some time. After delivery her pulse was 116. She could not swallow, and an enema of beef-tea and brandy was administered, but she never rallied, and died at 3 P.M. Respiration continued for upwards of a minute after the pulse had become imperceptible.'

The delivery was easy, and the patient had no fainting during its course.

The post-mortem examination was made by Dr. Littlejohn twenty-six hours after death. Body fairly nourished. Surface extremely pale. Cellular tissue and subcutaneous fat œdematous. Uterus reached as high up as the umbilicus.

Heart.—About two ounces of clear serum in the pericardium. Right side of the heart distended by a clot. A decolorized clot in the right ventricle, becoming darker in colour as you approach the pulmonary artery, which is also filled by a dark, tolerably consistent clot. The right auricle is very much dilated, and its walls are extremely thinned.

The right ventricle is also somewhat dilated, and its walls thinned and covered by fat. Left ventricle is filled by a dark clot, and the aorta contains a similar clot. The walls of left ventricle measure at apex 5 lines, midway between apex and base 6 lines, and at thickest part (close to mitral orifice) 9 lines. The left auricle contains a large, dark-coloured clot, and is somewhat thinned, but not greatly dilated. Aortic valve greatly incompetent, and all the segments have well-marked recent vegetations. The aorta measures 2¼ inches in circumference; no atheroma noticed. Mitral orifice stenosed, only admitting one finger. The heart weighs 1 lb. 3 oz.

Lungs.—Fully half a pint of sanguinolent serum in the left pleural cavity. Tissue œdematous.

Slight chronic adhesions on the upper surface of the liver, which weighs 4 lb. 3 oz. Spleen normal.

Kidneys.—Left kidney weighs 5 oz. Capsule strips off easily. Cortical substance pale, medullary congested. Right kidney weighs 6 oz., and presents the same appearance. Examined microscopically, both kidneys showed evidence of parenchymatous nephritis in the second stage of this disease.

Bowels slightly adherent to the left lateral and posterior surface of the uterus. Right ovary contains a large, well-marked corpus luteum. Some hæmorrhage into the folds of the right broad ligament. Uterine wall measures in thickness 1⅛ inch, the cervical wall ¼ of an inch.

This case came under my notice through the kindness of one of the resident medical officers of the Royal Infirmary, who was aware that I felt interested in this subject. My thanks are specially due to Professor Sanders and Dr. Keiller for allowing me access to the patient in the hospital and to the post-mortem, and also for the use of the notes of the case in their records.

In the first place, I would remark upon the frequency of the curious combination of aortic insufficiency with mitral stenosis in connection with pregnancy. It is really strange to find it in three out of six cases collected simply as they turned up. I have already indicated how the complication may be supposed to act, and therefore will not recur to this matter.

There are, however, several points in this case that deserve careful consideration. First of all, I should draw attention to the occurrence in connection with it of acute endocarditis, which is in substantiation of the views expressed on this head by Lebert, Löhlein, and others. The inflammation in this patient's case was clearly of the plastic form, as there was nothing of a septic nature to be even suspected about her. The onset of acute parenchymatous nephritis is a very striking example of the mutual interdependence of the vascular and renal systems during pregnancy. Doubtless the condition of the blood being, as is well known, during pregnancy very much allied to that of the blood in Bright's disease, strongly predisposed to the onset of the nephritis, whilst this tendency would be further aided by the serious defects in the central circulatory apparatus. On the other hand, so soon as the acute nephritis was established, the diseased heart would find its work very much added to by the consequent obstruction in the kidneys, and the onset of the convulsions. It is thus in no way difficult to account for the endocarditis, when we simply reflect upon those numerous disturbing influences.

It is striking to notice how ineffective delivery was in affording substantial relief to this patient. It was easily, safely, and skilfully performed by Dr. Keiller, and yet the patient sank all the same, no sooner apparently and no later than she most probably would have done had she been let alone. Doubtless the struggle in her economy was now placed in

the vascular and nervous systems. The state of the genital system, though potential as a starting-point for the development of the influences that had by this time become all-powerful, had at this moment become, as it were, a collateral phenomenon of no great importance as regarded the issue of the combat. For this reason, as I think, there is always to be faced, in those cases of eclampsia in connection with pregnancy, a considerable difficulty as to how far one is at liberty to interfere in the way of hastening delivery. As a prudent physician will always consider, in writing a prescription, whether he is certain that he is not ordering something to his patient's hurt, so a careful obstetrician, before doing anything in such a case, will always debate well with himself whether he is certain that, in endeavouring to free his patient from one risk, he is not involving her in another and a greater.

I need scarcely point out that the history of this patient's condition illustrates well the evil effects of pregnancy in deteriorating the condition of a heart already threatening to yield.

It will be noticed that, though there is a well-marked history of rheumatism of five years' standing, and though the patient had evidently not been in robust health since that period, yet that an exposure to cold some time between the third and fourth month of pregnancy was the first commencement of the patient's serious illness. This attack was got over, however, and it was not till the third trimester of pregnancy was attained that the heart symptoms, in the shape of severe palpitations, tendency to syncope, troublesome cough, cyanosis, dyspnœa, and hæmoptysis supervened. Removal from a cold and filthy dwelling in High Street, to the comparative comforts of an hospital ward and careful medical treatment, relieved her somewhat at first, till the shadows that had been hovering around her suddenly began

to close upon her, and the sad complexus of fatal influences recorded above proved too much for her feeble strength.

CASE XXVI.—*Severe Aortic Insufficiency, traceable to an attack of Acute Rheumatism two years before commencement of Third Pregnancy—Spontaneous Premature Delivery about end of Seventh Month—Death three weeks afterwards—Post-mortem: Lungs œdematous—Extensive Disease of Aortic Valve—Heart hypertrophied.*

(*Reported by* Mr. CHAPMAN, M.B., *Resident Physician, Royal Infirmary.*)

Mrs. P., aged forty, was admitted to the Royal Infirmary, 5th November 1877, complaining of palpitation, shortness of breath, and pain in the chest. Two years ago she had a severe attack of rheumatic fever, and at that time became aware that her heart had become affected. She had been previously pregnant on two occasions. She states that her present illness commenced about six weeks before her admission, with marked increase of her usual breathlessness and cough, along with swelling of the legs. On admission, she was found to suffer from general bronchitis and œdema of the lungs, with great aortic regurgitation. The murmur of insufficiency was of a highly musical character. She then stated that she was in the sixth month of her third pregnancy, but she was manifestly in the seventh month. The fœtal heart was distinctly audible. Appropriate treatment was directed to allay the patient's distress and support her strength. On 15th November, at 4 A.M., labour pains set in, and the patient completed a very easy delivery at 10 A.M. The child seemed between the seventh and eighth month. There was only slightly greater loss of blood than usual. The patient evinced no tendency to faint at any time during the delivery. Next night the child died. The patient at this time seemed also to be dying, suffering from extreme weakness and threatenings of syncope. Brandy and carbonate

of ammonia were freely administered, with the result that the patient rallied for a time. She, however, gradually became more and more exhausted. The dropsy increased, bloody diarrhœa came on, until the 8th of December, when she died.

The post-mortem examination showed that both lungs were engorged and œdematous. The pericardium contained about three ounces of fluid. The heart weighed 15½ oz. The left ventricle was much hypertrophied. There was incompetency of the aortic valves. On laying the aorta open from the front, the middle cusp of its valve was noticed to be peculiarly vibratile, in consequence of thickening so arranged along its free margin and attached border, as to leave the intermediate portions of the cusp almost entirely free from disease. The other cusps were slightly thickened.

This case calls for little remark. It merely emphasizes what we have been all along asserting during the consideration of these cardiac cases. It shows how readily a heart recently attacked with acute organic disease is likely to be injured by pregnancy. It also brings under our notice the fact, so often insisted upon in these papers, that the chief embarrassment is likely to appear about the sixth month. It likewise shows how delusive the onset of labour may prove in such cases, even although it is an easy and a spontaneous one, and what dangers may arise during the lying-in week.

The peculiarity of the middle cusp of the semilunar valves explains the mechanism of the strange murmur which was heard during this patient's life.

CASE XXVII.—*Severe Case of Aortic Insufficiency—Violent Præcordial Pain and Dyspnœa about Twenty-eighth Week of Pregnancy—Improvement under Treatment for a time, but return of Distressing Symptoms on slight Exertion—*

*Pregnancy interrupted by Spontaneous Premature Labour about Thirty-fourth Week—Labour easy, and remission of urgent Symptoms speedy.*

(*Translated from* SPIEGELBERG, '*Archiv für Gynäkologie*,' *Bd.* ii. *S.* 239.)

Patient thirty-four years old; pregnant for second time. Previous pregnancy and labour natural throughout, and had occurred six years ago. The present one had been so up to the end of its first half. Violent palpitation, præcordial pain, and dyspnœa supervened about twenty-seventh to twenty-eighth week. Evidence of great aortic insufficiency, with roughening of the edges of the cusps, and likewise of obstruction, was elicited. The left ventricle was found much enlarged, but no evidence of mitral incompetence. Cyanosis of lips, œdema (slight) of the feet, and a small amount of albuminuria were present. Saline purgatives, quiet, and low diet effected a beneficial change in the symptoms for a time, which, however, was completely undone by the patient attempting to return to her ordinary employment. An interruption to the pregnancy resulted about the thirty-fourth week. The labour was easy, and was followed by a speedy remission of the threatening symptoms.

CASE XXVIII.—*Aortic Insufficiency—Slight Case—Dyspnœa and Palpitation apparent about Sixth Month—Patient improved under Treatment—But Premature Labour came on in the Eighth Month, and was followed by Recovery.*

(*Translated from* SPIEGELBERG, '*Archiv für Gynäkologie*,' *Bd.* ii. *S.* 239.)

Patient nineteen years old, primipara; applied for aid when the pregnancy was about six months advanced. Had been suffering greatly for weeks from severe palpitation, and had had two attacks of angina pectoris. Countenance very much flushed, but no cyanosis, no œdema, no albuminuria.

With treatment similar to that of Case XXVII. patient

improved. Fell in labour, however, in the eighth month, but got easily over it. After this the symptoms disappeared, and the patient made a good delivery.

The two foregoing cases present examples of pure aortic lesion. The first was a severe case, the second a comparatively slight one.

It will be noticed that they support Spiegelberg's statement that in this lesion the danger is specially apt to arise during the latter half of the pregnancy, or, if that is got over, during the labour, and that at that period it is apt to be extreme. But once delivery is effected, then remission of the threatening symptoms may be expected with considerable likelihood.

The great relief afforded in these cases by delivery forms one of the most telling points in favour of Spiegelberg's theory of diminished aortic pressure immediately on the emptying of the uterus.

But I am not prepared to admit such a sweeping conclusion in the face of so many difficulties. My explanation of the beneficial result is different, as I have already pointed out, and is, that it is greatly due to the fact that the down-bearing pains are unquestionably associated with a very greatly intensified aortic pressure, and that this tension acts during the second stage of labour with a threatening effect; but so soon as the uterus is emptied, the original pressure is restored, and thus a calm of the disturbance is the result. I hold, therefore, that it is recurrence to the nearly normal tension operative before delivery that is the main explanation of the beneficial effects that follow labour in these cases, and not the occurrence of any sudden and great diminution of aortic pressure. Some beneficial effect must, however, result from the greater freedom with which the diaphragm is enabled to move after delivery, and consequently the greater

ease with which the respiration and the circulation in the chest are carried on, else delivery could give little relief to serious symptoms existing before labour. The aortic tension appears also to be slightly less, so far as one can judge from sphygmographic evidence.

The results of these six cases were not favourable,—three recovered, and three died, giving a mortality of 50 per cent.

In four of them it will be observed that premature labour supervened spontaneously. In a fifth case, though the first labour was at term, abortion occurred during the second pregnancy at about the middle of the fifth month. It thus appears that in this cardiac lesion we are quite as likely to meet with premature interruption to pregnancy, as we are in connection with mitral stenosis. It is of course to be remembered that three of these cases were complicated with mitral stenosis. But in so far as prematurity of delivery is concerned, it will be noticed that the cases of pure aortic insufficiency were not exceptional, for in all three the labours supervened spontaneously before term.

## PART IV.

### IRREGULAR CASES.

THE following three cases, though important, are not strictly capable of being included under any of the above headings.

CASE XXIX.—*Dilated Weak Heart—Right Side chiefly affected—The Right Auriculo-ventricular Orifice dilated—Chronic Bronchitis and Emphysema, aggravated by Pregnancy—Premature Labour coming on at Seventh Month under Threatening Symptoms—Delivery—Slight Improvement — Death twelve days afterwards, with Pulmonary Œdema and Congestion of Bronchial Tubes — Fatty Kidneys.*

For the opportunity of watching this case I am indebted to Dr. Matthews Duncan.

A. G., aged thirty-seven, a needlewoman, carrying her fourth child, was admitted, 11th of January 1877, to the Royal Infirmary under Dr. Haldane, and transferred on the 13th to Dr. M. Duncan's ward. She believed herself to be seven months pregnant. She was very cyanotic. Her habits have been rather intemperate. The present illness began thirteen months ago, with pain in the back and shoulders, cough, palpitation, and swelling of the face and limbs. In March 1876 she was in Ward XI. for one week, suffering from bronchitis and œdema of the lower limbs, but no notes of her case can be found. After leaving the Infirmary convalescent, she worked for two months before becoming pregnant. As the pregnancy advanced, however, the troublesome symptoms returned, and she lost strength. During the last two months her legs and labia have swelled

more and more, her expectoration has been profuse, she has suffered from severe dyspnœa, amounting at times to orthopnœa, and for the last month has not been able to leave her bed.

On admission she was livid and almost breathless. The lower limbs and labia extremely œdematous. Slight uterine pains occurred occasionally. On 13th January, after transference to Dr. M. Duncan's ward, he incised the labia preliminarily to inducing labour next day. Her breathing was then orthopnœic; lips cyanotic; pulse 120, weak, but regular; respirations 36. No abnormal dulness over the chest. At base of both lungs, before and behind, coarse crepitation mixed with fine crackling audible. Over the rest of the chest coarse crepitation heard, along with some sibilant and sonorous râles. Expectoration copious, rather thick, and adhesive. The presence of ascitic fluid could not be made out with certainty. Epigastric pulsation could not be determined, apparently on account of the uterine tumour. Cardiac impulse weak and diffused. Apex beat most perceptible on a line perpendicular with the nipple between the six and seventh ribs. The transverse dulness at the level of the nipple passes about half an inch outside it, but does not pass beyond the mid-sternum towards the right. First sound of the heart, in the mitral area, weak and muffled. Second sound clear, following very closely on the first; disproportionately strong, but not accentuated. No distinct murmur audible at any of the cardiac orifices. Urine scanty, sp. gr. 1020; acid, slightly albuminous, and with hyaline casts. Ordered poultice to front of chest, and brandy six ounces per diem; also,

℞ Tr. digitalis,
Tr. ferri perchlor., āā ♏x, thrice daily.

14th January.—Labour began without interference this morning. It lasted for six and a half hours, but was only

severe for about half an hour. A premature child was born alive, but died shortly after birth. The placenta was expelled twenty-five minutes after the child. No bleeding occurred.

At twelve o'clock on the day after labour she looked more cyanotic than on the previous day. Pulse extremely feeble. She, however, expressed herself as feeling somewhat easier since her delivery. The cardiac region could only be examined, and that imperfectly. There could now be made out epigastric pulsation quite markedly. There was, however, no murmur audible. Expectoration still copious.

She improved somewhat during the next four days, and on the 22d Dr. Haldane examined her heart, and considered there was dilatation of the right side, consequent on emphysema and bronchitis. From the fifth day she grew worse, and on the eleventh day after labour she died. She was semi-comatose for a day before death; passed urine involuntarily. Dry cupping was tried, but afforded only temporary relief to the dyspnœa.

Post-mortem Examination. — Patient died 26th January 1877, and the autopsy was made at one o'clock P.M. on 27th January by Dr. Wyllie.

The body was well formed and developed. The lower extremities were dropsical.

Thorax.—There were old adhesions over the apex and part of the external surface of the right lung. The right pleural sac contained about a pint of serous effusion. There were also some old pleuritic adhesions over the apex of the left lung, but the left pleural sac contained only two or three ounces of serum. The anterior edges of both lungs were markedly emphysematous. The bronchial tubes on both sides were congested, and contained a great quantity of viscid muco-purulent secretion. Some of the smaller bronchial tubes were slightly dilated, and presented a good deal of thickening and induration of their walls. The substance

of both lungs was greatly engorged with blood, and very œdematous. This was best marked in the left lung, for on the right side the pleuritic effusion had somewhat reduced the bulk of the organ, and in some degree had limited the congestion and œdema of its substance. The heart was enlarged, weighing twelve ounces. The chambers were filled with dark fluid blood. Its valves were natural, but the right auriculo-ventricular orifice was considerably dilated, admitting six fingers; cone-diameter was 1·7 inch. The left auriculo-ventricular orifice was natural, admitting only three fingers. The right auricle was much dilated, but not hypertrophied. The right ventricle slightly dilated, and its muscular wall was slightly hypertrophied, measuring three-eighths of an inch in thickness. The left ventricle was slightly dilated and slightly hypertrophied, its wall measuring half an inch in thickness. The left auricle was natural.

Abdomen.—There was about a pint of serous effusion in the peritoneal sac.

The uterus was well contracted. It measured in length, from the peritoneal surface of the fundus to the os externum, six inches, and in breadth, between the origins of the two Fallopian tubes, four inches. Internally, the cervix measured two inches in length, and the body of the uterus three inches. In thickness the anterior wall of the uterus measured one inch, and the posterior one and a quarter inch. Both the anterior and posterior walls were coated with a dirty grey débris, which presented an appearance something like that of a diphtheritic membrane. The grey coating was not present in the cervix, the mucous membrane of which presented the natural arrangement of folds.

The liver weighed three pounds. On section it presented a nutmeg appearance.

The kidneys weighed, right 7½, left 6½ ounces. The capsule could be easily stripped off, and the surface beneath

it was smooth; but on section the cortical substance presented in a moderate degree the mottled or streaked appearance characteristic of fatty degeneration, and on microscopic examination the epithelium of the tubules was found to contain very abundant fatty molecules.

In this case it will be noticed that there are a good many complications. We have a certain amount of degeneration of the kidney, some emphysema of the lungs, and a good deal of chronic bronchitis. Still the leading points of the case are so very like those we have already seen in cardiac cases pure and simple, when severe, and complicating pregnancy, that I am satisfied that the cardiac mischief formed here the most essential lesion. For example, we had extreme dyspnœa, amounting often to orthopnœa; we had also the pulmonary œdema, cyanosis, and latterly we had effusion into the cavity of the right pleura, all of which we have learnt to expect as accompanying severe cardiac disease when pregnancy is present. Finally, we have the usual spontaneous interruption to the pregnancy, precisely at the time when preparations were being completed to induce labour. It is positively amazing to notice how frequently the same occurrence has repeated itself in the course of our recorded cases.

There certainly was not a clearly defined murmur in this patient's case, but there was a distinct muffling of the first sound. There was also an obvious disturbance of the rhythm of the heart, as I noticed again and again. The second sound followed abnormally quickly upon the first. This was no doubt due to imperfect action of the ventricles, which permitted the semilunar valves to shut before the ventricles were completely emptied. One might have expected reduplication of the second sound here, as the resistance in the pulmonary circuit was markedly great, so that, notwithstanding the slight renal disease, one would have expected that the tension in the pulmonary artery would have

been greater than in the systemic circuit. But there was no evidence of such condition. That difficulty admits of explanation, however, in this way,—that both ventricles were clearly weak, and the right one more so than the left, so that from weakness the latter was not able to send the blood forward with such tension as to cause either marked reduplication or accentuation of the second sound.

Dr. Wyllie weighed and measured the heart with great care, as will be seen on reference to the report of the case. It is perhaps not safe to attempt any inference in regard to the normal heart from the condition of the diseased organ. Still, we may surely indicate what seems the direction towards which the condition in which this heart was found leads us. In the first place, the left auricle was dilated, but not hypertrophied, which agrees with Larcher's statements. In the second, the right ventricle was slightly hypertrophied. This is against Larcher's views of the healthy heart in pregnancy. But we have a ready explanation of its hypertrophy in the chronic bronchitis and emphysema of the lungs. Next, it is noticed that the left ventricle was slightly hypertrophied, which again supports Larcher's views. There was here no valvular lesion to induce hypertrophy, or any general cause, with the important exception of a certain amount of renal disease.

Those who deny a normal hypertrophy of the left ventricle of the heart during pregnancy, will of course maintain that the renal obstruction was sufficient to account for the existing hypertrophy in the left ventricle. To them I reply, that at any rate the condition of the left ventricle in this patient does not contradict, if it does not strongly support, the views of the French authors. It will be noticed that the weight of the heart as a whole was about one-half greater than normal. Besides this, in two cases I have already referred to, where there was no chronic renal disease and cardiac lesion, we

have ourselves found unmistakeable evidence of hypertrophy of the left ventricle as a result of pregnancy, so that it appears to me that it is impossible not to grant that it normally occurs. Certainly this case rather favours than contradicts this view.

CASE XXX.—*Acute Endocarditis — Vegetation on Mitral Valve, leading to Capillary Cerebral Embolism and Convulsions—Labour by Accouchement Forcé—Death.*

(*Abridged translation from* AHLFELD, '*Archiv für Gynäkologie*,' *Bd.* iv. *S.* 158.)

Patient, aged twenty-two, had unilateral convulsions of the right side, due to what appeared apoplectic extravasations. Labour began spontaneously, but was completed by accouchement forcé, in consequence of the severity of the convulsions. The patient had some post-partum hæmorrhage, and died almost immediately afterwards. Section after death proved the presence of acute endocarditis. Vegetations were found on the mitral valve, varying from the size of a millet seed to that of a pea. Extravasation of blood was seen under the pia mater. Hundreds of capillary apoplexies were found in the grey substance of the brain, some about the size of a millet seed or pea, one the size of a lentil in the pons Varolii. The opinion that the symptoms were due to embolism in connection with the endocarditis, though it approached certainty, was not substantiated by finding an embolism. Ahlfeld thinks the emboli must have affected only the minute vessels. Microscopic examination could not be made.

I have included this case, although it is outside the scope of my paper, because it exhibits a phase of the risks of cardiac disease which has hardly been touched upon in the records of the other cases. The most like it is Ahlfeld's other one (see Case XXI.). It proves that in acute cardiac disease the risk is greater even than in chronic cases. In

fact, we are certain that the more acute the condition is when pregnancy supervenes, the greater is the risk, up till the period has arrived when the compensation that has been established begins to be disrupted, when the risks begin to increase again in the other direction. The explanation is obvious,—viz., that in the more acute cases the compensation has not had time to become established, and there still exists a greater tendency in the tissues to become again the seat of inflammatory action similar to that which originated the lesion. In conditions such as we meet with here, we experience the additional risk of embolism, from the escape of granulations from the valves, as seems to have actually occurred in the present instance.

CASE XXXI.—*Acute Rheumatism in Fourth Month—Abortion in the Sixth, followed by Ulcerative Endocarditis, and Embolism of the Left Subclavian, and of Abdominal Aorta and Common Iliacs—Death.*

(*Abridged translation from* LEBERT, '*Archiv für Gynäkologie*,' *Bd.* iii. *S.* 40.)

The principal points of this case are as follows:—

The patient, aged twenty-one, had an attack of acute articular rheumatism in the fourth month of pregnancy, and made only a slow recovery. She was admitted to hospital on 5th June 1861, in sixth month of pregnancy, being pale and cyanotic. Her pulse 120, weak, and her breathing shallow, with occasional mucous râles. Cardiac dulness increased, especially the transverse, which extended from the left border of the sternum to beyond the nipple. There was only very slight increase in perpendicular dulness. At the apex the first sound was partially covered with a soft systolic blowing murmur, and the second sound was clear, but accentuated in the pulmonary area. At the other orifices there was nothing abnormal. Tongue furred; thirst great; urine free from albumen. On the 6th a rigor came on,

followed by fever and palpitation, and pain in the epigastrium. This state of matters repeated itself on 7th, and during the feverish attack much breathlessness and præcordial oppression were present. Digitalis in small dose every hour, and mustard plasters over the abdomen were ordered.

On the 9th matters were worse: pulse, 104; cardiac dulness advanced to mid-sternum. Systolic murmur increased. In the afternoon, sudden pain seized the whole of the left upper extremity, with coldness and loss of motion. Sensation and motion returned in an hour, but relapse of attack occurred in the evening; pulsation could be felt in the radial, bronchial, and axillary arteries.

By 13th the patient had got tolerably well, when the pains attacked the right lower extremity, but without loss of motion. Weak pulsation in the crural artery. Improvement till 16th, when, after a violent rigor, labour came on, and ended, after a few hours, with the birth of a six months' female child. After labour a fresh rigor occurred, followed by collapse, and irregular pulse; breathing difficult; palpitations great. Patient died on the 18th, with every symptom of pulmonary œdema. Post-mortem examination revealed a flabby, dilated, large fatty heart; extensive ulceration of the mitral valve, more especially of the posterior cusp, which was completely destroyed. There were two large emboli in the left subclavian, one two inches long as it left the aorta, and another an inch long at the distal end of it. The former was loosely attached, the latter firmly held to its walls. At the bifurcation of the abdominal aorta another embolism was situated, which sent a prolongation into either iliac. The lungs were congested and very œdematous. Uterus normal.

Case XXXI. is also anomalous. It is not strictly one of chronic disease of the heart, as the rheumatism came on

acutely during the fourth month of the pregnancy. Still it seemed to me valuable, as presenting a fresh peculiarity of the bearing of heart disease upon pregnancy and parturition. It is to be noticed that the acute rheumatic inflammation did not run the ordinary course upon the endocardium. It is only fair, I think, to hold that this was essentially due to the co-existence of the pregnancy with the rheumatism. But be this as it may, ulcerative endocarditis was the consequence. This led to great destruction of the mitral valve by the ulcerative process and the formation of large emboli, first in the left subclavian and then at the bifurcation of the abdominal aorta.

There continued to be a constant alternation of improvement and aggravation of symptoms, till it culminated in premature labour at the sixth month, after which collapse and pulmonary œdema carried off the patient.

It is thus seen that acute endocarditis is a much more serious matter if it occurs in relation to pregnancy than if it exists apart from that state, and that it is very multiform in regard to the possible directions in which it may undermine existence.

I ought here to mention a valuable contribution to this part of my subject, which was made as early as 1853, by the late Sir James Y. Simpson. In the first part of his article on puerperal embolism, at page 523 of his Selected Obstetrical Works, short records of several cases are given, which very forcibly bring out the extremely great risk which cases of recent rheumatism run of being the subjects of fresh endocarditis, and of severe embolism during the puerperal stage.

Of five cases recorded by him, in which subsequent post-mortem examination proved the embolism to be due to the escape of cardiac vegetations, one of the patients had suffered from rheumatic endocarditis only a year before the commencement of the pregnancy in connection with which the

embolism occurred. Another suffered from an attack of acute rheumatism during the pregnancy, and died a few days after delivery. A third seems to me to have suffered from endocarditis, supervening only after her premature delivery. A fourth is clearly a case of endocarditis puerperalis ulcerosa; whilst in a fifth case the data given are not sufficiently full to enable us to determine the probable commencement of the lesion.

The records of the cases presented by Sir James are extremely interesting from our point of view, and it is striking to find in 1853 his notions on the question so fully matured. He clearly groups together conditions that ought to be differentiated, however, and of course was not aware of the very special tendency of puerperal endocarditis to become destructively ulcerative, and thus to lead to the rapid formation of arterial embolisms.

This tendency for fresh outbreaks of endocarditis, during the latter months of pregnancy and in the childbed week, to introduce new risks into the combination of chronic heart disease with pregnancy and parturition, is abundantly dwelt upon by Löhlein, as I have already pointed out. He does not, however, to my mind, sufficiently discriminate between the increased risk which is likely to arise in connection with cardiac affections of a more recent date, and that involved in the more chronic forms of the disease. It seems to me indisputable that inflammatory action in the endocardium, if it is not septic in origin, is far more liable to attack cases of heart disease which are of a comparatively recent origin than those which are of long standing.

In these latter cases, disturbances of compensation appear to me to be the evils of which one has most to stand in dread.

We have thus at length got to the end of our consideration of cases. The last three, having been all fatal, require no remark in the shape of comparison between their results.

## CHAPTER V.

### GENERAL CONCLUSIONS.

I NOW proceed, in accordance with the original scheme of the work, to the final part of it, viz. :—

To make some general observations, and endeavour to draw some practical inferences, in regard to the prognosis and treatment of cases of pregnancy, parturition, and childbed, complicated with chronic disease of the heart.

I have endeavoured faithfully to present before the profession this important subject in as complete a form as possible, in order to be able to derive from its consideration accurate conclusions, with the aim of replacing, if possible, by well-founded principles and opinions, the uncertain views that at present regulate the action of the obstetrical practitioner in dealing with cases of the kind.

Now, from a careful review of the whole history of the subject, I am satisfied that the balance of impartial evidence lies in favour of those who predicate a decided increase in the size of the left ventricle of the heart as a normal condition during pregnancy. I am at the same time inclined to believe that the hypertrophy will, on more extended inquiry, not be found exclusively restricted to the left ventricle, although it is essentially so, but will be found partially extended to the other chambers of the heart, particularly to the right ventricle. But the amount of increase of muscular tissue in the walls of any of the chambers other than the

left ventricle is, it appears, so slight as to be unable to prevent their yielding under abnormal tension during pregnancy, so that for practical purposes the hypertrophy of the left ventricle is the only hypertrophy that need be taken into account. But, while conceding this much, I am prepared to admit that the amount of tissue change may have been somewhat exaggerated by the French authors. Still, on the other hand, I am also constrained to affirm that there has, it appears to me, been a needless amount of doubt expressed on the subject by some authors, and especially by Fritsch and Löhlein, notwithstanding the careful statements of Larcher, Blot, and others. This difference of opinion is doubtless partly due to a recoil from the somewhat extravagant statements of Duroziez in reference to the results which he professes to have obtained from percussion, and which I believe to be untenable. My convictions in regard to the reality of hypertrophy of the left ventricle have been greatly deepened from those dissections which I have myself superintended, and have referred to above at pp. 18 and 19. It is idle to reject the anatomical evidence afforded by every case of death from uræmic convulsions, as Löhlein would do, for the renal disease that leads up to puerperal eclampsia is usually developed so quickly that it could never materially alter the bulk of the heart.

I am also led to believe that, on account of this hypertrophy, as well as on more general grounds, satisfactory evidence of exalted vascular tension in the pregnant condition, particularly during the latter months, may be unquestionably obtained. Thus there is the peculiar condition of the blood, which may be fairly held to act in the manner pointed out by Dr. Mahomed, so as to produce an exalted arterial tension. There is also the increased bulk of the vascular fluid, which must require greater *lifting* power to keep it in circulation, and that greater lifting power must be met by the

left ventricle of the heart. Then there is the evidence derived from observation of the well-known tendency of pregnancy to induce varix in different parts of the venous system. We must likewise take into account the testimony supplied by the readiness with which disordered conditions, especially of the chest, directly referable to over-distension, spring up, provided the uterus or abdomen or both are in any way abnormally increased in bulk. Over-distension is also apt to occur without conditions being present that one could well call pathological. Though the experiment of Volkmann, given above, pp. 38–40, may be fairly used for our purpose thus far,—viz. to throw more than a doubt upon the views of Spiegelberg, that any great and sudden depression of arterial tension can arise from the simple emptying of the uterus, inasmuch as from that experiment it really seems, *a priori*, probable that the tension of the arterial system would be quite as great after as before delivery,—and in my opinion it really is nearly but not quite so great,—yet the results must always be carefully estimated along with those brought out in the experiments of Fritsch, which show that fluid passing through channels of varying capacity is either retarded in its course or requires to be propelled under greater pressure. The apparatus of Volkmann was composed of tubes of uniform dimensions throughout, and consequently could give no indication of the effects produced by alternate dilatation and constriction of the tubes. It must be constantly borne in mind that the blood vascular system is a part of the living economy under the control of the nervous system, whereby the area of its entire section is constantly varying in amount in consequence of partial or general changes in the lumina of the smaller arteries. The effects of these changes it is impossible to imitate or estimate by the most complex hydro-dynamic apparatus. It does seem to me probable that the placental and uterine

circulation must to a certain degree tend to heighten the general arterial tension; but, at the same time, it appears from the experiments of physiological observers that the sudden obstruction presented to the circulation by the powerful contraction of the empty uterus would, on the other hand, tend to increase the vascular tension quite as much as the presence of the placental circulation had done, so long as the quantity of blood in circulation throughout the body was not sensibly diminished, which it is not after an ordinary labour with no post-partum hæmorrhage.

I have been therefore forced to the conclusion that, on general grounds, there is evidence of increased vascular tension during the latter months of pregnancy, and I accordingly proceeded to institute a series of sphygmographic observations, with the view to determine whether these did not support the inferences derived from other sources.

It is scarcely necessary to repeat what has been stated at p. 20, that in a matter of this kind one is beset with numerous sources of error, and with many difficulties in the way of forming a correct opinion. There has been, and to some extent still is, for instance, considerable difference of opinion as to what forms evidence of high tension in a sphygmographic tracing. The introduction of a special arrangement to determine the pressure with which the instrument is applied to the vessel at the time the observation is being made, and also to ascertain the amount of force necessary to entirely obliterate the impulse of the artery, is a very great step in advance. An easy method of effecting these objects is accomplished by Mahomed's sphygmograph, the instrument which I latterly used. But even with it there are, as stated above, numerous sources of fallacy, so that at best only a rough approximation can be arrived at, and fine shades of difference in tension cannot be determined with certainty. This much, however, can be affirmed with safety,

viz., that with Mahomed's instrument one ought never to be in doubt on the question of whether a given pulse is of low or of high tension. It may be too much to expect the instrument to decide for us the exact amount of arterial tension in ounces in the face of so many obstacles, but, if properly adjusted, within certain limits it returns a positive answer to the question whether the tension is high or low.

The appearance of a pulse tracing is liable to vary greatly according to the accuracy or otherwise with which the instrument is applied over the vessel, and the degree of tightness with which it is fixed to the arm. Thus one may get a very different-looking tracing from the same pulse at the same time, provided any of these other arrangements vary, yet the tension must have been the same throughout.

In the earlier part of my sphygmographic observations I employed Marey's instrument, and found it extremely difficult to come to any fixed or definite ideas on the subject. These difficulties, however, have been greatly lessened by the use of Mahomed's sphygmograph, which, whilst it is more easily applied and retained in position, gives very much more exact information, and checks the results arrived at from the consideration of the appearance of the tracing, by the arrangement which registers the pressure at which the instrument is working.

The tracings were taken in the usual way, on paper smoked in the flame of turpentine, and were afterwards fixed by running some photographer's varnish over the surface of the paper.

The blocks from which the tracings have been printed were prepared from the originals by first photographing the latter on the wood, and afterwards cutting out the photographed line. Absolute accuracy of reproduction of the originals is thereby secured.

Those tracings in connection with which the exact amount of tension is not stated, were obtained by me before I had become acquainted with Mahomed's sphygmograph.

My observations on the subject, as already stated, lead me to conclude that the pulse tension in the latter months of pregnancy is high, that it decreases somewhat immediately on delivery, but that during the lying-in period it is still high, —at least fully as high, even though the patients are kept specially quiet and recumbent, as the pulse of non-pregnant women in full health,—and that it slowly returns to the normal during the lying-in period.

The differences are not, however, very great, and we have to be constantly on our guard not to allow attachment to theory to overmaster our duty to truth, or to lead us astray in the manipulation of figures, which are proverbially obedient to the most divergent methods of handling. It may be said in favour of the conclusion here arrived at, that my observations have partially altered my previous opinions. Till they were made, I did not believe that the difference in arterial tension before and after delivery was so decided as I now regard it to be.

I need not state the exact tensions except of those tracings that are given below, as I regard the numbers expressive of the tension to be possessed of little actual value, and to be merely of relative importance; but I may be allowed to state that, in my efforts at the attainment of a solution of the question, I have made a large number of observations under three classes:—

1. On patients shortly before labour.
2. On patients within the first two days after delivery.
3. On patients more than two days after delivery.

Ever since I began to employ Mahomed's sphygmograph, care was taken in each case to find, with as great accuracy as possible, what was the working pressure which developed the

best tracing on the smoked paper, and this was noted on the tracing as the working pressure.

The pressure necessary to obliterate all action of the artery was then ascertained, and also similarly noted.

Now, the results of these observations may be briefly stated as follows:—

The patients whose tracings were obtained before delivery gave, with Mahomed's instrument, an average working pressure of 5·6 ounces, and an average obliterating pressure of 16·9 ounces.

The patients examined within two days after delivery gave an average working pressure of 5 ounces, and an average obliterating pressure of 16 ounces.

The patients examined later in the childbed week returned an average working pressure of 4·8 ounces, and an average obliterating pressure of 13·5 ounces.

An examination by the same instrument of the pulse of twelve healthy non-pregnant women, whose hearts were perfectly sound, yielded an average working pressure of 4 ounces, and an average obliterating pressure of 13·5 ounces. These observations were made continuously, without any attempt to select the cases.

These results are not so complete as I could wish, especially owing to the fact that I have not the advantage of being physician to a maternity hospital, where such observations could readily be made in large numbers and continuously, but they have satisfied me of the truth of the general statement made above in regard to the vascular tension of the pregnant and puerperal woman.

I now proceed to give, in the first place, a selection of pulse tracings from women far advanced in pregnancy, and whose hearts were perfectly healthy. These will then be followed by a similar series obtained from lying-in women with normal hearts, examined during various stages of the

childbed period. The period before or after delivery at which the observations were made, will be noted in each case.

It may be safely said that all authorities—even those who consider the pulse of the puerperal patient as normally of low tension—regard the pulse of the pregnant female near the termination of utero-gestation as one of high tension. It is, however, of some service to science to prove it to be a fact, and I think the figures and statements that follow help towards that end.

No. 9.—Mrs. Y., aged 42. Before labour at full time. The tracing is characterized by a well-developed tidal wave and a perpendicular, well-proportioned up-stroke, both indicative of good tension.

No. 9.

No. 10.—M. T., aged 26. Multipara. Taken at 8½ months. Also exhibits evidence of good tension.

No. 10.

No. 11.—Mrs. G. Primipara. In the middle of ninth month. Indicates a steady pulse of good force.

No. 11.

No. 12.—M. B., aged 20. Primipara. Pulse 68. Working pressure 5 ounces; obliterating pressure 15. Before labour at full term. Well-marked and full tidal wave; dicrotic wave feebly indicated.

No. 12.

No. 13.—M. B., aged 22. Primipara. Taken two days before labour. Working pressure 7 ounces; obliterating pressure 19 ounces. Indicates specially high tension.

No. 13.

No. 14.—J. S., aged 20. Primipara. Rate, 72 per minute. Working pressure 6 ounces; obliterating pressure 19 ounces. Taken five days before labour. Notch between the tidal and dicrotic wave very peculiarly well marked.

No. 14.

No. 15.—M. G., aged 19. Primipara. Rate, 92 per minute. Working pressure 6 ounces; obliterating pressure 14 ounces. Taken four days before labour. Tidal wave

unusually defective, possibly owing to the rapidity of the pulsations.

No. 15.

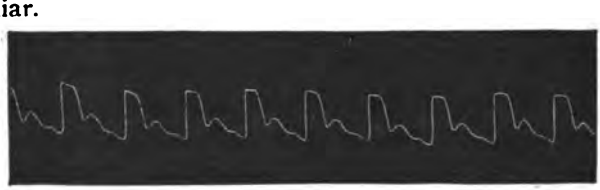
No. 16.—J. R., aged 21. Primipara. Rate 110. Working pressure 6 ounces; obliterating pressure 14 ounces. Taken the day before delivery at term. Very similar in character to No. 15.

No. 16.

No. 17.—M. P., aged 23. Multipara. Rate 72. Working pressure 5 ounces; obliterating pressure 14 ounces. Taken at term.

No. 17.

No. 18.—E. C., aged 45. Multipara. Working pressure 5 ounces; obliterating pressure 14 ounces. Taken at term. Patient insane. Relation of tidal to dicrotic wave very peculiar.



No. 18.

No. 19.—C. T., aged 24. Primipara. Rate 68. Immediately before labour. Working pressure 5 ounces; obliterating pressure 14 ounces.

No. 19.

The question of the tension of the pulse during the lying-in week has, unlike that of the pulse during pregnancy, given rise to much discussion, chiefly in connection with the slowness of pulsation so peculiar to that period. M. Blot and M. Marey,[1] supported by the weighty authority of Dr. Mahomed,[2] agree in regarding the pulse of the lying-in week as one of high tension. Dr. Meyburg,[3] in a recent paper, flatly controverts this opinion, and maintains that the pulse of the lying-in woman is normally one of low tension. From Fritsch's views regarding the causation of the slow pulse in the lying-in week, he also must be regarded as maintaining that the vascular tension is feeble during that time.

Dr. Meyburg's argument has failed to convince me. It may be shortly stated thus,—that the pulse tracing of the lying-in-period is characterized by the following peculiarities:—

1st. A short and slanting primary wave, or what we call usually the up-stroke or percussion wave.

2d. This slanting up-stroke is followed by a marked deviation towards the right, giving rise to a low level though well-marked secondary wave, or, as we usually call it, tidal wave.

3d. This is followed by a slightly marked dicrotic wave.

He then argues that such a pulse tracing must necessarily

[1] *Bulletin de l'Academie de Médecine*, t. xxviii. p. 926. 1863.
[2] *Medico-Chirurgical Transactions, London*, vol. lvii. p. 223. 1874.
[3] *Archiv für Gynäkologie*, Bd. xii. S. 114.

indicate low tension, inasmuch as he found what appears to him a similar tracing in that of a hemiplegic patient whose case is recorded by Eulenberg, and whose vascular tension must have been feeble. As already stated, Dr. Meyburg agrees with other observers in regarding the pulse tracing before delivery as indicative of high arterial tension. Thus far I agree with him. But I disagree with him *toto cælo* when he deals with the pulse of the lying-in woman. I allow that the tension immediately after delivery is less high than before labour. But I maintain that it is not so much less as to entitle us to call it a pulse of low tension. Furthermore, I consider that it is a pulse of distinctly higher tension than that of the ordinary healthy woman, and I have failed to satisfy myself that it possesses the characteristics which Dr. Meyburg describes as peculiar to it. In my experience with the sphygmograph, a slanting and short up-stroke in such cases is only to be obtained by a defective application of the instrument. I appeal with confidence to the tracings which follow in support of this contention. I have not been able to get a low-level secondary wave. It will be noticed that the tidal or secondary wave is in general not so well marked in the tracings obtained after delivery as in those taken before it. The only exception is when the pulse rate is slow, and then the tidal wave is particularly full. But it will be observed that with this well-marked tidal wave and slow pulse there is invariably found to coincide a high working pressure and a fully average obliterating pressure. Besides, shortness of up-stroke is far from indicating a pulse of feeble tension. The length of up-stroke seems to measure rather the degree of capillary resistance, and to vary inversely with the latter. Some comparatively feeble pulses have a very high up-stroke. In the case of Mrs. Moodie, p. 88, it will be noticed that, in the tracing obtained after her delivery, a diminution of 2 ounces of working pres-

sure and of 3 ounces of obliterating pressure was accompanied by an extraordinary elongation of the percussion wave. Obliquity of up-stroke appears frequently associated with a weak pulse, but it is more often an evidence of defective application of the sphygmograph. I may, I believe, without hesitation affirm that in the pulse tracings of lying-in women submitted below, there is no indication of special slanting in the up-stroke.

I accordingly lay before my readers the tracings which follow, taken during the lying-in period, in evidence of the contention, that though the pulse of the lying-in period is one of slightly less tension than that of the pregnant patient during the latter months of utero-gestation, it is nevertheless not a pulse of weak tension, but still one of high tension. It will also be noticed that when specially slow, as some of them are, viz. Nos. 22, 24, and 26, the working pressures and the obliterating pressures are correspondingly high.

The tracings obtained within the first two days after delivery are given first; then follow four tracings taken at later dates.

No. 20.—Tracing indicating strong pulse of good tension. Patient aged 44. Multipara. Two days after delivery.

No. 20.

No. 21.—Shows a tracing of a pulse of good tension. Patient aged 35. Multipara. Taken 15 hours after delivery. Rate 76.

No. 21.

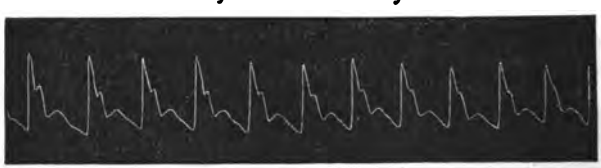
No. 22.—M. B., same as No. 12 of preceding series. Working pressure 5 ounces; obliterating pressure 17 ounces. Taken 14 hours after delivery. Rate 60.

No. 22.

No. 23.—M. C., aged 19. Primipara. Rate 80. Working pressure 6 ounces; obliterating pressure 19 ounces. Taken 18 hours after delivery.

No. 23.

No. 24.—J. S., same as No. 14 of previous series. Rate 92. Working pressure 5 ounces; obliterating pressure 18 ounces. Taken two days after delivery.



No. 24.

No. 25.—S. K., aged 20. Primipara. Rate 64. Taken 17 hours after delivery. Working pressure 6½ ounces; obliterating pressure 17 ounces.

No 25.

No. 26.—M. B., same as No. 13. Rate 62. Working pressure 6 ounces; obliterating pressure 18 ounces. 24 hours after labour.

No. 26.

No. 27.—M. G., same as No. 15. Rate 72. Taken 9 hours after delivery. Working pressure 6 ounces; obliterating pressure 15 ounces.

No. 27.

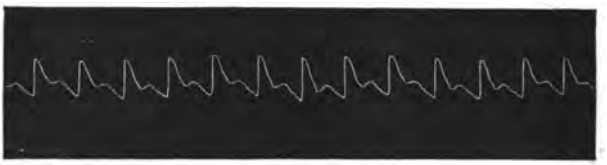
No. 28.—J. R., same as No. 16. Rate 92. Working pressure 4 ounces; obliterating pressure 12 ounces. Taken 19 hours after delivery.

No. 28.

The following four sphygmographic tracings were selected at random from a large number taken during the more advanced periods of the childbed week:—

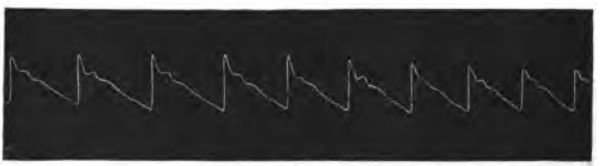
No. 29.—M. C., same as Nos. 23, 35, and 36. Taken 5

days after delivery. Rate 96. Working pressure 5 ounces; obliterating pressure 14 ounces.



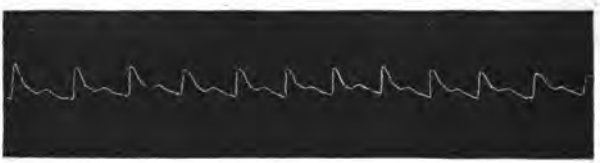
No. 29.

No. 30.—M. G., same as No. 15. Taken 5 days after delivery. Working pressure 5 ounces; obliterating pressure 14 ounces. Rate 80.



No. 30.

No. 31.—E. T., aged 19. Taken 9 days after delivery. Rate 100. Working pressure 5 ounces; obliterating pressure 13 ounces.



No. 31.

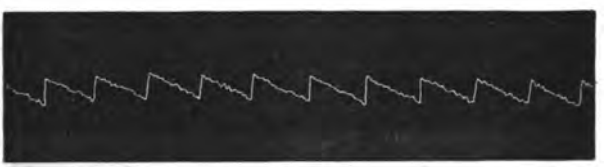
No. 32.—C. H., aged 22. Primipara. Taken 5 days after delivery. Rate 72. Working pressure 4 ounces; obliterating pressure 14 ounces.

No. 32.

These observations appear to indicate, as I have already asserted, a tendency towards gradual reduction of tension as the childbed period advances.

I shall finish by submitting four tracings which were obtained during labour. They are here given with the view of demonstrating the effect of the pains in increasing the tension of the pulse. This is seen chiefly in the greater fulness of the tidal wave, and in the nearly entire absence of the dicrotic wave. The steadying influence of the stronger pulsation during pain is seen in tracing 34, by the marked absence of vibratile tremor, and the much better marked tidal wave, when compared with tracing 33, obtained from the same patient during an interval between the pains :—

No. 33.—Mrs. Reid, aged 22. Taken during an interval between the pains in the first stage of labour, which supervened in the seventh month of pregnancy.



No. 33.

No. 34.—From the same patient. Taken during a pain. Tracing shows that lever has been much more steadily pressed upon.

No 34.

No. 35.—M. C., same as 23. Taken during a pain in first

stage. Working pressure 6 ounces; obliterating pressure 19 ounces. Rate 70.

No. 35.

No. 36.—From same patient deeply under the influence of chloroform. Taken during pain. Working pressure 7 ounces; obliterating pressure 19 ounces. Rate 70.

No. 36.

The difficulty of managing the instrument under the action of the pains is, however, so great that very little reliance can be placed upon tracings so obtained. The last two tracings on this account seem scarcely reliable, as they bear evidence of not being well taken. The pressure required to develope No. 36 is so great, that one is forced to believe that even with the sedative influence of chloroform the pulse tension was very high,—much more so than the character of the tracing would seem to indicate. Besides, to obtain correct results under such circumstances, one would require to be able to adapt the working pressure to the pulse during the acmé of each pain. This being impossible, the instrument works throughout the pain with the same pressure as gave the best tracing during the interval, which cannot be expected to bring out well the tracing of the pulse whose tension is suddenly increased by the pain.

I therefore conclude that physiological and clinical facts

tend to show that during pregnancy, especially during the latter months, arterial tension is increased, and that it is only slightly diminished during the lying-in period. According to the observations of Mahomed, it returns to the normal after about ten days. I do not mean to infer that the exhaustion of a very long labour may not be followed temporarily by low arterial tension, in the same way as any severe exertion is known to be. That the vascular tension is high during parturition, particularly during the second stage, when the down-bearing effort is present, may be held as a fact unquestioned by anybody. This normally increased vascular tension, though it is not of an amount to do harm during pregnancy if the heart is sound, cannot be considered without risk when the heart is diseased.

The spirometric observations of Wintrich, and the chest measurements of Dohrn, appear to me to establish it as a tolerably well-ascertained fact, that the vital capacity of the female chest, when the pregnancy is in all respects normal, is nearly, if not quite, as great the week before delivery as the week after it.

It is always to be remembered, however, that if any undue abdominal distension occur, either on the part of the uterus—say, if it is unusually distended—or from the presence of some distending force acting continuously within the abdomen, the vital capacity of the chest is extremely liable to be lessened, and in this state of matters we find the physiological condition of pregnancy border very closely, and indeed overlap, the region of pathology. So much for physiology.

We now proceed to pathological considerations.

A collection of 31 cases of pregnancy complicated with disease of the heart, selected from various sources, and presenting a mortality of 17, that is, 55 per cent. (or, if we regard Mrs. A., Case XV., as a death from heart disease and not from phthisis, of 58 per cent.), of itself

proves the combination to be extremely grave in its results.

There are, however, several considerations that diminish the value of such a collection as an expression of the average results, and in the interests of truth these ought to be pointed out.

In the first place, many of these cases were undoubtedly specially severe, and are, indeed, some of them examples of extremely serious heart disease, that would have speedily terminated fatally independently of pregnancy or of any other complication.

Secondly, it is manifestly inaccurate to calculate a ratio of fatality from the relation subsisting between the absolute number of deaths and the numbers of the terminal pregnancies. It would obviously be necessary, in order to obtain a just conception of the probable fatality of the aggregate and of the individual lesions, to add to the number of the pregnancies, in the case of the multiparæ, the sum of the pregnancies taking place between the *origin of the cardiac disease* and the *fatal pregnancy*. But unfortunately this is simply impossible, from the difficulty of determining the exact period at which the cardiac lesion commenced in most of the cases, so that we have to be content with the less accurate statement.

But, after all the deductions that the most scrupulous regard for accuracy and truthfulness require us to make, there is left such an amount of fatality in connection with cases presenting no purely obstetrical cause likely to lead to increased mortality, as leaves it beyond question that the combination is extremely liable to prove fatal. This is made more evident when we notice so frequently that the patients *before* becoming pregnant, and in the *intervals between* the pregnancies, enjoyed wonderfully good health; and this opinion is further strengthened by the large amount of fatal

cases that occurred in the primiparous patients, viz., so far as I can make out from the records, 9 or 10 out of 17 cases.

The effects of pregnancy, parturition, and childbed upon heart disease, and of heart disease upon these conditions, are mutually injurious, and they require to be constantly considered together.

The evils likely to arise from pregnancy in connection with cardiac lesions seem to be essentially referable to two classes—

Firstly. Destruction of that equilibrium of the circulation in heart disease, which has been established by compensatory arrangements. This result seems intimately associated with the high vascular tension and coincident hypertrophy of the left heart present during the latter months of pregnancy.

Secondly. Introduction of fresh inflammatory lesions upon the valves and endocardium of a heart already weakened by disease. These changes may either assume the type of ordinary plastic endocarditis, or the inflammation may follow that of ulcerative endocarditis.

Destructive results from rupture of compensation may be expected to arise either in connection with cardiac lesions of comparatively recent origin, in which the compensating arrangements have not been fully established at the time when the pregnancy commenced, or in conjunction with advanced cardiac disease, in which there is already developed a greater or less tendency to rupture of compensation from degeneration of cardiac tissue and other causes.

Inflammatory affections, on the other hand, would appear to attack by preference cardiac cases of more recent origin.

In connection with all cases of heart disease complicating pregnancy, but especially with those in which acute symptoms of endocarditis appear, there is great danger of embolism.

The fact is also to be constantly kept in mind, that

pregnancy is apt to act injuriously, not only upon the valvular arrangements of the heart, but upon the *whole blood vascular system*, and even upon the muscular tissue of the heart itself. This is especially made manifest by the researches and cases of Lebert. It ought also to be constantly borne in mind in judging of the probable effects of pregnancy in every case of cardiac disease, and it has important relations towards the condition of the lungs and kidneys, especially in regard to the production of hæmoptysis, the development of albuminuria, and the causation of eclampsia.

Slight exposure to cold and exertion in the case of pregnant patients who suffer from heart disease is very liable to set up destructive and dangerous pulmonary disturbances, which may give rise to pulmonary œdema, congestive bronchitis, hæmorrhagic infarctions of the lungs, pneumonia, pleurisy, nephritis, etc.

But independently of such exciting causes, there appears to be engendered by the pregnant condition a special liability in such patients to become sufferers from these diseases.

These pulmonary and renal affections in their turn react extremely unfavourably upon the cardiac disease, and tend to precipitate the disruption of a feeble compensatory arrangement.

The records of most of the cases contained in these pages agree completely with the statements made by M. Peter, that the dangerous pulmonary symptoms do not usually appear till after the first half of pregnancy has been run.

In severe cases it is seldom that we find pregnancy continue uninterrupted to the termination of the ordinary period of utero-gestation. It is extremely striking to look back to the records of the cases, and observe how very frequently premature delivery occurs spontaneously, thus :—

No fewer than nine of the fourteen presystolic cases were

prematurely delivered, the labour supervening spontaneously in every one of them.

One of the eight systolic cases was certainly prematurely delivered. But I am somewhat at a loss to make out, in Fritsch's fourth case (Case XXII., p. 136), whether it was premature or not.

Of the six aortic cases, five were prematurely confined. In four of these the labour occurred spontaneously; in one, accouchement forcé was performed on account of convulsions.

Of the three irregular cases, two were prematurely confined, the labour coming on spontaneously.

The most distressing subjective symptoms are found to be dyspnœa, a sense of suffocation, precordial anxiety, and, in aortic cases more especially, a very marked feeling of syncope during delivery. These, along with palpitation and cyanosis and other threatening symptoms, are seen usually to attain such a height, at a period within the third trimestre of pregnancy, that the accoucheurs in attendance have usually resolved to induce labour. But in every one of the cases recorded by me, this intention will be seen to have been curiously anticipated by the onset of spontaneous premature delivery, except in Case XXV., which was complicated with convulsions, and in Case XXX., which was one of acute endocarditis, with apoplectiform convulsions, in both of which accouchement forcé was practised. These cases, however, both terminated fatally.

In all the cases of heart disease which have been recorded in this work, it will be observed that if the lesion was at all severe, the labour was found to be invariably accompanied with extreme cardiac irregularity, with, also, a feeble, irregular, and intermittent pulse, much dyspnœa, and cyanosis. In a certain proportion of cases unconsciousness was noticed, the patients having the appearance of persons under the influence of chloroform. In some cases the perturbation of the

circulation was such as to end during the labour in sudden death. More frequently, however, we notice that the confinement was tided over, and a temporary but very frequently delusive improvement succeeded it.

In the case of aortic insufficiency, it is found that with the delivery the serious symptoms disappeared in three out of six cases. The amelioration of symptoms is so strongly pronounced in these three cases, that we are warranted, to a certain extent at least, in holding with Spiegelberg, that if the risks of labour are got over tolerably safely, except the case is all the worse, as in our XXIV., XXV., and XXVI., the prognosis may then be favourable, but guarded. Much more seems to depend upon the severity of the individual cases than the distinguished German obstetrician appears to allow. The results of our six cases, being three deaths and three recoveries, certainly do not lead us to expect any very special immunity for aortic cases of a severe form, even after delivery is safely effected.

But if the lesion is mitral, there appears to be fully as much risk of death during the lying-in period as during the pregnancy, provided the diseased conditions are severely developed.

Again, lesions of the mitral, in which constriction is the main element, appear from our cases to be more dangerous than when the leading defect is of the nature of insufficiency of that valve. But the cases recorded are too few to permit us generalizing much from them.

When death results in cardiac cases, the post-mortem examination reveals almost invariably pulmonary congestion, especially of the bronchial mucous membrane, and pulmonary œdema. Often, also, we find apoplectic extravasation of blood into the lungs, of recent or of older date, and occasionally pneumonia, with, very frequently, pleuritic effusions. The above results occur independently of any acute changes

in the heart. But, in a certain proportion of such cases, acute, plastic, or ulcerative endocarditis introduces fresh elements of danger, and if the latter occurs, embolisms in various parts of the vascular system are almost certain to be produced.

The view urged by Spiegelberg, that the sudden removal of the placental circulation occasions great diminution of resistance in the arterial system, leads to sinking of aortic pressure and exaltation of venous pressure, and thereby to sudden death during delivery, is, to my mind, essentially untenable. To this conclusion I am led more particularly by the evidence of the sphygmograph that the pulse tension is still high during the lying-in period, and by the study of Volkmann's experiment, which, abstractly considered, in no way supports such an idea ; as also by the fact that physiological experimenters agree in maintaining that when a considerable portion of the arterial system is suddenly obliterated, the effect is to heighten the tension in the rest of it. Besides, I have failed to find in my experience such a difference between the casualties resulting from cases of aortic and of mitral lesion as would warrant me in entertaining strongly the opinion of diminished arterial tension immediately after delivery. I believe it is somewhat less after labour than it is before labour, but not much so, and never to such an amount as could be expected to perturb seriously the action of any heart. Indeed, it seems to me impossible to conceive how the slight diminution in arterial tension following delivery can ever act otherwise than beneficially.

The evil effects of pregnancy in heart disease seem to be due mainly to the fact, that in the latter part of the period of utero-gestation more work falls to be done by the heart than previously. It is effected under an increased arterial tension, owing to the increased size of the left ventricle ; the quantity in the heart in a given time is

slightly greater than in the non-pregnant condition, and the remaining three chambers, while required to do more work, are not correspondingly strengthened by addition of muscular tissue during the pregnancy, as the left ventricle is. These effects, though slight in each individual factor, all act in the same direction, and tend to aggravate blood stasis in the lesser circulation. If incompetency of the mitral is present, its effects are added to the other pernicious influences, as the enlarged ventricle then pumps back the blood under increased tension upon the pulmonic circuit.

It seems to me that the evil effects are most prominent in the case of mitral stenosis, because all the factors that conduce towards a disastrous issue act continuously in the direction of producing dilatation of the left auricle and of the right heart, which is always the tendency of this lesion. The pulmonic circuit is thus kept, in severe cases of this lesion, in a state of continuous congestion.

Mitral insufficiency, on the other hand, is not so serious, as the pulmonic circuit is kept less continuously under tension; and it is an acknowledged general law that vital tissues will long bear an amount of interrupted strain or pressure, when they will speedily succumb to a much smaller application of a continuous force.

At the labour the chief evil effects seem to me to be produced from the exhaustion of the weak heart by the extra strain it is subjected to during the second stage. This effect is great, and appears to me greater and graver the more I observe and study these lesions, as the exhaustion and extra strain produce destruction of compensation in the heart, imperfect filling of the left ventricle, over-distension of the other chambers, especially of the right ventricle, congestion in the pulmonic circuit, imperfect aëration of the blood, extreme irregularity of the pulse, unconsciousness, and even death in some cases.

During the lying-in period we have still the evil effects of the defective compensation rendered operative by the persistent action of the powerful left ventricle; and it must further be remembered that the evil consequences of the severe strain of the labour upon the weak and diseased central organ is very difficult to recover from, and is doubtless frequently never surmounted.

In this manner we explain the marked tendency of mitral lesions to prove fatal during the lying-in period, when on superficial observation one might consider himself warranted in expecting recovery. In some cases, doubtless, an endocarditis is set up before the delivery, the pernicious results of which continue themselves into the lying-in period.

On the whole, therefore, our experience of mitral lesions in connection with the lying-in period is such as does not warrant a favourable prognosis for at least three weeks after delivery. The cases seem, from one cause or other, quite as likely to go wrong for ten days or a fortnight after delivery as they are during pregnancy or parturition.

In the case of aortic disease, the chief dangers are complications leading to suddenly increased aortic tension during the latter months of pregnancy, and the extra strain of the bearing-down efforts at the confinement. If these latter are got safely over, and if the case is not hopelessly severe, either in itself or in its complications, there seems reason, from the history of one-half of our cases, to anticipate good results. But the cases of this lesion complicating pregnancy as yet recorded are not sufficiently numerous to warrant us in making any very positive statement on this point.

Any undue distension of the abdomen, by encroaching upon the thoracic space and limiting the movements of the diaphragm, is apt to introduce most serious disturbances into a case complicated by cardiac disease, because it introduces additional difficulty into the circulation within the

pulmonic circuit. Such conditions, when removable,—for instance, if due to hydramnios,—ought to be treated. Under such circumstances only is premature labour warranted in connection with cardiac disease.

I cannot see any substantial ground to believe, with Fritsch, that the right side of the heart is ever specially empty after delivery. I am decidedly inclined to believe that on that point Spiegelberg has reason on his side when he declares that the right side of the heart is distended. Pathological evidence appears to favour this view. I believe it will be noticed that in post-mortem examinations the right side of the heart, and indeed the lungs also, are invariably found congested.

Besides, there is not evidence to prove that there is the reflux of venous blood into the abdomen after delivery which Fritsch seems to think there must be. There is reason, on the other hand, to believe that the tension of the vena cava inferior is always greater than that of the abdomen, which Schatz maintains it to be.

At any rate, cases fulfilling the conditions which Fritsch predicates seem to me to be very few and far between. His arguments from the beneficial results derivable from the application of a sack of sand, or of a hard board, to the abdomen in cases of bleeding post partum, are disposed of sufficiently by the observation of Löhlein, that in such cases the post-partum hæmorrhage is much more likely to be prevented by the irritation of those bodies stimulating the uterus to contract, than by the influence of their weight in restoring the equilibrium of the abdominal pressure.

But these theoretical problems are extremely difficult of solution, and we require much patient investigation before they can either be completely established or refuted.

I shall now conclude my subject by one or two practical

deductions from the views that have been maintained in the preceding pages:—

1. Chronic heart disease ought to be looked upon as a grave contra-indication of marriage, more especially if it assumes the form of anything approaching to severe stenosis of the mitral, or to serious aortic incompetency; in such cases we ought, if consulted, to dissuade from marriage.

2. There is less danger in the case of mitral insufficiency pure and simple. But still the risk is even then considerable.

3. In all cases when consulted, we ought not to give our sanction to marriage if in connection with chronic heart disease there are any serious symptoms of cardiac disturbance present, such as attacks of dyspnœa, breathlessness, palpitation on exertion, hæmoptysis, etc.; and this injunction ought to be the more imperative the younger the patient, and the more recent the acute disorder which has given rise to the chronic lesion.

4. Such patients as are married and have chronic heart disease ought not to be allowed to suckle their children, as that appears to tend to keep up the cardiac hypertrophy and increase the risks likely to arise from the defective heart.

5. All possible causes likely to produce inflammatory action in the lungs, and all severe exertion, should, if possible, be avoided during the pregnancy, and more particularly during the latter months of it.

6. Premature labour should seldom or never be recommended, because it is so much more likely to do greater harm, by disturbing the action of the heart and the condition of the lungs, than any good it might produce by terminating the evil effects of the pregnancy. It is always to be remembered that relief of symptoms is not certain after delivery, or indeed anything like certain.

7. The only conditions which seem to warrant the induction of premature labour are the presence of influences which

unduly distend the abdomen, and thus keep the diaphragm in a state of continuous elevation.

8. The same general principles of management ought to guide us in the case of a patient with chronic heart disease during pregnancy and the lying-in period, as are followed by us in dealing with patients who suffer from heart disease apart from pregnancy.

9. In almost all the cases I have met with chloroform has been given, and apparently with benefit, during delivery. If carefully administered, I think it cannot but be useful in all cases. My reasons for this view are given above, page 79.

10. All legitimate means ought to be used to lessen the effects of the down-bearing efforts, and therefore the judicious and timely application of forceps, or in suitable cases the performance of version, is extremely important if the second stage of labour happens to be in any way prolonged. In case of a large amount of liquor amnii, timely rupture of the membranes, as in Case XXIV., is calculated to be of great service, as it allows the diaphragm to descend, and thus lessens the embarrassment in the lesser circulation.

11. Increased experience warrants me in believing that the mortality following pregnancy complicated by chronic disease of heart may be greatly lessened by due precautions during the pregnancy and delivery—especially during the latter.

## ON TWO CASES OF PUERPERAL PLEURO-PNEUMONIA.

THE following cases of singular interest occurred in my practice in the latter part of 1876. As they were observed with a considerable amount of care, as I cannot but think they are not of very usual occurrence, and as I believe, moreover, they may serve for encouragement to some medical brother in charge of patients similarly affected, I have deemed it advisable to publish them.

It is to be noticed that the one occurred in September and the other in December of the same year, and the first had completely recovered long before the other took ill, so that there could be no relation of community of origin. Yet the cases present a wonderful number of points in common in regard to their symptoms and course, the principal difference being that the second ran a shorter course, and did not present symptoms of phlegmasia alba dolens, which affection retarded so seriously the convalescence of the first patient. With these preliminary remarks, I proceed to record the cases.

### CASE I.—*Pleuro-Pneumonia—Endocarditis—Phlegmasia alba dolens—Recovery.*

Mrs. P. L., Edinburgh, æt. 26, was confined by me of her second child on the evening of the 10th September 1876. The child presented the vertex with the occiput backwards, and to the right sacro-iliac-synchondrosis, but the labour was

extremely easy. Though I lived close to my patient's house, and was early summoned to her assistance, when I arrived the head was already well down in the pelvis, and one good pain effected forward rotation of the occiput, and completed delivery immediately after my arrival. The child was a healthy female, and in every respect normal. Mrs. L. did well also till the evening of the 14th September, when she had a rigor after some undue exertion. Still she passed a good night, and felt better in the morning. On the 16th, and also on the 17th, her elder child was unfortunate enough to meet with a slight tumble down-stairs, and on each occasion Mrs. L. was greatly, though quite unnecessarily, frightened. In the course of Sunday (the 16th) Mrs. L. began to suffer from pain on the right side, just below the nipple. This at first was slight, but as it increased on the Monday (the 17th), I was sent for in the evening. My patient had appeared so well the previous day that I had not thought it necessary to visit her.

I found her suffering much from pain in the right side, as also breathless and pale. Temperature was 101°, respiration 40, and pulse 110. There was also pain in the right shoulder, and the characteristic symptom of the pleuritic stitch. There was a demonstrable amount of dulness at the right base, both before and behind, more so behind than before, with feeble breathing, but no increase of vocal resonance, and neither crepitation nor friction. The lochial discharge was red in colour, plentiful in amount, and free from any disagreeable odour. The tongue was foul. Urine rather scanty, but it contained no albumen. I ordered a poultice to the side, and a Dover's powder to be taken.

18th.—Patient very much as on the previous night. No friction to be heard. Temperature, 101·8°; pulse, 110; respiration, 40.

19th.—Patient passed a bad night, but feels slightly easier.

Pulse, 110; temperature, 101°; respiration still 40; pain in the chest slightly diminished. Expression of countenance anxious. Cough very slight. Dulness posteriorly decidedly increased, but no friction nor crepitation audible. In the course of this day the patient spat up a mouthful of red expectoration, and in the evening the temperature rose to 103°. The poultices were continued. Another Dover's powder was administered, and the patient slept a little better than on the previous night.

20th. — Respiration, 36; pulse, 100; temperature in the morning, 101·5°; in the evening, 102·5°. Expectoration characteristically prune-juice in colour, mixed with some gelatinous rusty sputum. It comes up with tolerable ease and in considerable quantity. Cough still slight. Pain on inspiration still present. No distinct friction murmur audible, though I heard what sounded suspiciously like a scrape once or twice below the right nipple. The dulness behind is markedly increased, passing up as high as the upper angle of the right scapula, and is very absolute; but the level of dulness does not appear to be altered by change in position. The dulness anteriorly is difficult to determine with precision, as the mamma is distended and tender. But, so far as can be made out, it is not extensive. The breath-sounds are extremely feeble and short over the region of dulness; but there is no distinct crepitation ordinarily audible, though now and then I was convinced I heard a distant crackle. Neither is there increase of vocal thrill nor of resonance. No tubular breathing can be heard. The breathing on the left is not markedly puerile. The same treatment continued. Diet light, and no wine.

21st.—Had a rather better night. Physical signs of right side about same as yesterday, only I heard with tolerable distinctness some crepitation over an area on the right back bounded externally by the lower half of the right scapula,

and internally by the spinal column. Slight increase of vocal resonance is also perceived there.

Morning pulse, 92; respiration, 33; temperature, 102·2°.
Evening „ 96; „ 30; „ 101°.

Dr. Matthews Duncan saw the patient at my request this afternoon. He likewise thought he heard an indication of friction over the spot in front where I had previously believed I had heard it. There were positively no uterine symptoms to be detected. We agreed to apply a blister (6 × 8) over the right side, and to give a diuretic containing five grains of nitre and a teaspoonful of Mindererus's spirit in each dose. There was some irritation of the urinary organs from the cantharides, though I had taken the precaution to insist on my patient drinking plenty of fluids during the night.

22d.—Patient feels on the whole better.

Morning pulse, 96; respiration, 30; temperature, 99·3°.
Evening „ 90; „ 28; „ 99·4°.

23d.— In the morning, pulse 84; respiration, 34; temperature, 99·4°. The evening temperature, etc., not taken. Tongue more clean; appetite a little better; expectoration free, the sputum chiefly of the prune-juice character; uterine discharge still red and copious, but it has no special fœtor. There is now crepitation audible along the whole of the right back over the region of dulness, and there is also a certain amount of increase of vocal resonance.

24th.—Pulse, 84; respiration, 30; temperature, 99°. Patient feels better. Dulness posteriorly absolute as high as the upper angle of right scapula, and it is considerably above the upper limit of the liver in front. Milk returning to the breasts. Expectoration still bloody, free, and copious; comes up without much effort. There is a soft systolic mitral murmur distinctly audible at both the base and apex of the heart. I then left for the country, and my friend Dr. Playfair took charge of the patient in my absence.

On the 27th he reports as follows:—The front of right chest is clear on percussion, with an occasional râle. Posteriorly, the state of the right chest is much as reported on the 24th; but on the left side a dull patch is found to exist, stretching from the extreme base to a height of about three inches. The percussion note is peculiarly high pitched, and wooden in character. On auscultation, no fine crepitation is audible anywhere, only an occasional coarse râle with inspiration. Vocal resonance slightly increased over the dull area. Sputum still of the prune-juice colour. Perspiration is copious. Tongue, except at tip and edges, coated with a brownish fur, but it is moist. Appetite is fair. Bowels moved twice to-day after taking some Tamar Indien.

28th.—Had a tolerably good night, but sleep was very broken. Complains of stiffness of left leg and pain in the left groin. The leg is not swollen nor tender; only a slight swelling can be felt in the groin, and this part is tender on pressure. Sputum now mixed with mucus, and not so red. Diet to be very light.

29th.—Improving on the whole. Lochial discharge has increased in quantity, and is of a brighter red colour than for days past. Ordered two glasses of claret per diem.

2d October.—Left leg swollen to about twice the size of the right; pain in the groin continues. The femoral vein is felt to be hard, swollen, and very painful on pressure, and along the inner surface of the thigh the internal saphenous is found to be similarly affected. Along the calf of the leg the branches of the internal saphenous are also felt to be hard, swollen, and painful, but not so much so as the vessels higher up the limb. The general condition of the patient does not seem to have been much impaired by the onset of the phlegmasia, her health having if anything improved, except that she does not sleep well at night. The leg was now ordered to have a lotion of lead and opium applied to it constantly.

and to be wrapped in cotton-wadding from the knee to the groin. A mixture of quinine and iron was also prescribed to be taken thrice daily.

3d.—The flat wooden note has entirely disappeared from the bases of both lungs. On auscultation, the respiratory murmur on the right seemed rather louder than natural, but no crepitation or rhonchus is audible, and vocal resonance does not appear to be increased. On the left side, about the middle of the lung, the breathing is remarkably bronchial, and accompanied by coarse crepitation on inspiration. Vocal resonance over this area is much louder than on the right side. Above and below the middle of the lung the vocal resonance and the respiration approach the normal, but both seem if anything rather louder than in health. The sputum is now almost entirely composed of mucus, with only a slight intermixture of blood.

8th.—I again took charge of my patient, and found the condition of the chest nearly normal. Not a trace of pain or other disturbance in the uterine region, but still there is a considerable amount of sanguineous discharge from the vagina. There is also some blood in the expectoration, but no moist sounds are audible in the chest. Left leg is painful. The veins of the groin are hard, and the leg is slightly swollen. Pain is also complained of on pressure along the course of the large vessels in the thigh. There is great pain on pressure over the right groin, the veins there feel corded and knotted; but there is little swelling of this leg. Pain is likewise complained of when pressure is made along the course of the vessels; but there is no enlargement of the superficial veins of this leg, and no elastic œdema. The legs are being fomented frequently, then rubbed with oil and laudanum, and wrapped in cotton-wadding. The tonic mixture and wine are being continued.

11th.—Condition very much as at last report. It is

noticed that the soft systolic murmur still persists, being heard both at base and apex. It is, however, more distinct at the apex than at the base. The left leg is nearly free from pain; the right leg is in the same condition as on the 8th. Discharge from vagina has nearly ceased. No uterine tenderness. Patient sleeps better.

13th.—Right leg in region of thigh considerably swollen and painful on pressure, also œdematous at the ankle. Left leg quite well. Patient ordered 10 minims of the tincture of the muriate of iron and 5 grains of the chlorate of potash every six hours.

15th.—Much improved.

19th.—Patient feels very comfortable. Tongue is cleaning. Lochial discharge slight, and yellowish-white in colour. Swelling in right leg now quite gone. There is still a little tenderness on pressure in right groin, and along the course of the large vessels in the thigh. Ankles are free from œdema. Appetite good.

2d November.—Patient able to be up. Expectoration still dark. She has, however, no cough.

29th December.—Patient still rather emaciated. Lungs are both normal. Slight blow is heard at apex (mitral area) of heart with the systole. Cod-liver oil was prescribed.

13th March 1877.—Mrs. L. continues to do well. The first sound in the mitral area is slightly muffled and prolonged, but there can scarcely be said to be a murmur present. The pulmonary second sound is somewhat intensified.

On the 14th November 1877, Mrs. L. called on me, and complained that she has felt a sense of tightness for some time back across the lower third of the sternum, and that she suffers from breathlessness on going up stairs. She is still thin, and slightly anæmic. At the mitral area there is heard a distinct soft systolic, somewhat musical murmur, which is also audible at the tricuspid area, but not so loudly.

The murmur is also heard at the base of the heart, but not so distinctly. There is no appreciable increase of cardiac dulness. The impulse is normal.

20th September 1878.—The cardiac symptoms have been decidedly increasing. There is a loud, rather harsh systolic murmur audible at mitral, tricuspid, and auricular areas. The left side of the heart is clearly hypertrophied, as the apex beat is thrown downwards and outwards, and the transverse dulness is increased towards the left. The second sound is accentuated at the base, and the action of the heart is rather irregular and thumping.

CASE II.—*Pleuro-Pneumonia—Endocarditis—Recovery.*

Mrs. B., aged thirty-six, was attended by me during her second confinement, on the 3d December 1876. Her first labour occurred on 12th November 1875, and on that occasion I had to terminate the case instrumentally, and needed a considerable amount of traction effort to bring the head through the bony outlet.

On the present occasion I was called at 7 A.M., and found that my patient had been in tolerably severe labour since 2 A.M. The os was fully dilated, and I accordingly ruptured the membranes immediately after my arrival, and administered chloroform. The head presented in the right oblique diameter of the pelvis, with the occiput posteriorly and towards the right. It had nearly, but not quite, come down to the floor of the bony pelvis, and the anterior fontanelle was very markedly lower than the posterior. In the course of the following two hours, however, under the influence of tolerably powerful pains, the occiput was pushed down and rotation of it forwards effected; and instead of, as before, too much extension of the head, an abnormal amount of flexion was developed. Being convinced by this mechanism that the outlet of the pelvis was abnormally tight for the

head, and also finding that the latter was not making satisfactory progress, I applied forceps, and, after one or two pretty strong pulls, succeeded in drawing the head through the outlet. I noticed that the posterior parietal bone had been pushed over the anterior in the line of the sagittal suture. The child was a healthy female, and did well.

On the night of the 4th of December, Mrs. B. felt a stitch immediately below the lower border of the false ribs, on the left side posteriorly. I saw her early on the morning of the 5th. Her expression was decidedly anxious. She complained of a catching pain on inspiration in the left side, which, as compared to the right, was rather fixed. Auscultation could only detect the slightest suspicion of friction occasionally. There seemed to be a shade of dulness at the extreme base, and pain was complained of in the left shoulder. Pulse was 96; temperature, 99°; respiration, 22. Skin moist; tongue clean and moist at edges, dry in the centre. There was no history of any rigor, nor of exposure to cold. Lochial discharge natural. No abdominal tenderness. In the evening the respiration was 22; pulse, 120; temperature, 100·4°. Poultices were applied constantly to the left side.

6th.—Loud dry friction is audible at left base, both anteriorly, posteriorly, and laterally. Milk is present in the breasts. Tongue and skin are moist. Pulse, 120; temperature, 100·4°; respiration, 30 in the morning. Pulse, 124; temperature, 100·8°; respiration, 28 in the evening.

7th, Morning.—Perspiration copious; sudaminal eruption. The friction at the left base is less audible, but the dulness is much more pronounced. Still it does not reach so high as the lower angle of the left scapula. Breathing somewhat bronchial, and vocal resonance increased. An occasional crepitation is heard beyond the friction sound. No expecto-

ration. Vocal fremitus is not increased. Temperature, 100·2°; pulse, 120; respiration, 30.

Evening.—Had motion by means of enema. No cough or vomiting present, but a mouthful of bloody sputum was expectorated. Temperature, 101·8°; pulse, 124; respiration, 46.

8th, Morning.—Dulness has advanced up to level of lower angle of left scapula posteriorly. Loud friction is audible over the dull area, and crepitation distinctly to be heard beyond the friction murmur. Breathing bronchial, vocal resonance increased over the area of dulness. On the right side an occasional crepitation is heard in front. The patient was not turned round to examine the right back. The first sound of the heart, at the base, is distinctly muffled. Urine is free from albumen. Temperature, 103°; pulse, 132; respiration, 36. A large blister was ordered to be applied over the left side. It rose well. The patient was at the same time ordered to take an alkaline diuretic, and to have mild, non-stimulating food, but no alcoholics.

Evening.—Feeling decidedly anxious about my patient, I asked Dr. Matthews Duncan to see her with me. We then found a slight degree of extra consciousness of pressure over the uterus, which, however, did not by any means amount to tenderness. Physical signs very much as in the morning, only that, on turning round the patient to the left side, an occasional crepitation, with bronchial breathing and some increase of vocal resonance, was heard at the right base. No friction was audible on the right. The heat rash was very general in extent, mottled, not unlike that of scarlet fever, chiefly confined to the trunk, and not affecting the vulva. Lochial discharge still normal. Dr. Duncan recommended, in addition to what had been done, the administration of a tablespoonful of castor-oil and a teaspoonful of turpentine. This gave rise to two copious stools during the

night. Temperature, 103·4°; pulse, 132; respirations not counted.

9th, Morning.—Slept occasionally during the night. Looks easier. Enjoyed some toast and tea this morning. Chest symptoms not worse. Temperature, 101·6°; pulse, 132; respiration, 34.

Evening.—Dulness at left base not increasing. Some crepitation audible at right base, where there is also dulness and increased vocal resonance. Temperature, 101·6°; pulse, 120; respiration, 28.

10th.—Chest symptoms as yesterday, only crepitation at right base posteriorly more distinctly marked. Patient slept six hours last night. Cardiac systolic murmur more loudly heard at the base, and rather harsh and grating in character. At apex (mitral area) a boom is heard with the first sound, but no distinct murmur. The second sound is clear. There is no increase of cardiac dulness. The cardiac murmur is suspiciously like friction. Bowels moved unassisted yesterday afternoon, and again to-day. Patient feels decidedly better. Is taking ten drops of turpentine thrice a day in water.

Morning.—Temperature, 102·8°; pulse, 125; respiration, 24.

Evening.—Temperature, 102·4°; pulse, 120; respiration, 24.

11th.—Had a very good night. Bowels slightly relaxed. Last night a mouthful of bloody sputum expectorated. The expectoration is usually tinged with blood, but is slight in amount, and there is scarcely any cough. Friction nearly quite gone from the left side. The dulness is also diminishing. Crepitation is still audible. Cardiac murmur persistent.

12th.—From this time the patient improved slowly, but uninterruptedly, as a glance at the accompanying chart will show. The signs of pleurisy and pneumonia gradually disappeared from both lungs.

The cardiac murmur, which, from its character and situa-

tion, led me to suspect the existence of pericarditis, gradually lost its harsh character, and developed into a very soft systolic blowing murmur, which was propagated in the direction of the inner third of the right scapula, forcing on me the conviction that it had been all along, in great part at least, if not entirely, endocardial.

On the 29th December I made the following note:—Patient doing well, and is to get up to-day. The systolic murmur is most loudly audible in the second intercostal space close to the left edge of the sternum. It is soft but distinct, and *is audibly* propagated along the course of the aorta and great vessels. It is audible at the lower edge of the inner third of the right clavicle, but not under the middle third of the left clavicle. It has now entirely lost the creaking and grating character it formerly possessed.

27th November 1877.—The patient feels quite well. The systolic aortic murmur still persists.

The two cases above related are the only specimens of this kind of lesion with which I have been called upon to deal in the course of practice. They both presented symptoms at once serious and threatening. The first gave me a great amount of anxiety in case the illness should terminate fatally; and the second, I must also confess, caused me great, though, compared with the first, less uneasiness. As I have already stated, it was the conviction that the record of these cases, if published, might prove a solace and comfort to some gynæcological physician, who might be called upon to deal with his first case of this kind, that prompted me to record them. I cannot think that conditions such as we find in the patients before us, possessing as they do so many special points in common, do not occur now and again, though I have not been fortunate enough to fall upon them either in practice or in reading. Dr. M. Duncan, who was

kind enough to see both cases with me, informs me that he has met with other two of late having a considerable resemblance to them. The one, however, was a case of pleurisy only, the other was one of empyema, complicated with perimetritis. The first case recovered. The second is still living, but in a very doubtful condition.

Let me now endeavour to trace the leading peculiarities of the cases.

We had nothing very special in respect to the labours in either case. The delivery in the first one was remarkably easy. In the second, it could hardly be called severe. I merely terminated the case with forceps to save my patient what I considered quite an unnecessary amount of suffering. Her previous labour had been rather difficult and severe, and yet nothing untoward happened on that occasion. I have drawn attention to the mechanism of the head in this case merely as a point of interest to the exact obstetrician. My patients were both in perfectly comfortable circumstances, possessing every necessary means to enable them to avoid exposure to cold or other injurious influences, both at the time of their delivery and afterwards; and the nurses were also trustworthy, careful, and intelligent, so that nothing in the management of the patients could have occurred contrary to the strictest rules of caution and propriety. Besides this, both patients were in good health at the time of their confinements. There was the influence of slight fret, on account of her elder child meeting with a first and then a second fall, which was dwelt upon by the attendants of the patient in the first case. But then we know that patients and their friends are very apt to place too much importance upon the influence of trivial incidents such as these upon the causation of disease. And, besides, there was no such cause even thought of in the second of the two cases. It is therefore extremely difficult, if not impossible, to discover any likely cause for

the seizures. In both cases the pain was slight at first, and gradually though rapidly increased in severity. There was no sudden seizure, with fainting and dyspnœa, in either case. I examined with very special care, in regard to the first patient, whether any sudden disagreeable feeling had arisen, when, on the first occasion of alarm about her child, it appears she sat up rather hurriedly in bed. But nothing of the kind could be elicited. She did not feel faint at that time, nor did she complain of pain in the side nor in the abdomen. Breathlessness was not experienced. In fact, she noticed no bad effects from this premature effort.

I mention these particulars, because my mind was strongly inclined to predicate the possibility of pulmonary embolism, if the facts were such as could be squared with that idea. But it is difficult to find sufficient grounds for such a belief, although I have sought very carefully for them. As negativing embolism, I ought to mention that there was no antecedent varicosity in the legs of either patient, and indeed nothing abnormal in the lower limbs other than a slight degree of that passive œdema of the legs which one almost constantly sees towards the end of pregnancy; and further, there was no distinct evidence of any metritic mischief in either of the cases. On the other hand, the course of the chest symptoms was very unusual. The friction, in the first instance, was extremely ill-defined, and the rapidity with which the dulness increased was very remarkable. The dulness in both instances was very absolute, and did not appear to be due to the effusion of serum, but to an exudation of a more solid element. At any rate, change of position seemed to make very slight alteration in its level. Pneumonia accompanied, if indeed it did not precede, the pleurisy in both cases, and yet it did not exhibit the ordinary phenomena associated with this disease. The vocal resonance was slowly and imperfectly developed. There was little con-

sonance of the breathing, and fine crepitation was never audible. Indeed, for days no crepitation of any kind was heard, and then it was distant and specially coarse for pneumonia. But the most peculiar point in regard to both patients was the presence of sanguineous sputum. The cough was in neither case severe, and the mucous expectoration was slight. But in both cases mouthfuls of bloody expectoration were frequently spat up without effort. In the second case this was slight in amount, but in the first case it was copious and long-continued, the prune-juice colour of the sputum forming a marked feature in the case, and the colour of the expectoration remaining darkish-red for months after the severity of the illness had passed off. There were thus very many points that could be explained on the assumption of embolic infarction of the lungs more readily than on any other consideration. For this would explain the bloody expectoration, the situation of the commencement of the lesion in both cases at the extreme base of the lungs, the sudden supervention of dulness without at first corresponding fever, and the defective amount, as well as the retardation of the appearance of the crepitation. The ultimate height attained in both cases by the temperature—about 103°—also would fall in with the embolic theory. But, on the other hand, the absence of severe dyspnœa, the gradual onset of the attacks, the want of evidence of any demonstrable metritic mischief or of inflammation in the right side of the heart, and the favourable termination of the cases, rather oppose the acceptance of that explanation. At any rate, if the cases were due to embolism, the plugs must have been of a purely non-septic character, and could scarcely have been the result of phlebitis affecting the pelvic organs. Still the embolic theory, though not very probable, is by no means excluded as an explanation of these peculiar cases. On reading with care Gerhardt's article upon hæmorrhagic infarction of the lungs,

*German Clinical Lectures*, vol. ii. p. 272, it cannot fail to be noticed that the local signs in my cases, so far at least as they were peculiar, are remarkably like the local signs given by him as characteristic of embolic infarction.

A limited thrombosis of some of the terminal twigs of the pulmonary artery would no doubt explain the facts of the cases. But, notwithstanding the able support which belief in the comparative frequency of thrombosis of this artery receives from the observations of Dr. W. Playfair, Dr. Meigs, and others, I do not feel warranted to put it forward as a probable explanation.

In both cases there was evidence of endocarditis, affecting in the first case the competence of the mitral valves, and in the second producing a certain degree of aortic obstruction. I am really at a loss to determine whether in Mrs. B.'s case there was not a certain amount of pericarditis superadded to the endocarditis, as the murmur was of so creaking and harsh a character and so strictly confined to the base. But be that as it may, there was undoubtedly a certain degree of endocarditis affecting the semilunar valves, as evidenced by the permanent obstructive murmur at the aortic orifice.

From the observation of cases like these, as well as in consequence of my researches on the bearing of heart disease upon pregnancy contained in the former part of this volume, I have been led to believe that the puerperal condition is apt to act very prejudicially on the vascular system generally, and to suspect that endocarditis occurs much more frequently in connection with the lying-in period than is ordinarily believed. I am very strongly inclined to believe that, though, luckily, ulcerative puerperal endocarditis is a rare disease, a slight amount of endocarditis in the puerperal state may be common, and has not been often observed because seldom looked for. At any

rate, since I met with the above cases, I have certainly seen a third, and I think, although I am not so positive in regard to it, a fourth case, in which there existed slight and transient endocarditis, as evidenced by some cardiac irregularity and feeling of discomfort in the cardiac region, with a loud, or at least distinct, blowing systolic murmur at the base. I do not mean by that the soft mitral systolic of the lying-in period, so much talked of by German obstetricians, who, I am convinced, however, exaggerate the frequency of its occurrence. My opportunities as yet have not been sufficiently great to warrant me to do anything more than merely throw out a suspicion regarding the frequency of slight non-pyæmic endocarditis in the puerperal condition. That we had to deal in both these cases with endocarditis, and that the murmurs were not merely functional in origin, is put beyond all doubt now, supposing such a view had ever been entertained, by the persistency in both cases of the murmurs after the lapse of many months. Indeed, it has now become evident that in Mrs. L.'s case the heart is suffering from chronic endocardial changes, for the murmur is much more loud, and is accompanied by a feeling of tightness under the lower part of the sternum, as also with breathlessness on exertion. Other symptoms of chronic cardiac disease have also established themselves unmistakeably in her case.

That the vascular system was generally in a condition prone to inflammatory change in Mrs. L.'s case, is further evidenced by the supervention of phlegmasia alba dolens, with its usual concomitants of corded and enlarged veins, and of tenderness along the course of the blood-vessels of the affected limb.

That venous thrombi, originating in a limb affected with phlegmasia alba dolens, may occasion in the first instance pulmonary infarction, and then lead indirectly to pleurisy,

pneumonia, and other results of an inflammatory nature in the lung, is abundantly insisted upon by Virchow, Trousseau,[1] Gerhardt,[2] and a host of other observers, so that I need not refer to this point, except to observe that, seeing that the phlegmasia only appeared after the chest symptoms began to subside, it is impossible to regard the latter as occasioned by the former.

But the credit is due, so far as I have been able to make out, to the elder and younger Begbie of having pointed out that phlegmasia alba dolens is not unfrequently either an antecedent or consequent of pleurisy, the swelled arm or leg appearing on the side on which the pleuritic lesion was situated.

Dr. Warburton Begbie[3] says, in speaking of four cases of acute pleurisy in which he performed the operation of thoracentesis: 'In one of those four, a young gentleman of twenty-four years of age, the pleural effusion was associated with a peculiar swelling of the corresponding limb, both in the leg and in the thigh. This swelling was not of an œdematous character, but firm, and resembling a good deal the condition of the extremity when affected by phlegmasia alba dolens; but Dr. Begbie informs me—and the observation appears to be very interesting—that in three cases of pleurisy occurring to him within a limited period of time, he has observed the limbs corresponding to the pleuritic side swollen. In one of those the lymphatic disturbance in the leg —for such it would appear to be—preceded the pleurisy; in the others was consequent on it.'

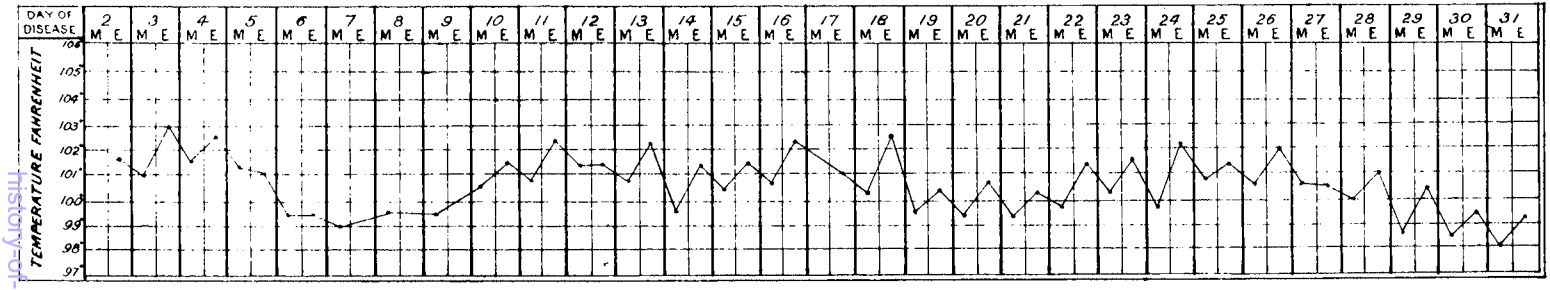
I am further informed by Dr. Matthews Duncan, that he has distinct recollection of phlegmasia alba dolens following

[1] *Clinical Medicine*, vol. v. p. 331.

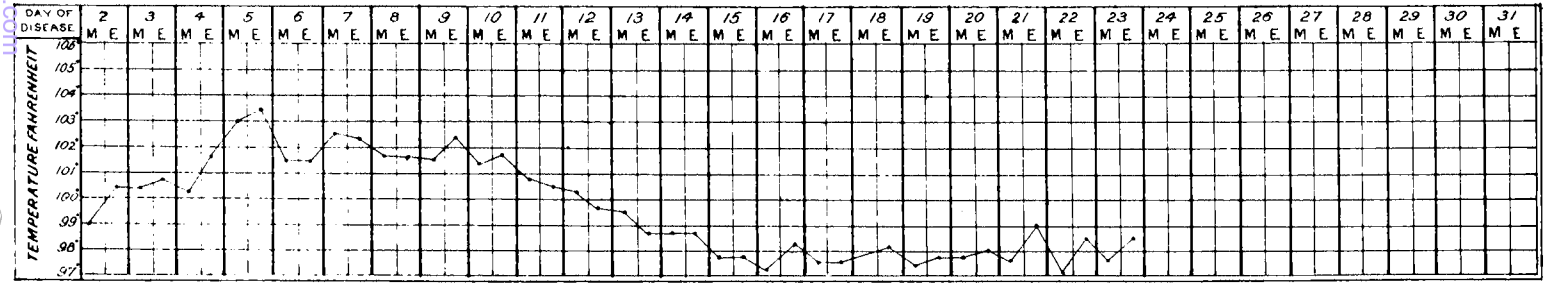
[2] *The Hœmorrhagic Infarction, German Clinical Lectures*, second series, p. 264.

[3] *Edin. Med. Jour.* vol. ix. p. 1095, June 1866.

I. Chart of Temperature in Mrs. L.'s Case.




II. Chart of Temperature in Mrs. B.'s Case.



an attack of acute pleurisy in a young unmarried lady, a patient of his. He also knew of the same sequence of events occurring in a young gentleman, a relative of his own. But in this case the pleurisy itself arose in the course of convalesence from an attack of typhoid fever. As phlegmasia alba dolens is not an uncommon sequela of typhoid and typhus fevers, of course the value of this example is somewhat less than if it had occurred independently.

It would therefore appear that there is some occult connection subsisting between pleurisy and phlegmasia alba dolens even under conditions that exclude the possibility of a common origin in some pyæmic process, or in any form of blood-poisoning.

That there was no pyæmic condition and no metritic inflammation likely to set up a pleurisy or a phlegmasia in either of these patients' cases, seems beyond dispute. At the same time, I can hardly avoid believing—in fact, I ought rather to say I am very strongly convinced — that the puerperal condition had a very special influence in determining as well as in modifying the course of the chest affections in both cases.

A single word as to the temperature. It will be observed, on inspecting the accompanying charts, that it ran its course on somewhat lower lines than ordinary pneumonia would have done.

In the first case it ran up to 103° on the evening of the third day. From the fourth to the seventh it declined. It began to rise again on the ninth, when the left lung became affected, and then showed signs of declining from the thirteenth to the fifteenth, when it rose with the occurrence of the phlegmasia. With the disappearance of the acute symptoms in the left leg, it again oscillated upon the line of 100°, till the right leg in its turn became affected, when it

rose to 102·3° on the twenty-fourth day, after which it steadily and persistently declined.

In the second case it reached its acmé on the evening of the fifth day, when it rose to 103·2°. It then continued much about the same for four days, when it declined steadily, but very gradually.

As to the treatment, I have very little to adduce. It was partly symptomatic, partly antiphlogistic.

The pain was relieved by poultices and opiates. I believe that the blisters did good in both cases. These may have acted beneficially as general stimulants to the vascular system, as well as local counter-irritants. I think we are often apt to overlook this important quality of cantharides. Those who have seen much of bronchial affections in children, and have given cantharides a trial internally in their treatment, will, I feel assured, agree with me in thinking that it is at times very useful in the treatment of pulmonary affections. Turpentine seemed of service in the second case. This it could be expected to be, both by its antiseptic and its astringent qualities, and it is more than probable that it so acted in the second case.

The advantage accruing from having the bowels freely cleared out is particularly observable in the second case, and indeed is a point that cannot be too firmly insisted upon in dealing with lying-in patients, in whose large intestine there are so frequently great accumulations of fæces at the time when delivery comes on. These collections cannot but predispose to septic changes, unless they either come away spontaneously, or are cleared out by some safe laxative medicine. But it is extremely dangerous to dogmatize in a matter of treatment,—the sources of fallacy are so very numerous.

It appears to me to be sufficiently obvious, from a consideration of the above cases, that we may now and then

expect to meet with pleuro-pneumonia in connection with the puerperal state of a very threatening and severe type, without the lesion proving necessarily fatal,—a fact which ought to be of great importance in helping us to establish a prognosis in consultation upon cases of this kind. Is there, then, any guiding principle on which one may rely in forming his opinion as to the probable result in such contingencies?

The consideration of greatest moment is doubtless to determine whether in a given case septic infection is or is not present. For if we have reason to believe that the pulmonary symptoms are due to embolic infarctions from septic embolisms, then our prognosis is necessarily of the gravest character possible. But if we have reason to suppose that the symptoms accompanying the attack are of such a nature as to warrant us in excluding septicæmia, we then may give a favourable though guarded prognosis. The absence of any pelvic or peritonitic inflammation, of any fœtor in the lochia, and of rigors, combined with the fact that the temperature never rose much over 103° (though the pulse was very high in the second case), enabled us to be tolerably certain that there was not septicæmia in either of the two cases recorded. Similar conditions ought to enable us in like circumstances to come to the same conclusion. I need scarcely say that the whole appearance of the patients was threatening and alarming in the extreme, even although the evidences of septic poisoning were absent; and, indeed, for some time the recovery of both patients appeared in the highest degree doubtful. It is of great practical importance to record cases of peculiar and severe lesions that turn out well, as well as those that terminate unfavourably, seeing that such information is calculated to be exceedingly useful for our guidance in dealing with similar conditions when they happen to arise.

Whether we entertain the view that these cases were

embolic in origin or not, we must, it appears to me, seek the chief explanation of their occurrence in the alterations which the pregnant and puerperal conditions impress upon the blood and the blood vascular system. Considered from this point of view, it would therefore appear that in such occurrences we have merely a fresh exemplification of the general principle that pregnancy profoundly modifies the condition of the whole blood vascular apparatus, as well as of the vital fluid which circulates within it. On these grounds I have deemed it not inappropriate to include the present paper in the same volume with that upon the mutual bearings of heart disease upon pregnancy, parturition, and childbed.

# ON THE ESSENTIAL PATHOLOGY OF PUERPERAL ECLAMPSIA.

IT is impossible to imagine a subject of greater interest to the obstetrician than that of puerperal eclampsia. The suddenness of its usual onset, the terribleness of its phenomena, and the extreme risks it brings with it to mother and child, all combine to make its appearance an occasion of the greatest concern and anxiety to the patient's friends, but especially to her accoucheur.

But not only is the obstetric physician bound to concern himself deeply with this lesion,—it is one of those numerous subjects that are situated in the boundary land between the territories of the pure and the obstetric physician, or, as I should rather express it, between the domain of the physician who restricts his attention to pure medicine, and that of the physician who practises obstetrics.

It therefore ought to secure the attention of both classes of physicians. For though eclampsia occurs in the pregnant and parturient female, it is even more commonly met with entirely apart from these conditions, and as a symptom of severe renal disease. It follows, therefore, that the obstetric and the pure physician meet the condition each on his own ground, and that, as both are concerned in its management, both are equally interested in everything that tends to the elucidation of its real nature. Yet it must be admitted, although with great regret, that its pathology is at the present time in a very unsatisfactory position.

In a former paper published upon this subject, and embracing the records of nine cases, I ventured to suggest that more attention ought to be paid to the condition of the brain in eclampsia than is usually given to it. The more I have since seen of the lesion, the more I have been impressed with the correctness of this idea, and I have consequently been on the outlook for opportunities to put my ideas into practical shape.

For many reasons, an opportunity to examine the brain of a puerperal patient dying of eclampsia is not an occurrence of every day, and it consequently requires time to collect the necessary material. Since then I have been able to obtain on only two occasions dissections of the brains of women who have died from puerperal convulsions. The post-mortems in both these cases were performed for me by Dr. D. J. Hamilton, Pathologist to the Royal Infirmary. He has, moreover, subjected the brain and kidneys of the former of the two cases to a scrupulously exhaustive microscopical analysis, of which he has furnished me with a valuable report, illustrated by the two drawings given below, p. 240.

The first case is a most typical one, and I hope to be able to show, as I get further on in my paper, that it is full of valuable information in respect to the condition of both the brain and the kidneys in eclampsia. The second case is not so well marked, because it was complicated with severe post-partum hæmorrhage, to which, apparently, the fatal result was mainly attributable. Still, while the evidence of the occurrence of the eclampsia is borne out by the account of the case, as given by a most intelligent and trustworthy nurse, the post-mortem appearances in the kidney were such as we might expect would have given rise to eclampsia; and the evidences of brain changes, so far as they were observable, completely accorded with the conditions we met with in an exquisitely-pronounced degree in our former case.

Some years ago, I reported a case of convulsions arising not during delivery, but within the lying-in period, and which, from the character of the attacks, I recorded as puerperal tetanus. As the brain changes found therein were very peculiar, I mean also to include the chief facts we observed in it as part of the material of this paper.

I am the more inclined to do so, inasmuch as I must, in the course of my remarks, embrace some notice of the recent able papers on the subject of so-called uræmic convulsions by Dr. Mahomed,[1] in which he endeavours to prove that small cerebral apoplexies are one cause, at least, of eclampsia. We shall thus have in all three cases to consider in this paper. With these introductory remarks, I proceed to give an account of the first two cases.

CASE I.—*Severe Puerperal Eclampsia and Albuminuria—Accouchement Forcé — Death — Post-mortem; Intense Anæmia of the collective Cerebral Centres—Engorgement of the Meninges—Apoplectic Extravasation in the Right Nucleus Lenticularis.*

C. M., æt. 18, residing at Leith Walk, Edinburgh, primipara, said to be in the eighth month of pregnancy, enjoyed good health till 20th July 1877. She is a well-nourished girl, of rather over average height. Face rather pale. On that and the following day her attendants noticed that her eyelids were somewhat swollen, and she was heard to complain of a little headache. She was, however, greatly depressed about her approaching confinement, and ashamed of her condition, she being unmarried. She seemed also, from what information could be gathered respecting her, to have been of a nervous disposition. Mr. Hanson, one of my dispensary pupils, was engaged to attend her in her confinement. On the morning

[1] 'On the Pathology of Uræmia and the so-called Uræmic Convulsions,' *British Medical Journal*, 7th and 14th July 1877, pp. 11 and 42.

of the 22d July, about 10 A.M., the patient vomited, and continued to do so severely for some time. At 12 noon, while she appeared to be laughing, she was observed to pass into a convulsion fit, and after that the convulsions continued at varying intervals. After the first fit she sat up in bed and looked wildly around, but was able to put up her own hair. When spoken to, she answered 'Yes,' but nothing more. She gradually lapsed into unconsciousness; she never complained of blindness. Mr. Hanson, on being summoned to the case, and recognising its seriousness, at once called to inform me of the matter. I was unfortunately out at the time, but Dr. John Playfair went with him instead till I should return.

Dr. Playfair saw the patient at 7.5 P.M., up to which time, according to the statement of the attendants, she had had ten fits. She was then completely unconscious. Breathing was heavy, almost stertorous. Respiration 28, and of a distinctly cerebral character,—that is, some of the respirations were long and loud, others short and almost noiseless. Skin was hot and moist. Temperature 102°. Pulse 88, weak but regular, and not intermittent. Pupils slightly contracted, equal and insensible to light.

At 7.15 the patient went into a convulsion, which lasted about two minutes. Respiration was suspended for almost a minute, and the face and neck became extremely livid. The eyes were turned to the right and downwards, and the conjunctivæ were suffused. The mouth was *open* and the lower jaw fixed, so that there was no grinding of the teeth, and the tongue was not bitten. The arms, legs, and body were in strong tonic and clonic spasm, both sides being affected alike, the convulsions possessing no one-sided character. After the fit the pulse was noticed to be 92, and the respiration irregular and stertorous. The face soon regained its wonted pale colour.

On examination per vaginam, Dr. Playfair found that the outer os was about the size of a shilling, or barely so large, and felt rather rigid. The vagina was secreting mucus copiously, and the head was ascertained to present, the membranes being unruptured.

Dr. Playfair sent Mr. Hanson immediately for me. I arrived at 8.30, but meanwhile the patient had at 7.30 a threatening of a fit, which Dr. Playfair arrested by administering chloroform ; after a few minutes another fit threatened, which was arrested in the same manner. At 7.40 a very severe fit ensued, upon which the chloroform did not seem to exert the slightest influence. The mouth was noticed to be still kept open, the lower jaw being powerfully depressed, and there being neither protrusion nor biting of the tongue. The face and lips were intensely livid.

At 7.55 another fit threatened, but was warded off by the administration of chloroform. At 8 o'clock the patient became generally convulsed, the chloroform exerting no beneficial effect. Froth, but not blood, exuded from the mouth. The labour pains were now found to recur frequently and regularly (one in five minutes). There were no further fits till after my arrival.

Considering the number and intensity of the fits, and seeing that the patient was threatening to collapse if not delivered speedily, I judged it proper to hasten delivery by accouchement forcé. On drawing off a teaspoonful of urine by catheter, it was found, on heating, to assume the consistency of newly-made cheese.

Having brought Barnes's dilators with me, I now proceeded with their assistance to dilate the os. The smallest size only could be introduced at first. As the os was very rigid, I was only able to dilate the cervix very slowly.

At 9.30 a fit of terrible severity came on. It lasted for about five minutes. The spasms were violent, but the face

did not assume the livid hue it had done on the former occasion. The jaw was still depressed. Breathing after the fit was quite stertorous. The pains were now tolerably strong, and recurring about every four minutes.

At 9.55 another severe fit occurred, lasting almost four minutes. At 10.10, the outer os being now fully the size of half-a-crown, I was able by gradual dilatation to get the cervix sufficiently dilated to admit my hand. Then seizing hold of the child's left knee, I succeeded in effecting version, but with considerable difficulty, as, after the leg was brought down, the head, which presented the vertex occiput to the left and anteriorly, did not readily recede from the pelvic brim, owing to the oblique shape which the uterus by this time had assumed. After practising Sigmund's double manipulation, however, I got the head pushed up and the breech safely brought down and lodged in the brim, and thereafter delivery was easily completed. The child was small, of the male sex, and still-born; but, after its lungs had been artificially inflated by blowing air into them through a catheter, and both Schultze's and Sylvester's methods for the maintenance of artificial respiration had been repeatedly employed, as well as other means, it came round, breathed, and cried.

The placenta was separated and expelled spontaneously. There was no post-partum hæmorrhage.

A slight fit occurred immediately after delivery. The pulse was now 92; respiration 22, still irregular, but not so stertorous. Dr. Playfair and I shortly afterwards left the patient, Mr. Hanson remaining by her for some time.

At 11.10 the patient had a convulsion, which lasted ten minutes. Pulse during the time was weak.

At 11.30, another that lasted five minutes.

At 11.45 twenty grains of chloral were administered. This

had been ordered to be repeated every four hours. The temperature was noted to be 103·5°, pulse 120.

From 11.45 to 12.45, three slight convulsions. The patient, having fallen asleep, was then left by Mr. Hanson. Temperature, 104°; pulse, 120.

At 4.30 A.M. Mr. Hanson again visited the patient. The attendants stated that the convulsions had recurred every five minutes for the last two hours,—that three of them had been very severe, and affected one side only.

During Mr. Hanson's stay at this time the patient had one very violent convulsion, which lasted two minutes, but both sides of the body were affected. Temperature now 104°; pulse, 100, but very feeble; skin soaked with perspiration; patient cannot swallow.

At 10 A.M., temperature, 104°; respiration, 52; pulse almost imperceptible. The patient has had no fit since 5 A.M.

One and a half ounces of dark smoky-coloured urine were drawn off, which on examination was found, like the first specimen, to become semi-solid on heating, and to contain numerous blood casts. The patient can now swallow. Brandy ordered to be freely administered.

At 12 noon temperature was 104°; pulse imperceptible. Patient continues quite unconscious. At 8.30 P.M., temperature, 103·5°; and at 10.30 P.M. the patient died.

A post-mortem examination was allowed, and was performed by Dr. Hamilton twenty-two hours after death. The following is the result of it:—

Body well nourished. Post-mortem rigidity not well marked. Lips livid. Some frothy mucus tinged with blood exudes from the mouth. Pupils semi-dilated. Mammæ well developed, the areolæ being well marked. The uterus extends to about midway between the symphysis pubis and the umbilicus. Slight œdema of the lower part of the chest. None of any other part of the body.

Head.—Dura mater much congested. Longitudinal sinuses engorged with blood. Vessels of surface of pia mater extremely congested, both large and small vessels being enormously distended with extremely dark blood. No adhesion among the membranes. No effusion on the surface of the pia mater, everything being normal except the excessive congestion. Cerebral convolutions ill developed, but not specially flattened. Membranes at the base peculiarly healthy. The vessels in this situation are not much congested. The medulla and pons seem unnaturally pale on outward inspection. The large vessels appear perfectly healthy.

The corpus callosum is extremely pale and anæmic, so likewise is the fornix.

The cerebral convolutions, when cut into, present no capillary injection, no extravasation. The large vessels contain blood, and are apparently perfectly healthy and normal.

The left corpus striatum appears peculiarly anæmic.

At the anterior portion of the right corpus striatum, where it dips down to form the nucleus lenticularis, there is a dusky red spot, about the size of a pea or a small bean, in which are seen a number of apparently punctiform hæmorrhages. Otherwise the right corpus striatum is as the left.

The choroid plexuses are anæmic. The veins over the right optic thalamus are deeply congested. Both optic thalami are markedly anæmic. The pons varolii is extraordinarily anæmic; so also is the medulla oblongata. The cerebellum is apparently healthy.

The crura cerebri are also anæmic. There is no fluid in any of the ventricles. The fourth ventricle seems larger than usual, and its floor very anæmic.

The vessels of the meninges of the cord are somewhat congested. The cord itself is extremely anæmic.

Chest.—Lungs are congested, œdematous, and puffy. The

heart is about normal in size, but bulges decidedly to the left. Both ventricles are firmly contracted, especially the left. The valves and endocardium are healthy. The heart weighs 9½ ounces. The left ventricle is hypertrophied; the thickest part of its wall measures ¾ inch. The right ventricle is very slightly hypertrophied.

Abdomen.—Omentum covers the upper two-thirds of the abdominal cavity. The uterus lies exposed in front; the small intestines are arranged around its upper border, but not covering it to any great extent.

The liver is fatty in certain places, congested, and contains an excessive quantity of blood. Spleen normal. Both kidneys are large and flabby. The capsule strips easily off. Cortex pale. Medulla deeply congested. No cysts. No adhesions.

The brain and kidneys were taken away for further examination under the microscope. The following is Dr. Hamilton's report of what he found regarding them. I take this opportunity to state that no words of mine can adequately express my sense of obligation and gratitude to him for the trouble and pains he has taken with the case.

1. CONDITION OF THE NERVOUS CENTRES.—The nervous centres were carefully hardened, and afterwards thoroughly and systematically examined. The hardening was conducted chiefly by placing the parts in a mixture of Müller's fluid and spirit, with the subsequent use of a weak solution of ammonium bichromate. The examination was commenced in the cerebral convolutions, and different means were adopted for the purpose of bringing out the structural elements.

It was found that the 'Dammar-lac method,' whereby the preparation was rendered half transparent, was the most serviceable. Nothing of any great note was observed in

the cerebral convolutions, further than the most marked anæmia.

The whole of the smaller-sized arteries and veins, and the capillaries without exception, were totally empty, unless where here and there they contained a stray leucocyte. The lumina of the small arteries seemed *small*, as if the vessels had been in a state of contraction. But otherwise the whole of the cerebral convolutions seemed normal, the nerve elements and neuroglia corpuscles having evidently undergone no change. A similar anæmic condition, but even, if possible, more marked, was seen in the cerebellum. But its structure otherwise was also quite normal. The small spot of a reddish colour seen in the nucleus lenticularis was specially set apart, and examined with the greatest care.

A representation of it is given in Fig. 1, where it will be seen that a small hæmorrhage had been the cause of the discoloration. It was not sharply circumscribed, the colouring matter having left the effused blood corpuscles and stained the neighbouring brain substance. The vessels at this particular spot were peculiarly abundant, and it will be noticed that they are almost totally devoid of blood corpuscles, and in some cases irregularly contracted. The brain substance in the vicinity of this punctiform apoplexy seemed to be considerably disorganized, but no changes of an inflammatory nature were noticed. A slight amount of blood was seen in the adventitious space of one small artery, and in others there were what appeared to be a few wandered leucocytes in the same situation. On examining the upper level of the medulla oblongata, nothing further than the above-named anæmia was revealed. But a little further down, towards the level of the middle parts of the corpora olivaria, the vessels seemed peculiarly prominent and abundant. In the corpora olivaria they formed a most beautiful plexus, but were all totally devoid of any coloured blood

FIG 1.
Hemorrhagic spot in right Corpus Striatum.

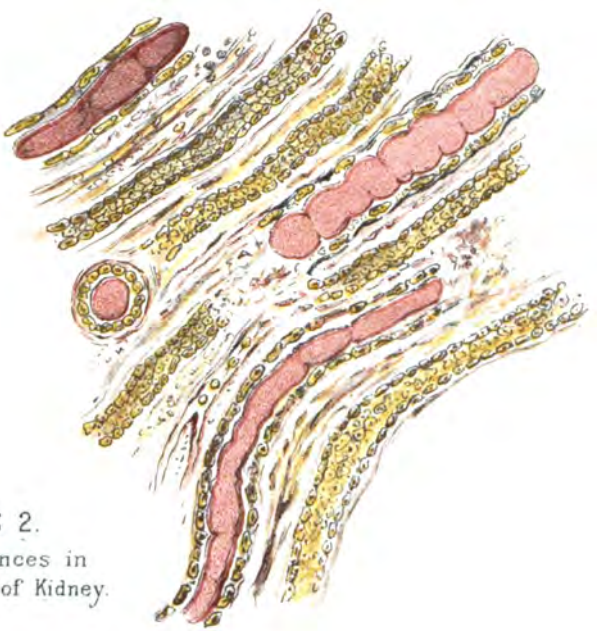


FIG 2.
Appearances in Medulla of Kidney.

corpuscles, although some of them contained a leucocyte here and there. At the same level, in the region between the two corpora olivaria, a further change was visible. This consisted in a deposit of large numbers of leucocytes in a somewhat peculiar manner. They were most abundant immediately around the capillary vessels, and were arranged in little groups of five or six, while, at some distance from these vessels, they ran in lines, and also formed little rounded groups. The vessels here also seemed to contain in some places a considerable number of the same bodies, and they were seen protruding from the outer aspect of the capillary wall, and then becoming detached, apparently passing into the tissues.

The appearance was chiefly noticed in this region,—between the corpora olivaria,—but not to any great extent in the corpora olivaria themselves. In the cervical region of the cord the vessels contained a few coloured corpuscles, and in some places exudation of leucocytes had apparently occurred to a slight extent.

2. CONDITION OF THE KIDNEYS.—The kidneys were likewise carefully examined, and the appearances which were found seemed to me to be quite different from anything else I have ever seen in acute renal disease. Judging from the naked-eye appearances, we expected that parenchymatous inflammation of the tubular epithelium in the cortex passing into a state of fatty degeneration would be revealed. The first glance at a section of the organ, however, showed conclusively that this was not the case, and that the lesion was not an ordinary parenchymatous inflammation. On the contrary, it seemed as if the epithelial cells lining the convoluted tubules, in place of being swollen and granular, were peculiarly small, and the nuclei abnormally distinct. To convey as clear an impression as possible of the appearances, we shall commence the description with the capsule of the organ, and proceed inwards.

The capsule seemed quite normal, and its fibrous attachments neither increased in number nor in thickness. The convoluted tubules immediately underlying this were very wide, their lumina about twice as large as in the normal organ, and the epithelium lining them very distinct. The individual epithelial cells were much more defined than they usually are; their nuclei abnormally prominent. In a great many of the tubules these were the only changes present; but in many of the others a further alteration was observed. This consisted in the development within the epithelial periplast of a minute drop of a clear homogeneous substance, which, after increasing in size, burst through the cell substance into the uriniferous tubule. Similar drops were discharged into the tubule from neighbouring cells, and finally these ran together and formed one large globule. In tracing this globule downwards into the medulla, it was found to increase in size to an enormous extent, apparently by coalescing with similar globules, and eventually to form a long hyaline cast, distending the tubule to twice or thrice its original calibre. The straight tubes in the medulla were in great part choked up with this substance, in some places universally so, whilst in others only every second or third tubule was so occluded. The appearance in the straight tubules is represented in Fig. 2, where the homogeneous appearance of the tube-casts is evident. The substance was stained deeply and readily by even weak solutions of carmine. In no instance was the formation of this material in the manner above described observed in the epithelium of the straight tubules. It appeared to arise entirely from the convoluted tubules in the cortex, and to be carried downwards mechanically into the medulla. It is not within my recollection that I have ever seen anything like this in an acute case without the presence of old-standing interstitial nephritis. The foreign substance in the tubules was exactly

like what is usually designated 'colloid,' as met with in the tubules in chronic interstitial nephritis, in the thyroid gland, in adenomatous ovarian tumours, and in colloid degeneration of cancerous tumours. It is apparently always the product of a true degeneration of epithelial cells, and is extremely apt to accumulate in shut cavities lined by epithelium, such as the thyroid vesicles, obliterated uriniferous tubes, and ovarian cysts.

It is apparently similar to what is sometimes called a fibrinous tube-cast. But how it ever came to be so designated seems strange, as in its appearance, mode of formation, and behaviour with re-agents, it is totally different. There seems to me very little doubt that it is the same material which occurs in ordinary colloid degeneration, and it would appear to arise from a peculiar transformation of the epithelial periplast.

The blood-vessels were little if at all altered, and there was no inflammatory exudation between the tubules. The sole change was apparently the degeneration of the tubular epithelium above described.

CASE II.—*Delivery Natural — Placenta retained for two hours; meanwhile four severe Eclampsic Seizures occurred —Post-partum Hæmorrhage—Death—Post-mortem Examination: General Anæmia, but especially of the Cerebral Centres—Congestion of the Cerebral Meninges—Kidneys in the Second Stage of Interstitial Nephritis.*

Mrs. M., æt. 37, residing at Plewlands, near Edinburgh, was confined of her third child on the 18th of January 1878.

The labour pains began about 10 A.M., and were pretty steady and severe from the first, recurring at intervals of not more than five minutes.

There was present throughout the delivery a great deal of

hiccup and yawning, from which, moreover, the patient had been observed to suffer greatly for three days previously. Her look also was peculiar, and her eyes very staring. She maintained a very reticent demeanour, and indeed never spoke, unless interrogated, from the time she was put to bed till the birth of the child, which took place at 2.35 P.M. At this time her extremities felt cold, and, on being asked if she felt pain, she replied in the negative, but at the same time stated that she had a strange feeling over her heart, and that there were peculiar noises in her ears. Subsequently to this, when spoken to, she took no notice whatever of what was said to her. The after-birth did not come away spontaneously, and there was no medical man to be got for some time to remove it. The patient was covered up from the cold, and some spirits and warm water were administered to her.

Meanwhile there was little if any hæmorrhage perceived. At 2.45, there being still no bleeding, the patient was seized with a severe general convulsive fit. The tongue was protruded and bitten during it. The head was also turned strongly to the left. Three similar fits followed this one before four o'clock. During the interval between the attacks there was stertorous breathing, and also some yawning and hiccuping.

About four the patient became quiet; but there was now pretty free hæmorrhage, especially when the cord was pulled upon, or the belly squeezed.

Dr. Ronaldson and Mr. M'Callum arrived about 4.40, when the former removed the placenta from the uterus.

The patient died at 5 P.M.

A post-mortem examination was made by Dr. Hamilton forty-seven hours after death. The conditions observed were as follows:—Lips extremely anæmic, pupils dilated and equally so. Post-mortem lividity extremely ill-marked,

rigidity slight. Lineæ albicantes seen on abdomen. Abdominal wall flaccid. Slight œdema of the lower limbs and also of the hands. On cutting into the abdominal wall, almost no blood visible. Large amount of fat anteriorly. Lower border of the omentum on a level with a transverse line passing an inch below the umbilicus. The uterus occupies the middle of the upper part of the pelvis, the fundus being exactly midway between the upper edge of the symphysis and the umbilicus, in this case three inches from either point. Small intestine pushed upwards. Left ovary situated above the brim along with its corresponding Fallopian tube. The right ovary was in the hollow of the pelvis. The fold of Douglas was quite empty. The right ovarian vein throughout its lower half was enormously distended with air or gas, being about the thickness of the thumb, and containing very little blood. Left vein also somewhat large, but contained no air.

Chest.—Lungs extremely pale; do not readily collapse. The right lung the more prominent; both lungs non-adherent. The left œdematous, and slightly congested towards the centre. The right anæmic, at upper lobe somewhat congested, and extremely œdematous at the lower part.

Heart.—The right side contains scarcely any blood, but when pressed produces a gurgling noise as if from the presence of gas. Left side firmer. On that side no gurgling is elicited. The blood is extremely fluid.

Two milk spots are visible on the surface of the heart, one anteriorly, and one to the right side.

The organ as a whole seems large. Aortic valve is competent. Mitral valve is thickened at the edges, but admits two fingers. Left ventricle at its thickest part measures $\frac{1}{4}$–$\frac{1}{2}$ inch.

The tricuspid valve almost admits five fingers.

The whole tissue of the heart is anæmic and pale, but apparently not fatty.

Liver pale, and its substance anæmic. Spleen pulpy, and also anæmic.

Left Kidney.—Anæmic and pale. The capsule strips off easily in certain parts, in others it is slightly adherent, and leaves a pale granular surface, with here and there congested venous radicles. A number of isolated roundish pale nodules, of the size of pin-heads, having when cut into a somewhat elongated shape, are seen on the surface of the cortex.

The cortex is diminished in thickness, being only about 1-5th of the thickness of the kidney. It is extremely pale, with congested vessels running through it. Medulla also pale, with congested vasa recta, and extremely flabby. The right kidney is in an exactly similar condition.

Head.—Dura mater apparently healthy. The vessels of the pia mater *congested.* Considerable amount of subarachnoid fluid on the surface of the brain. No fluid at the base of the brain. Substance of the pons and medulla pale, but the vessels over them congested.

The corpus callosum, substance of the right and left hemispheres, as also of the fornix, corpora striata, and optic thalami, extremely pale and anæmic, showing scarcely any puncta cruenta. The pons and medulla, when cut into, were also seen to be anæmic, with here and there a punctum cruentum.

My observations in the sequel of this paper, in reference to the cases above described, must be regarded as almost exclusively based upon the facts observed in the former of the two. That one was a typical example of the lesion whose pathology is under consideration; and, besides, the brain and kidneys have been subjected to an exhaustive examination. There has not as yet been time to examine the brain of the second patient in the same manner. The second case can only be looked upon as, on the whole, so far

as the brain changes were developed, corroborative of the facts observed so pronouncedly in the first, and nothing more. Thus it will be seen that, although there was scarcely a drop of red blood in the rest of the body, the cerebral meninges were congested, whilst at the same time the central parts of the brain showed an anæmia nearly as well marked as in our first case.

The doubts at first suggested to our minds as to whether this was a case of true puerperal convulsions, and not rather one of acute anæmia with general convulsions, were satisfactorily settled by the intelligent statement of the nurse, and by the detection in the kidney of chronic interstitial nephritis. The latter condition, which was previously unknown to exist, is of itself quite sufficient to account for the fits. I therefore believe that, in this patient's case, we have reasonable grounds for believing that we had genuine puerperal convulsions, followed by post-partum hæmorrhage. This complication, according to many authors, *e.g.* Tyler Smith and Spiegelberg, is exceedingly apt to arise, though I am not able to say that my own practice would lead me to that conclusion. The gas which was found to distend the ovarian veins, and partially also the right heart, appears in this case to have been most probably the result of post-mortem decomposition.

## RESUMÉ OF THE PATHOLOGICAL CONDITIONS FOUND IN CASE I.

But, before I commence to grapple with the main problem in hand, let me first recapitulate the chief peculiarities in this extremely interesting case. And perhaps it may not be considered altogether unimportant to state here that the condition of the brain was quite different from what either Mr. Hamilton or myself expected to find.

I was specially on the hunt for sand-grain apoplexies in the cerebral tissue, in consequence of having had my mind previously directed in that line by my case of so-called tetanus, recorded below as Case III., and by reading Dr. Mahomed's papers on uræmic convulsions. It will be noticed that it was a little time before we could thoroughly realize the condition of absolute anæmia presented by the cerebral centres. This is made evident, on reference to Mr. Hamilton's report, by the progressive emphasis with which he dictated the statements regarding the appearances. These I took down at the time verbatim et literatim, and purposely retained them in their original form in order to bring out this fact. The circumstance that the appearances came upon us unexpectedly, indeed as a surprise, appears to me to afford additional and important evidence, if such were needed, of the truthfulness of the description we have given of the facts observed. Now these may be briefly stated to have been—

In the Brain and Cord,—

1st. Intense congestion in the meninges, and engorgement of the venous sinuses on the inner aspect of the cranium and spinal canal.

2d. Intense anæmia of the deeper portions of the brain, and especially of the collective motor centres, including the cord.

3d. Complete absence of any evidence of this anæmia having been preceded by cerebral œdema, as the Traube-Rosenstein theory of puerperal convulsions predicates.

4th. In the portion of the medulla oblongata between and down to the level of the middle portions of the olivary bodies, and also in the olivary bodies themselves, there were found vascular and extravascular changes indicative of commencing inflammatory change.

5th. There was observed a limited extravasation of blood in the anterior portion of the right corpus striatum where it dips down to form the nucleus lenticularis.

6th. It is at the same time to be noticed that all those changes occurred, although the cerebral blood-vessels, and indeed the entire vascular system, were found to be peculiarly healthy.

In the Kidneys,—

7th. The naked-eye appearances of ordinary parenchymatous nephritis were found; but, on microscopic examination, an entirely different condition was detected. This consisted of degeneration of the epithelial cells of a certain proportion of the tubules in the circumferential aspects of the cortex. The degeneration appeared to be of a colloid nature. The products of these degenerated cells ran down and blocked up, more or less completely, the other convoluted and straight tubules, so as to render them functionally useless, although their tissues were not diseased.

8th. Throughout the body, the general appearances coincided with those ordinarily found when death results from puerperal eclampsia. Thus the liver and lungs and other internal viscera were seen to be markedly congested, etc.

## CONSIDERATION OF THE PROBABLE RELATION BETWEEN THE ECLAMPSIA AND THE CEREBRAL APPEARANCES OBSERVED.

Such being the peculiar appearances found in this post-mortem, what deductions do they warrant us to draw in respect to the important problem enunciated as the theme of this paper, it being granted that the second case, so far as the changes were developed, may be held as corroborating the cerebral phenomena detected in the first case?

In the first place, it is quite conceivable that some might argue that the brain changes were caused by the fits, and were not a result of them; that, in fact, a fit of great severity

had so profoundly modified the cerebral circulation, that it was never restored to the normal condition.

But I can hardly imagine this seriously maintained by any one; for, in the first place, the changes were too rigidly limited and too emphatically pronounced to be the result merely of the temporary interference with brain circulation that a fit could produce. Then, again, had the patient died in a convulsion, a certain amount of value would doubtless have been assignable to such a suggestion. But the patient had had no fit for seventeen hours before death, so that it is quite impossible to conceive that the cerebral disturbance originating in the fits should have remained so long, if the mechanical conditions connected with the convulsions had been the sole factors determining the changes in the brain circulation. But it is perfectly natural to suppose that, if those extraordinary conditions in the cerebral circulation had been once established, they would have persisted till death, though the motor nervous system had become so exhausted that it could no longer respond to the stimulus they exerted upon it.

It might also be maintained that the alterations found were merely a result of the special mode of death,—a terminal symptom of the final struggle for existence.

As the patient died quietly, and apparently from asthenia, it appears to me that this suggestion is really not worthy of being seriously discussed. It is therefore, I think, almost beyond question that the cerebral appearances were established some time before death, and that they were quite different from anything that we could expect as resulting from a convulsion.

If, therefore, the cerebral appearances were not caused by the convulsions, it seems to me only rational to assume that they were at least one important, possibly the most important, set of factors in the production of the convulsions,—

that, indeed, they were the proximate cause of the fits. It becomes, accordingly, extremely interesting to know whether we are able to offer any reasonable explanation of the origin of those cerebral changes.

The most striking alterations, of course, and those, I think, which we are entitled to regard as the most essential to the eclampsia, are—(1st) The extreme anæmia of the collective cerebro-spinal centres; and (2d) The coincident equally extreme meningeal engorgement.

These two conditions seem to me, however, to be complementary, the latter being the direct result of the former. My explanation of this mutual relation is as follows:—The sudden *anæmia* of the deeper portions of the brain would necessarily lead to loss of bulk or shrinking in those parts of the organ. But, in consequence of the special conditions under which the cerebral circulation is maintained, the cranial cavity must necessarily be always full. This would necessitate the retention, towards the surface of the brain, of venous blood to occupy a space corresponding with the loss of volume resulting from the deep anæmia. In this way, it appears to me that the main congestive changes in the meninges can be accounted for. The arterial engorgement on the surface of the pia mater will not admit, however, of being so explained. That condition is indicative of the presence of a state of the arteries on the surface apparently the opposite of that assumed to be present in the centre of the brain.

The chief burden of this contribution seems to me, therefore, to be to explain, if possible, in what manner this extraordinary anæmia of the cerebral centres arose. For, after the exhaustive researches of Kussmaul and Tenner[1] upon this subject, it may be taken for granted that sudden anæmia of the cerebral centres, such as was detected here, is at least one cause sufficient to induce general convulsions.

[1] Kussmaul and Tenner *On Convulsions*. New Sydenham Society, 1859.

It is well known that many other causes may produce general convulsions; but if it should be found—and these two cases point in this direction—that case after case of puerperal eclampsia presented anæmia of the cerebral centres as a constant accompaniment, and if, at the same time, on other grounds, such a condition is allowed to be capable of inducing general convulsions such as we see in eclampsia, it does appear to me that we are most probably on the road towards a solution of the essential pathology of this terrible disease.

Assuming, therefore, that this extreme anæmia of the cerebral motor centres was the proximate cause of the eclampsia in these cases, how did it arise? What were the conditions that led to its development?

## THE TRAUBE-ROSENSTEIN THEORY FAILS TO EXPLAIN THE CEREBRAL APPEARANCES.

We turn almost instinctively to the beautiful theory of Traube, as applied to puerperal convulsions by Rosenstein,[1] and which is ordinarily known as the Traube-Rosenstein theory.

According to this view, eclampsic or so-called uræmic convulsions occurring in the pregnant or puerperal condition are not occasioned by the presence of any poison in the blood, but result from cerebral anæmia, which, again, is a consequence of cerebral œdema. The explanation of how this condition is brought about is as follows:—The blood of pregnant women is normally increased in quantity, but of defective quality,—being, in fact, too watery. It is, moreover, propelled under increased tension, inasmuch as the left ventricle of the heart hypertrophies during pregnancy, especially during its latter months. During the labour,

[1] Rosenstein, *Die Pathologie und Therapie der Nierenkrankheiten*, 1870.

particularly during the down-bearing pains, the already abnormally exalted tension is very greatly increased. If, now, to those physiological conditions kidney lesion is superadded, with its deteriorating effects upon the blood and its special influence in the development of cardiac hypertrophy, we have produced a state of matters that presents us with a large amount of blood of defective quality circulating in vessels subjected to a very high tension.

The result of this, according to the Traube-Rosenstein theory, is that we have first produced cerebral hyperæmia. This leads to effusion of serum from the watery blood into the cerebral tissues. So soon as the œdema has thus been produced, it reacts, from the incompressibility of the fluid composing it, so as to prevent the dilatation of the cerebral vessels traversing the œdematous areas, and anæmia is the result. If this occurs in the cerebrum, according to our authors, we have coma; but if it arises in the motor centres, convulsions are the result.

Before we could accept this explanation, we clearly ought to be able to perceive some traces at least of œdema within the brain. But when we examine for traces of cerebral œdema in our first and most typical case, it is nowhere to be found. It will be noticed, on reference to the report of the sectio, that the cerebral ventricles were found to be quite empty of fluid, which could not have been the case had there been œdema of the cerebral centres. Besides, there was no evidence otherwise of cerebral œdema. In the second case, it is observed that there was an excess of subarachnoid fluid on the cerebral surface. But this appears to have been rather of the nature of a complementary arrangement for the contraction in bulk arising out of the sudden anæmia in the central parts of the brain. Also, there was no evidence of antecedent general dropsy in either case. In the first case the patient was strong and healthy, and never complained

till two days or so before she was struck down with this illness. It is true her heart had the normal hypertrophy which we know accompanies pregnancy, and, moreover, her kidneys were suddenly rendered functionally almost entirely useless. There was thus abundant reason to assume specially high vascular tension when the labour came on. But the eclampsic attacks appeared before the pains of labour were fully established; and, altogether, the cerebral changes were not such as we could expect to arise from excessive vascular tension.

From the latter cause one would have looked for general œdema of cerebral tissues or general congestion. But how intravascular pressure alone could bring about extreme anæmia of one part of the brain, without œdema of the cerebral tissues of that portion and intense congestion of other portions, seems to me inexplicable on any mechanical principles which I can conceive. And if we assume that the anæmia arose according to mechanical laws, which the Traube-Rosenstein theory assumes, we are bound to give at least a probable rationale of the physical conditions under which it was produced.

On these grounds I am reluctantly compelled to confess that it seems impossible to me to apply the Traube-Rosenstein theory, beautifully fascinating though it be, to the explanation of the main specialty in these cases.

Bartels[1] has expressed similar difficulty in reference to the cerebral appearances in non-puerperal patients dying under his care from so-called uræmic convulsions. He, however, with much truth, directs attention to the difficulty experienced by the physician in determining by ordinary inspection the presence or absence of œdema in the cerebral tissues, and at the same time states that by the most

[1] *Handbuch der Krankheiten des Harnapparates*, Erste Hälfte, S. 120, Ziemssen, Bd. ix. a.

careful inspection by the naked eye he failed to discover such a degree of wateriness as would agree with Traube's theory.

The record given by him of one interesting patient, who died from the second of two epileptiform seizures, is extremely important from the point of view from which we are now regarding this problem. This refers to a young woman, aged twenty-two, who suffered from chronic interstitial nephritis, with marked hypertrophy of the left side of the heart. Shortly before admission she had had a severe hæmoptysis, and was reduced to a condition of extreme anasarca and anæmia. This deplorable state was further made worse, during her short stay in hospital previously to her death, by her inability to take anything except fluids, by much vomiting, and finally by a blood-letting of about four ounces when she was in the first fit. After recovering from this seizure, she took a good draught of water, and five hours afterwards a second fit came on, for which she was again being bled, when she died.

If there ever had been conditions likely to establish anæmia of the cerebral centres, as a result of primary œdema, such a case was that one. But I feel persuaded I give a fair idea of the post-mortem appearances as given by Bartels in this case, when I state that the cerebral tissues presented appearances not at all unlike those detected by us in our cases, only that the peripheral congestion was less marked than in our first case, apparently because there was extremely little red blood left in the poor woman's body at the time of her death.

## ARGUMENTS IN FAVOUR OF THE THEORY THAT THE ANÆMIA WAS DUE TO IRRITATION OF THE VASO-MOTOR CENTRE IN THE MEDULLA OBLONGATA.

I am therefore driven to the conclusion that the cerebral anæmia in those cases of mine was not the result of over-

distension of the smaller arteries and capillaries, with consecutive œdema and anæmia of the cerebral motor centres. But this brings me face to face with the question, How then did the anæmia arise? If the disturbance in the equilibrium of the brain circulation was not so brought about, what condition or conditions did produce it? Looking at the state of the smaller cerebral vessels, as recorded in Dr. Hamilton's report of the first case, I am led to believe that the anæmia in the centres of the brain was brought about in an active manner, and that it must have followed from spasm of the arteries in the anæmic areas, however this condition may have been determined.

But, while rejecting the Traube-Rosenstein theory as not applicable to the explanation of the phenomena, I am, I confess, greatly at a loss how to account for them. It appears necessary for us to impress into our service a great many facts brought out in the examination, before we are in a position to give a probable, not to say adequate, explanation of the very peculiar conditions observed.

I shall now proceed to point out those facts and considerations which I regard as at once important and likely to aid us in forming an intelligent conception of the probable pathology of this peculiar condition of the brain.

In the first place, I would draw attention to the fact, which is abundantly proved from many phenomena connected with pregnancy, and especially by the effects that pregnancy produces upon cardiac disease, when the latter is found to co-exist with it, viz., that pregnancy seriously modifies the condition of the vascular system generally, alters its tension during the latter months, and makes it in some way very specially liable to derangement and disease. Abundant evidence in support of this statement is furnished in the chapters on the *Bearings of Chronic Disease of the Heart upon Pregnancy, Parturition, and Childbed*, forming the first part of this volume.

That the same is equally true of disturbances in the domain of the nervous system, is evidenced by the great frequency with which nervous affections long latent are called into activity during a pregnancy, and by the frequency with which mania and other nervous diseases follow in the wake of a confinement.

That such is the case has been lately pointed out with characteristic ability by Dr. Barnes,[1] so that further details on my part are unnecessary.

But the most peculiar, and, I cannot help thinking, the most active component of all the factors that co-operated in the production of the intense cerebral anæmia, seems to me to have been the sub-inflammatory changes described by Dr. Hamilton as affecting the part of the medulla oblongata about the level of the olivary bodies, and intermediate between these structures. These structural changes had advanced not only to capillary distension, but actually also to the escape of leucocytes into the extra-vascular tissues, so as to evince unmistakeably the existence of commencing inflammatory action. In the olivary bodies themselves, it will be noticed that the only change observable was special prominence of the vascular plexuses.

In reference to the bearing of this condition of the medulla oblongata upon the subject in hand, let me here refer to the well-known observations of Owsjannikow,[2] to the effect that in the rabbit the vaso-motor centre of the whole body is restricted to a space in the medulla oblongata, limited inferiorly by a point 4 millimetres above the extreme point of the calamus scriptorius, and superiorly by a point 1–2 millimetres behind the posterior border of the corpora quadrigemina, the centre itself being about 4 millimetres in

[1] 'Pregnancy and General Pathology,' *Transactions of American Gynæcological Society*, vol. i. p. 144.

[2] Ludwig's *Arbeiten*, 1871, p. 21.

extent. According to the same author, this centre is bilateral, being symmetrically disposed on either side of the mesial line, whilst its position, measuring from before backwards, is nearer the anterior surface of the medulla than the floor of the fourth ventricle.

If, now, we take the measurements as given for the rabbit, and apply them merely relatively to the human medulla, it appears that if we divide the line running from the posterior edge of the corpora quadrigemina to the apex of the calamus scriptorius into ten equal parts, the vaso-motor centre would be situated in the second and third fifths of this distance, measuring from above downwards. That measurement would take us pretty nearly to the middle third of the distance, but with a slight inclination upwards more than downwards.

As this localization of the vaso-motor centre given by Owsjannikow would bring it very much into the space that was found altered, it appears only fair to assume that this centre was most probably involved in the sub-inflammatory changes observed by Dr. Hamilton.

If anything, the position of the sub-inflammatory action was situated rather too low down. But it must be remembered that even if that were so, and if the irritative process had not involved the whole of the centre, yet there is physiological evidence to show that the collective vaso-motor fibres must have passed through the affected space, from the proximity in the downward direction of these changes to the centre. It is a known fact that the efferent fibres from this centre pass all downwards at first.

I have further to draw attention to a fact, which appears to me to indicate that this sub-inflammatory change in the medulla acted as an irritant to the nerve centres. It will be noticed that our first case of eclampsia has an important peculiarity in its symptomatology, namely, that during the

attacks the lower jaw was powerfully depressed. It is almost always found in eclampsic cases that the tongue is not only protruded but badly bitten, in consequence of the lower jaw being powerfully shut. But it is noted again and again in this patient's case, that whereas the muscles generally, including those of the head and face, were thrown into a condition of tonic and clonic spasms, the lower jaw was observed to be powerfully depressed.

I read this as indicating that the origin of the hypoglossal nerve, which supplies the depressors of the lower jaw and the larynx, had been within the range of the sub-inflammatory irritation. This nerve, as also the vagus and the glosso-pharyngeal, takes origin about the level of the middle third of the olivary bodies, but in the floor of the fourth ventricle. Further, the respiratory action was noticed from the first to be affected specially, indicating participation of the vagus in the irritative changes.

The masseters, on the other hand, do not seem to have acted so powerfully as usual, because it would appear that the main irritation in the medulla had been operating in a situation somewhat below the origin of the motor roots of the fifth pair of nerves that supply these muscles with motor fibres.

Then the extraordinary manner in which the tubules of the kidneys were choked up by the rapid accumulation of the colloid matter, resulting from the degeneration of certain of the epithelial cells, at once arrested their functions as blood depurators and as excretors of water. In consequence, the blood would not only tend to increase abnormally in amount, but to rapidly depreciate in quality, so that it would not only be injurious by its bulk, but would also act as an irritant, through the instrumentality of the excrementitious materials retained in it.

Dr. George Johnson[1] has laboured to show that under

[1] *Lectures on Bright's Disease*, p. 72, London, 1873.

such conditions the smaller arteries contract, and refuse, so far as in them lies, to allow such an objectionable material to pass into the tissues. He refers this result to the regulating action of the vaso-motor nerves, but he does not explain how it arises.

If I am correct in my reasoning, it would seem that the nerve cells of the vaso-motor centre were most probably directly irritated by the impure blood circulating through the centre, and that they in consequence induced by excess of action an exalted degree of inhibition on the collective arterial system. As a result of this, the blood would tend to leave the arterial system, and to accumulate in the veins. There would be pallor and rather a slow pulse, which, however, would feel weak, as the radial would necessarily be small. This state of matters is not contra-indicated by the general appearance of the patient in the first case, for it is noted that between the attacks she was pale, and that the pulse was small. The rate, moreover, considering the temperature and the great exhaustion, was, on the whole, slow; for it is noted, after the fits had been well established, as 88, though the temperature was then as high as 102°, and even after delivery it was only 92. It ultimately rose to 120, apparently in consequence of the exhaustion following the labour and the fits.

I have assumed that the blood, charged with the collective excrementitious matters which it is the duty of the kidney to remove, acted as a direct stimulant and irritant to the vaso-motor centre, and thus led to general arterial contraction acting centrifugally. It is, however, quite possible to conceive that the poisoned blood should have acted throughout the tissues upon the afferent fibres of the vaso-motor system, and thereby have originated a stimulus which was conducted from the tissues generally to the vaso-motor centre, whence it was reflexly communicated to the arteries

throughout the body. In that case the sub-inflammatory changes discovered in and about the vaso-motor centre would have resulted from prolonged and severe reflex stimulation, and not from the local action of an impure blood upon the centre itself.

It is also possible that the results observed may have been partly occasioned by the one of these conditions and partly by the other, the impure blood acting as a local stimulant to the centre itself, and also as a reflex stimulant to the afferent nerves leading to the centre from the various regions of the body.

That there did exist over-stimulation of the vaso-motor centre appears to me extremely probable, when we consider the results as to the localization of that centre obtained by Owsjannikow, and at the same time view, in the light which this knowledge gives us, the conditions above described in the part of the medulla intermediate between the upper portions of the olivary bodies.

The essential element, therefore, in the pathology of this case would seem to me to be reduced to a condition of over-action of the vaso-motor centre, from the stimulating effect upon it of a blood rendered impure through retention of the collective excrementitious matters which the kidneys ought to have removed. By this over-stimulation there would be produced a high degree of anæmia of the deeper cerebral regions and central portions of the cord, and coincidently a collection of the blood in the venous sinuses within the cranium and the spinal cavity, and in the meninges. This result of defective blood-supply would be at the same time aided by the peculiar conditions of the venous and blood vascular systems conditioned by the pregnancy.

Why the anæmia produced by arterial contraction should be so marked and persistent in the deeper portions of the cerebrum and the cerebral parts of the cord, it seems very

difficult to explain. It does occur to one as a partial explanation, that once this state of matters had become fully established, it would be difficult for the cerebral arteries to dilate freely, so long as the central irritation continued, as the venous congestion in the periphery of the brain, by exerting a state of persistent high tension over the cerebral surface, would tend to act as an obstruction to their dilatation; and the more so if I am correct in believing that there had been over-action of the arterial system generally, as this would lead more and more to the accumulation of the blood within the venous system under consequently ever-increasing tension, so that it would constantly become more and more difficult for the meninges to be cleared of their congestive contents under the action of the small stream of blood that the spasmodically-contracted arteries would allow to pass.

An unexpected amount of support to the conception of the essential pathology of eclampsia, which I have been here striving to present, was afforded by Dr. D. J. Hamilton during the discussion on this paper in the Edinburgh Medico-Chirurgical Society. This consisted in a reference by him to the results he had obtained some years ago from the examination of the cerebro-spinal system in a series of four chronic epileptics. The paper in which these observations are contained, Dr. Hamilton has not yet published, but he has since the meeting kindly allowed me a perusal of the manuscript. In all the cases examined, it was found that in the medulla oblongata, near the situation in which the sub-inflammatory changes were noticed in my case, there existed a great amount of induration, approaching the cartilaginous in character. Dr. Hamilton concludes, from the pathological appearances presented by the specimens, that the induration was most probably of the nature of a cirrhosis in the medulla oblongata, and believes that he can trace three stages in its progress,—the first stage consisting of an exudation of

a highly nucleated fluid plasma; the second being occupied in the organization of this plasma, and its conversion into a fibrous texture; whilst during the third stage there is observed compression of the nerve tubes within the meshes of the fibrous stroma formed as indicated, and consequent fatty degeneration of them.

The diseased conditions in Dr. Hamilton's cases extended to considerable distances down the cord, and only slightly upwards towards the brain proper. Their point of maximum intensity was opposite the first cervical vertebra, but it was very markedly developed in all the cases in the medulla oblongata.

Dr. Hamilton believes that these cirrhotic changes in the medulla and cord are traceable to sub-inflammatory action. The observations were made upon the bodies of four fatal cases taken as they occurred, no selection being practised. It would thus appear that changes similar to those which were observed in the present case are not unfrequently, if indeed they are not invariably, to be found in the medulla oblongata in cases of severe epilepsy. As, moreover, the symptomatology of the two diseases is so very much alike, it certainly seems to furnish a considerable amount of support to any theory of their causation, that such theory should show both diseases to possess an identical pathological basis. Besides, I have always felt that neither the toxæmic theory nor the mechanical theory, taken alone, is capable of explaining eclampsia; and, in my previous paper on 'Albuminuria and Puerperal Convulsions,' published in 1876, I strongly insisted upon the necessity of predicating a special nervous condition as a predisposing cause in all cases of eclampsia. In this view I am supported by the expressed opinions of Dr. Tyler Smith and Dr. W. Playfair. May not, therefore, the view of an irritated vaso-motor system, advocated in the present paper, be fairly

regarded as supplying an explanation of that peculiar condition of the nervous system predisposing to eclampsia, which we have hitherto sought for in vain?

I am likewise very much confirmed in my opinion regarding the nature of the various processes at work in the production of these effects, by the results obtained therapeutically from two agents that are very unlike, and yet agree thus far, that they lower the tone of the vaso-motor centre,—I mean chloral and blood-letting.

I can confidently appeal to the experience of all my confrères who have had the opportunity to watch cases of puerperal convulsions, and who have tried both chloral and other medicaments in the treatment of eclampsia, and feel certain that they will assure me that chloral has given them by far the best results.

The older authors, long before we knew anything of chloral, obtained astonishing success from blood-letting. I have myself recorded one case where blood-letting gave excellent results. But wherein is the common bond of action? Certainly blood-letting and chloral do not appear to have much in common. I was interested, when ransacking the literature of the vaso-motor system, to find what I think helps materially to answer this question in Foster's *Physiology*.[1] The following is the part that bears on this subject:—

'Now, if the central stump of the divided sciatica (or any other nerve containing afferent fibres) be stimulated under urari, a rise of pressure, sometimes the exact reverse of the fall caused by stimulating the depressor, is observed. The curve of the blood pressure, after a latent period, rises; it begins to rise without any change in the heart's beat, gradually reaches a maximum, and after a while slowly falls again, even though the stimulation be still kept on. So constant is this result, that it has proved of great value in

[1] *A Text-Book of Physiology*, 2d edition, p. 163.

determining the existence of afferent fibres in any given nerve, and even the paths of centripetal impulses through the spinal cord. If, on the other hand, the animal be under chloral instead of urari, a fall quite similar to that caused by stimulating the depressor is observed, instead of a rise. Thus, according to the condition of the vaso-motor centre, or to circumstances affecting it, the same stimulation of the same nerve may at one time produce a fall and at another a rise of blood pressure, *i.e.* may either depress or exalt the action of the centre.

'The causes of this difference are not yet clearly worked out. Variations in respiration will not explain it. Nor can the solution be found by supposing that in urari poisoning cerebral functions are active, while in chloral poisoning they are in abeyance.

'If the brain be removed without much bleeding, subsequent stimulation of a sensory nerve under urari still gives a rise of pressure. If there be much bleeding, however, a fall is witnessed. This suggests the idea that after bleeding and under chloral the vaso-motor centre is enfeebled or exhausted, and that stimulation of the enfeebled or exhausted centre always causes depression.'

Now, the effects of chloral in lowering vaso-motor action seems sufficiently to explain its therapeutic use in considerable and sustained doses in eclampsia, whilst there is no doubt about the effect of blood-letting, even to a moderate amount, in diminishing vascular tension, *i.e.*, as I take it, in depressing the action of the vaso-motor centre.

This fact, further, in a great measure removes from my mind a consideration that has always weighed very much with me as a serious objection to the acceptance of the toxæmic theory of eclampsia. I mean the argument that it was difficult to understand how any substances of a poisonous nature should be present in the blood in such amount as to

cause convulsions, whereas, if a small fraction of their absolute amount were removed by blood-letting, the convulsions ceased. Regarded from the point of view of simple toxæmia, such a result has always appeared to me exceedingly unlikely, and this belief has hitherto compelled me to lean towards the Traube-Rosenstein theory. If, however, we look upon the effect of the blood-letting, not as a mere separation from the system of a few ounces of blood, but as an influence directly lowering the action of the irritated vaso-motor centre, it does seem to me that its beneficial consequences are very much more intelligible.

The temporary congestion of the face during an eclampsic seizure in no way militates against the view that there was general arterial contraction, as that phenomenon is merely the result of the partial asphyxia arising during the fit in consequence of the tetanic condition of the respiratory muscles. The patients in the intervals between the attacks are observed to be pale. The accumulation of an undue proportion of blood in the venous channels throughout the body, in consequence of contraction of the arteries, would account for the congested appearances of the liver, lungs, and other internal viscera found invariably in the post-mortem examinations of patients who have succumbed to an attack of eclampsia.

It has also occurred to me that the intense headache, which patients so frequently complain of when an attack of eclampsia is imminent, may possibly result from the congested condition of the meningeal membranes, as well as from the dragging that would arise upon the cerebral membranes from the shrinking centripetally of the brain substance consequent upon the loss of volume in the more central portions of the organ, which the acute anæmia would necessarily induce. The flashing of light, and other disturbances of vision, may also be referred to the central circulatory changes taking place in the brain.

It will thus appear that I accept the old toxæmic theory as after all the most probable starting-point in cases of puerperal eclampsia; but that, by reference to exact anatomical observations, I am driven to the conclusion that the poison acts by irritation of the great vaso-motor centre for the body in the medulla oblongata. My conclusions, therefore, merely tend to localize an effect that was previously assumed to act somehow, without any attempt being made to determine very particularly its exact mode of action.

Whether such a condition of the medulla oblongata will be found in all cases of puerperal eclampsia, can only be determined by many additional post-mortem examinations. The value of the present case, taken by itself, in settling such a question cannot be great. Still it is to be hoped that observers may be led by it to explore with great care not only the condition of the brain, but also that of the medulla oblongata and the cord in similar cases, as they arise, with the view of detecting the presence or absence of the pathological lesions indicated. The extremely interesting observations of Dr. Hamilton on the medulla oblongata of chronic epileptics surely of themselves demand such an amount of attention from pathologists, more especially when they consider the obscurity which at present envelopes the whole series of important questions that group themselves around the term 'epileptiform.'

In thus bringing forward the idea that irritation of the vaso-motor centre may prove to be the most essential point in the pathology of puerperal eclampsia, I merely do so tentatively, and because this hypothesis appears to me to include within its sweep a larger number of the facts of the case than any other I could find.

Our conceptions of the essential nature of such convulsions are at present so crude and defective, that any idea thrown out regarding it must of necessity be tentative.

It may ultimately be demonstrated that some of these cases arise in the manner described above, and that others must be explained by reference to secondary anæmia produced according to the mechanical idea of Traube.

### OBSERVATIONS ON THE CONDITION OF THE KIDNEYS IN CASE I.

When I leave the consideration of the brain changes, and proceed to remark upon the cause of the peculiar alterations found to exist in the renal epithelium, I feel unable to suggest any likely ætiology, except by assuming that it resulted from some special influence exerted by the pregnant condition. This is the view that Bartels[1] is led to entertain regarding the cause of the frequent attacks of parenchymatous nephritis, which he believes is the form of the renal disease specially associated with pregnancy. All attempts to explain the very frequent occurrence of renal disease in connection with pregnancy on purely mechanical grounds, such as local pressure upon the renal veins, must be allowed to have proved in a great measure failures. We, however, see a great disposition to degenerative changes in the epithelial cells of the liver in connection with pregnancy, and yet we are unable, with any degree of certainty, to explain the ætiology of these alterations, except by referring them to some occult influence exerted by the pregnancy, whether this arises in the condition of the nervous system, the blood and blood-vessels, or in the other tissues. The view that what is usually called the parenchymatous nephritis of pregnancy is not a nephritis at all, but a degeneration of certain renal epithelium, establishes, it appears to me, a certain analogy between the renal condition in eclampsia and the hepatic condition in acute yellow atrophy, and leads us to expect that they may both be ultimately found referable to a similar cause.

[1] *Loc. cit.* p. 277.

Some observations by Virchow, quoted by Bartels, p. 282, *loc. cit.*, point so clearly in this direction, that I cannot refrain from translating them at length:—'In both organs (the kidneys and the liver), and it is a question whether we should not have added the spleen also, the same parenchymatous swellings present themselves, conditioned by the taking up of a granular, cloudy, seemingly albuminous mass into the interior of the gland cells, whereby the organ becomes larger, loses in consistence, and, after separation of the capsule, seems more flabby. These changes frequently assume the inflammatory character; and one can at once describe them as parenchymatous nephritis and hepatitis. At other times their inflammatory nature is less striking, and it may then be sufficient to speak of an albuminous infiltration. In both cases the secreting power of the organs appears to suffer, and further investigation only will show which exerts the greater influence.'

Bartels, from his observations on this note, evidently thinks that the two conditions referred to by Virchow are only different stages of the same disorder. But, from the very peculiar character of the epithelial alterations noticed in our first case by Dr. Hamilton, it seems as if the epithelial degenerations detected were something quite different from the first stage of an ordinary parenchymatous nephritis. Besides, our case was one that it would almost be impossible to surpass in regard to severity. So that, if we are to consider it as being only the first stage of the disorder, it seems difficult to imagine what the advanced stage could be.

If we regard the renal condition as a degeneration, and not a true nephritis, another peculiarity of the kidney affection when it arises in connection with pregnancy receives considerable elucidation, viz., why it is found to occur without fever symptoms, such as rigor or high temperature. If it shall turn out that the majority of cases of acute renal disease

occurring in connection with pregnancy are similar in essential characters to this one, then the absence of fever symptoms would naturally be explained by the fact that the changes taking place in the kidneys were not of a truly inflammatory nature.

Furthermore, it has always been a subject of astonishment, that in cases of puerperal albuminuria, however copious the amount of albumen might be, however severe the general symptoms, and however plentiful the tube-casts, so soon as delivery is effected and improvement once begun, the return to health, in the vast majority of cases, is with a rapidity and completeness quite foreign to the experience of physicians in dealing with acute nephritis traceable to other causes.

If, in the future, we are entitled to regard the renal condition as one of limited degeneration of epithelial cells in certain peripheral tubules, with consequent mechanical closing up of the rest of the tubules, as already explained, their tissues being quite healthy, it is to my mind easy to understand that, so soon as a more healthy condition was initiated within the kidneys, the colloid plugs would drop out from the tubules, leaving the great bulk of the organ in a condition of health.

## QUESTION OF THE ORIGIN OF THE MINUTE CEREBRAL APOPLEXIES FOUND IN CONNECTION WITH ECLAMPSIA, AND THE RELATION OF THESE TO THE CONVULSIONS.

Let us now direct our attention to the small extravasation observed in the nucleus lenticularis of the right corpus striatum, and endeavour to account for its origin and define its relation to the convulsions.

There is no doubt that in such cases small apopletic extravasations are occasionally found in the cerebral tissues, both in its deeper and more superficial parts; that they

frequently occur in the meninges, is testified by many authors, such as Kiwisch, Braun, Spiegelberg. I may be allowed also to mention that Dr. J. Matthews Duncan informed me, several years ago, that he was present along with Dr. Littlejohn at the post-mortem examination of two patients who died of puerperal eclampsia, in whose corpora striata and in the parts of the brain adjoining there were detected numerous minute apoplectic extravasations. This statement is referred to in my papers upon 'Puerperal Eclampsia,' published in the *Obstetrical Journal* in 1876; and a similar state of matters is advocated as a cause of so-called uræmic convulsions by Dr. Mahomed, with his distinguishing ability, in the *British Medical Journal* of 7th and 14th July 1877. The extravasation mentioned was the only one present in this instance, for we searched with the greatest care every portion of the brain, without being able to find another.

How, then, do these extravasations arise, and what relation do they bear to the eclampsia?

It is comparatively easy to understand the occurrence of apoplexies in the meninges. In this case we trace them to extravasations which have been developed out of the pre-existent meningeal hyperæmia, which we have observed to be present in a very pronounced degree.

But when we try to discover a cause for the apoplectiform extravasations that are found in the more central portions of the brain, we encounter a task of much greater difficulty.

I believe that in this case the apoplexy was the consequence of the exhaustion occasioned by long-continued vascular spasm. There is no evidence of any commencing inflammatory change in its neighbourhood, such as was detected in the medulla oblongata, so that we cannot assume that there was any directly irritative influence at work upon the blood-vessels. It accordingly appears to me probable that the condition of arterial spasm, to which we trace the

cerebral anæmia in this case, had led to defective nutrition of the vessels of the parts most under its influence, and thus had so exhausted their retentive power as to cause the formation of this small apoplexy. It has also been suggested that more than probably the cerebral anæmia from spasm alternated with occasional dilatation of vessels during life, and that during one of these periods of vascular turgescence rupture may have taken place. According to either of these views, the apoplexy is merely a collateral phenomenon, and bears no causal relation to the eclampsia.

Dr. Mahomed[1] has made some important observations upon the production of albuminuria in connection with pregnancy, in which he brings forward a new idea of the relation between these apoplexies and eclampsia. According to him, the blood during pregnancy, containing an excess of effete matters, tends to induce and maintain high tension in the arterial system, and thus to alter the normal relation that exists between the blood and the tissues. This condition, if sufficiently severe, is followed by transudation of the crystalloids of the blood, and these, notably hæmoglobine, will be found in the urine. If this state of matters is allowed to continue unchecked, albumen is subsequently found. According to this author's view, the renal disease and albuminuria, so frequently met with in relation to pregnancy, follow from the altered condition of the blood as to quality and tension. The proximate cause of these diseases, according to this supposition, is some accidental circumstance which throws an amount of work upon the kidney, which, under its unfavourable conditions of secretion, it is unable to perform. In this way the kidney disease is made dependent upon the circulatory system, and not upon any primary defect in the renal organs.

Either pregnancy or albuminuria, and much more, therefore,

[1] *Medico-Chirurgical Transactions*, London, vol. lvii. p. 272.

both conditions when coexistent, tend to unduly exalt vascular tension; and if this produce rupture of minute vessels in the cerebral tissues, we may have as a result eclampsia. According to this view, the cerebral apoplexies that cause the convulsions are due to the vessels yielding to the extra tension under which the circulation is maintained, when diseased kidneys, an hypertrophied heart, and possibly also pregnancy, coincide in maintaining a specially high pressure in the blood vascular system.

It is in cases of chronic renal disease in which Dr. Mahomed has found these minute apoplexies which he is inclined to regard as one cause of eclampsia. We of course would naturally expect to meet with these extravasations more readily in chronic cases of renal disease, in which we usually find general vascular degeneration associated with cardiac hypertrophy. But the influences, whatever they be or however they are evolved, that tend to the development of these cerebral extravasations are manifestly very efficient in connection with eclampsia. Otherwise they could not have led to rupture in our case, in which the vessels were in a perfectly healthy condition.

Whether these minute apoplexies could of themselves be the efficient cause of eclampsia, as suggested by Dr. Mahomed, seems to me extremely doubtful. I certainly am more inclined to believe that they are rather a result of the main lesion which causes the fits, than themselves the cause of the convulsions. Certainly the minute apoplectiform patch revealed by the sectio in my case could never by any imaginary arrangement have been the cause of these terrible convulsions.

In connection with this question, I am constrained to refer, in closing, to an interesting case published by me in the *Edinburgh Medical Journal* for June 1875, where the exciting cause of the convulsions, which, however, assumed a tetanic

form, was intense venous congestion of the central cerebral ganglia, due to occlusion of the straight sinus and venæ Galeni by a firm thrombus.

But, after all, severe anæmia of the cerebral centres and intense venous congestion are practically the same in their effects,—that is, in both cases no arterial blood gets to the brain tissues.

The corpora striata in the case referred to were riddled with small extravasations, the result of the venous obstruction. I have not referred to them in the earlier part of this paper, as I am convinced that the cause of this lesion was so distinct from that of ordinary eclampsia, that facts observed in relation to this case could not be applied in explanation of the latter condition, except in so far as they indicate the effect of irritation of the motor cerebral centres in the production of convulsions of a general character.

The following are the chief points of the case. They are worth some attention, were it for nothing but the very unique pathological condition in which the brain centres were found:—

CASE III. — *Convulsions of a Tetanic nature supervening twelve days after Delivery — Death from Exhaustion — Post-mortem; Thrombus of Straight Sinus and of the Venæ Galeni—Both Corpora Striata riddled with small Apoplexies, Clot in Lateral, Third and Fourth Ventricles.*

About 10 A.M. on the morning of the 9th of March 1875, I was hurriedly summoned by Mr. Bentley, one of my pupils at the New Town Dispensary, to see a patient to whom he had been that morning called, and who, he stated in the note, seemed to suffer from puerperal convulsions. She had been married for three years, and had had two children. The eldest, a healthy girl, was born about one year after

marriage, and is still alive and well. This child, however, was suckled by the patient during the whole period of her second pregnancy, and even up to the commencement of her labour.

The second confinement took place on the 26th day of February 1875, at 3.50 A.M., when she was delivered by Mr. Bentley of a male child, after an easy labour of a few hours' duration. All the stages were natural and normal. The placenta was removed about fifteen minutes after the birth of the child, with little or no effort on the part of the attendant. The labour would appear to have supervened about three weeks or so before the completion of the full term of utero-gestation.

The patient had continued to progress favourably until about 2 A.M. on the morning of the 9th of March, when she was observed sitting up in bed, bent forwards. It is to be noticed that on the previous day she had been in her usual health. When she sat up at this time she complained of no pain, and in fact never spoke, but appeared very restless, rubbing her legs and clutching at her hair.

On Mr. Bentley's arrival at 9 A.M., the patient was completely unconscious, and there were present strong tetanic spasms, similar to those described below, only more violent in character.

On my arrival I made the following note of her condition: —Face pale; general appearance anæmic; skin soft and dry, but feels natural in temperature; pupils somewhat contracted, and almost completely insensible to light; conjunctivæ insensible to touch; eyes glazed-looking. Patient completely unconscious both during and between the attacks of spasm. Urine expelled involuntarily. Spasms of a purely *tetanic character* occur at intervals of about a minute. During these seizures the heels and the occiput are the only parts of the patient's body which touch the bed, the contractions being thus mainly opisthotonic.

During each seizure the muscles of the back are seen to be strongly contracted. The upper extremities are powerfully flexed, the fingers being bent forcibly towards the palm and the thumb turned inwards, whilst the forearm is powerfully flexed over the upper arm.

The slightest irritation applied to the skin, such as the touch of a cold hand, is sufficient to throw the patient into complete opisthotonos; similar attacks are also induced by every effort at examining the patient's condition.

The muscles of the neck are powerfully contracted during the seizures, more particularly the sterno-cleido-mastoid; but there is no risus sardonicus, and it is only during the more severe attacks that trismus occurs. Every 1½ minute or so an attack occurs, and every fourth or fifth attack is much more severe than those intermediate. There is no congestion of the face during the spasms. Though the opisthotonos can be induced at any time by application of external stimuli, the attacks recur spontaneously at regular intervals of about 1½ minute.

Pulse 74, weak, soft, compressible, and somewhat irregular. Respirations during intervals between the fits shallow and quick, being 26 per minute, but during the attacks they become stertorous. Temperature per vaginam, 101°.

Attempts to get the patient to swallow induce violent fits, but prove wholly abortive.

On examination per vaginam and per hypogastrium, the uterus, parametria, and vagina seem perfectly normal.

A catheter was now passed, and about a tablespoonful of water extracted from the bladder.

This, on cooling, exhibited a profuse deposit of urates, and was of acid reaction, but on heating with nitric acid did not give the slightest trace of albumen.

The following treatment was then ordered :—

℞ Hydratis chloralis, . ʒiij
Aquæ, . . . ℥iij. Solve.

*Sig.* A tablespoonful to be mixed with a little warm water and injected into the rectum every four hours.

The patient gradually got weaker, and died at 2.15 P.M.

A post-mortem examination was performed by Dr. John Wyllie, forty-six hours after death, with the following results :—

Brain.—On opening the skull, about two ounces of serous fluid escaped from the lateral ventricles. The cerebral convolutions were flattened, and some venous congestion of the cerebral surface was present. Both lateral ventricles were filled with dark blood-clots, which passed forwards into the anterior cornua, and backwards into the posterior cornua. The amount of clot seemed to be about half an ounce in each ventricle, and there was some laceration of the ventricular walls. In each corpus striatum there were a vast number of small extravasations, varying in size from that of a millet-seed to that of a pea. This condition was found to extend throughout the entire tissue of both corpora striata. The third ventricle also was filled with blood-clot, which again was found to extend along the iter to the fourth ventricle, and hence to the inferior sulcus between the two lateral lobes of the cerebellum.

As a result of the hæmorrhages into their tissue, both corpora striata were torn up and softened. The venæ Galeni and the straight sinus were found occupied by a large, dense, firm, and adherent thrombus. This condition extended also into the left lateral sinus, but in its cavity the clot was not so firm. The blood in the right lateral sinus was fluid. Other viscera were found healthy.

It will be seen that this case presented many symptoms

akin to eclampsia, only that the spasms were tetanic and not clonic, or mixed tonic and clonic, affected the muscles of the posterior aspect of the body especially, and were capable of being excited by the slightest external stimulus, such as the application of a cold hand to the skin.

So far as I can gather from a somewhat careful study of the scanty literature of so-called puerperal tetanus, I am inclined to regard it as a lesion entirely distinct from surgical tetanus, and to be more frequently traceable to some form of septicæmia. At least the cases recorded seem to me to point in that direction. In the present instance the character of the convulsions was unmistakeably tetanic, and no doubt they were referable to the structural lesions observed in and about the corpora striata. Occurring as these tetanic spasms did in close relation to the puerperal state, they are perfectly correctly designated puerperal tetanus. But it is never meant by this expression, so far as I understand the terms puerperal tetanus, that the disease is necessarily identical with ordinary traumatic tetanus. When we seek for an explanation of the multiple apoplexies in this case, we find an immediate and obvious cause in the obstruction produced by the thrombus in the straight sinus and venæ Galeni. In consequence of the presence of the thrombus, those portions of the brain draining into the straight sinus and venæ Galeni, whether directly or indirectly, must of necessity have been thrown into a condition of intense venous congestion so soon as the impermeability of the straight sinus was established. This restriction as to drainage includes, among others, the venæ corporum striatorum, and the venæ choroideæ, as well as some cerebellar veins. In the congestion arising from this blocking up of the venous channels, it seems to me that we have the proximate cause of the fits.

Whatever view may be entertained of the precise share taken in such convulsive movements by the other parts of

the brain, and especially by the cerebral convolutions, as also of the relation of these ganglia to one another and to the brain, it may be held as proved that the optic thalami, the corpora striata, and the corpora quadrigemina are great co-ordinating centres of motion for the various parts of the body; and that, when any irritative influence is at work within the space of the brain occupied by them, we are certain to have general convulsions, with a tendency towards opisthotonos if the corpora quadrigemina are specially affected, and to pleurosthotonos if one corpus striatum is more irritated than the other. Destruction of the tissue of these centres, again, leads to arrest of motion, or paralysis.

It appears to me more than probable that, had we seen this patient in the early part of the attack, we should have found the spasms general throughout the body, and probably very like, if not identical with, what we call eclampsia; but that after the attack had lasted for some hours, and the extravasations into the ventricles and into the tissue of the corpora striata had advanced so far as to lead to pressure upon and destruction of the tissue of these ganglia, their functional activity necessarily failed, and, instead of exalted motor activity in the muscles regulated by them, arrest of such action resulted.

Up to the last, however, the corpora quadrigemina were in a different condition. Though the great mass of blood supplied to them drains into the straight sinus, and though, consequently, the same condition which congested and broke up the corpora striata led to irritative congestion of the former ganglia, yet whether, owing to the vessels in relation to them being of stronger tissue, or for some other reason, the congestion had not in their case led to extravasation, and consequently we had exaltation of their special function in the form of opisthotonos.

There is some difficulty in accounting for the sudden onset

of the seizures, as, from the amount and the firmness of the venous thrombus, we cannot doubt but it must have existed for several days previously to the commencement of the fits. This circumstance is most satisfactorily accounted for by the supposition that only immediately before the attack did the venous sinuses become absolutely closed, having previously admitted an amount of blood to pass, sufficient to prevent the occurrence of that degree of congestion capable of causing convulsions. A further difficulty suggests itself regarding the origin of the venous thrombus. It is impossible to affirm with certainty how it originated. It would seem, however, that we must seek the explanation of its appearance in the peculiar condition of the blood that is almost universally allowed to accompany the puerperal condition. At any rate, there does not appear to have been any other cause in this patient's case likely to establish venous thrombosis in any part of the body.

I have thus been able to show, by direct appeal to dissection, that ordinary puerperal eclampsia is apt to be accompanied by minute apoplectic extravasations in the region of the corpora striata. But I have already said that I am compelled to look upon this condition as a result of the continued operation of the brain changes that cause the fits, and not as itself a cause.

I have also been able to refer to a case in which the venous congestion that led to the apoplexies seemed to me to have been the cause of severe general convulsions of a tetanic character. It appears, however, that in this case the extravasation that followed from the prolonged congestion limited, and ultimately terminated, rather than caused the fits.

Indeed, the prevention of the access of arterial blood to the brain motor centres, however induced, would appear to have been the essential condition that determined the convulsions in all three cases. Only for want of a better

term, we have called it in the third case irritative venous congestion.

GENERAL CONCLUSIONS. — Leaving, therefore, this collateral question of the relation of minute cerebral apoplexies to eclampsia, let me finish this paper, already too long, by one or two remarks.

I have honestly endeavoured, by an examination and analysis of the very peculiar appearances in the brain and kidneys in my first case, to build up a reasonable conception of the essential pathology of the convulsions, which I have traced to the production of extreme anæmia of the cerebral centres, caused by the irritative action of an impure blood upon the vaso-motor centre in the medulla oblongata.

Whether my views be correct as regards even that case, is a subject that I freely admit is open to question. Whether, also, that case may be taken as a type of its class in regard to the cerebral and renal appearances in eclampsia, is a matter that can only be settled by a careful examination of the brain and kidneys of many fatal cases of the disease. All I wish to say is, that the conditions were so very decided, that I cannot help thinking that most of them will be found present in well-marked cases of this disease, and also that my second case, to a certain amount, supports me in this view.

It is highly probable that in cases of puerperal eclampsia in which the kidneys are perfectly sound, irritation originating in the uterus and terminating in an eclampsic seizure, acts by irritating reflexly the vaso-motor centre in the medulla oblongata, and thus inducing sudden anæmia.

This theory would explain the mechanism of Cohen's[1] eclampsia uterina.

But it is well known that many causes extremely dissimilar in character may lead to similar convulsions.

[1] *Archiv für Gynäkologie*, Bd. vii. S. 107.

I have, however, no wish to push my conclusions to an undue length, and shall be glad if this effort of mine stimulate other observers to inquire into and elucidate this dark region, even though the result should be to negative my favourite hypothesis.

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THE END.

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