Contribution to the pathology of puerperal eclampsia / by J. Braxton Hicks.

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CONTRIBUTION

TO THE

PATHOLOGY OF PUERPERAL ECLAMPSIA.

BY

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CONTRIBUTION TO THE PATHOLOGY OF PUERPERAL ECLAMPSIA.

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The present imperfect state of our knowledge respecting the pathology of puerperal convulsions has induced me to bring before this Society certain facts in connection with this disease which I think have hitherto been unnoticed, but which are of importance to be borne in mind when we endeavour to discover the cause of these attacks; particularly as the real value and relative frequency of these points have not yet been ascertained: and therefore I need scarcely apologise, if, in presenting them to your notice, I do not attempt to build any theory upon them.

It may be well, however, before stating them, to call to mind the state of our present knowledge of the phenomena which are found in connection with eclampsia of the pregnant or puerperal woman.

When Dr. Lever, in 1843 ('Guy's Hospital Reports,' 1843), first showed the connection between albuminuria and puerperal eclampsia, his discovery was soon confirmed by numerous observers, both at home and abroad, whose names need not here be mentioned; and the almost constant presence of albumen in the urine of the convulsed patient gave, in the then state of our knowledge, strong support to the opinion that the condition was one of uræmic poisoning.

For sixteen years before this Bright had put forth to the world his account of that disease which now passes by his name; namely, where albuminous urine was found associated with disease of the kidneys, and in which Christison showed an excess of urea was retained in the blood.

As many patients suffering under these conditions died in a state of eclampsia, it is highly probable the discovery of albumen in those attacked by puerperal convulsions was in a great measure owing to the knowlege of these facts, especially as Bright had been attached to the same school. However this might have been, it was noticed that albuminous urine was intimately associated with eclampsia of the pregnant or puerperal woman; and it was soon generally believed that these attacks were the result of uræmia: for inasmuch as anasarca had been present in many of these cases before the convulsions had begun, it was inferred that the eclampsia resulted from the association of Bright's disease with pregnancy.*

Again, it was supposed by Frerich that the symptoms of Bright's disease were owing not so much directly to the presence of urea in the blood, as to its decomposition into carbonate of ammonia. In this view he was followed by a considerable number of writers, and numerous supporters exist at the present time. But when Dr. Richardson † published the result of his researches, and showed that ammonia could be detected in the expired gases and in the blood of the healthy, and that it might be considered a normal constituent of the blood, some doubt entered the minds of observers as to the importance of Frerich's suggestion.

It is true that many investigators,‡ by experiments on animals after removal of the kidneys and injection of urea

* See Frerich, 'Die Brightsche Merenkrankheit,' &c., 1851, p. 211, "Die Brightsche Krankheit der Schwangeren."

† See 'Astley Cooper Prize Essays.'

‡ Stokvis, "On Urea as a Cause of Uræmia," 'Neder. Fijdschr.,' 1860; Schmidt's 'Jahrb.,' vol. cxviii, p. 26; Feldmann, 'Bayer Intell. Be.,' 1861; Petroffa, "On Uræmia," 'Virchow's Archiv,' xxv, p. 91, 1862.

into the veins, came to the same conclusion as Frerich; yet it must be admitted that unless it could be proved that there was present in the blood more ammonia than normal, it could not be considered as a cause of the symptoms observed.

Now, experiments have been instituted to clear up this matter. Dr. Hammond, of the University of Maryland,* in a long paper on the subject, gives the results of his experiments. He tried many plans; in some injecting at once large quantities of urea into the blood; in others throwing in smaller quantities, but more frequently; in others removing the kidneys, and watching the effect; and also, after removal of kidneys, injecting urea and urine into the veins. He examined the amount of ammonia in the breath and blood before the operation, and came to the conclusion that the extirpation of the kidneys and the injection of urea into the circulation did not cause an increased quantity of carbonate of ammonia; and he, so far as his experiments went, considered there was no evidence to cause us to believe that urea did at all decompose into carbonate of ammonia: consequently, that Frerich's theory—namely, that the symptoms of uræmic poisoning were produced, not by urea directly, but by its decomposition into carbonate of ammonia—was not borne out by experiments. These experiments are important, and appear to be conclusive.

There was another point, the importance of which I will allude to later on, which was elicited by these experiments; it had been noticed by others, but clearly shown here; viz., that when the kidneys were extirpated, at least twenty-four hours elapsed before any symptoms of poisoning appeared: sometimes it was two or three days before they occurred.

If, then, these experiments, which appear to have been very carefully carried out, are free from error, then it is clear those experiments which have gone to support Frerich, and to prove the presence of carbonate of ammonia, do not really do more than prove what had already been shown by

* 'American Journ.,' 1861, January, p. 55.

Dr. Richardson, namely, that ammonia was found in the blood.*

The experiments of Petroffa,† however, show that the quantity of carbonate of ammonia increases gradually after the extirpation of the kidneys according to the time elapsed. This, however, cannot be considered as proof that urea decomposes into carbonate of ammonia in the blood-vessels, inasmuch as it only shows that the kidneys carry off ammonia, as we well know they do under the form of urate of ammonia, which would otherwise accumulate in the blood.

Hence, in the present state of our knowledge, I hold that it would not be incorrect to say that it is not probable, when death takes place after ablation of the kidneys, that it is not in consequence of decomposition of urea into carbonate of ammonia.

It is not here disputed that carbonate of ammonia, when injected into the veins, produces convulsions; but unless a great excess of it in the blood could be proved, clearly it would not be the agent in producing the symptoms of uræmic poisoning.

Those writers upon the subject who have, without any experiments of their own, assented to Frerich's theory, must not, of course, be taken as evidence of any value in support of the theory; yet these writers, from the deserved reputation, have done much to give establishment to it, as the names of Professors Braun, Stokvis, Feldmann, &c., testify.

The larger number of writers on the subject of puerperal convulsions, however, have apparently assumed that albuminous urine is a proof of the existence of uræmia. Whether it be so or not is a most important question, as albumen forms, in fact, our only ready means of diagnosis in practice.

* Lehmann has doubts as to the existence of ammonia in fresh healthy blood. He has detected it in acute diseases. He says, also, that urea in health is present in so small a quantity, that it is only recently that he had been able to convince himself of its presence.

† Virchow, 'Archiv,' xxv, p. 91, 1862.

There has also been a tendency with many, amongst Continental writers particularly, to mix up all these cases with pre-existent Bright's disease; for as albuminous urine is the most marked symptom of that complaint, so it has too readily with some—in past time, perhaps, rather than at present—been considered synonymous with that complaint.

That puerperal convulsion is very frequently associated with two of the most marked symptoms albumen and anasarca is well recognised; but that in a certain proportion of cases neither chronic Bright's disease nor any organic affections of the kidney has previously existed, the cases I bring forward will prove, though the exact proportion of these has yet to be made out.

No doubt, a condition of the system very similar, probably identical with Bright's disease, exists in pregnant women with anasarca and albuminous urine, &c.; that is to say, intimately associated with pregnancy, commencing only after pregnancy has begun, and passing off when it is ended; to again be lit up during each successive pregnancy, till a more or less permanent state of the disease is established: and thus pregnancy may be said to be one of the causes of Bright's disease. With this state eclampsia may be associated.

Upon true pre-existent Bright's disease, of course, pregnancy may supervene; and then also we may have eclampsia associated, not infrequently, but by no means universally. Most probably with these we have uræmia, because the kidneys have been affected for a sufficient time to have allowed the accumulation of urea in the system, enough to produce some, at least, of its effects. But in this class we are more likely to have more coma than usual with the convulsions; and it may be frequently remarked that the disease of the kidneys, which was in a quiescent state before, now becomes lit up, as it were, afresh, and that its secondary effects become severe or fatal. However, in both these cases, before the convulsions there is clear evidence of anasarca and albuminous urine.

At first the frequent presence of albumen in the urine of

patients suffering from eclampsia led to the belief that it was constantly present, and that the convulsions were owing to the uræmia so commonly held to be associated with it. But I shall bring forward cases to show that up to the time of the first convulsion, and even after for a short time, the urine is perfectly free from albumen, and that there was no evidence of anasarca beforehand; but that serious kidney symptoms shortly after begin to arise, as shown by the condition of the urine.

The cases I allude to progress in this order:

A woman approaching the full period of pregnancy, apparently in perfect health, without œdema, without albumen in the urine, is suddenly seized with an epileptiform attack. After a certain time has elapsed, albumen is noticed in the urine, at first in small quantities, shortly in profusion; then blood-globules, waxy and epithelium casts, are found in it. At this time the urine becomes scanty, of high specific gravity, with very high-coloured crystals of lithic acid in considerable quantity. The case, which is now one of acute desquamative nephritis, may terminate by gradual recovery, the albumen slowly disappearing; or death may ensue from the violent effects of the original attack, or from the retention of urea, &c., in the system, in consequence of the acute mischief in the kidneys.

Now, if these cases can be shown to occur, and if albumen in the urine be an indication of uræmia, and if those experiments above quoted be right—viz., that twenty-four hours at least, after the kidneys have ceased to act, must elapse before symptoms of uræmic poisoning can occur—then it follows that the convulsions cannot be owing to uræmia, at least the result of kidney-disease.

If this point be granted—and it seems that, so far as our present knowledge extends, it must be—then the only modes of explaining the occurrence of the acute nephritis are in one of these three ways: either—

1st. That the convulsions themselves are the cause of the nephritis;

2ndly. That the nephritis and the convulsions are produced

by the same cause, *e. g.* some detrimental ingredient circulating in the blood, irritating both cerebro-spinal system and other organs at same time;

3rdly. That the highly congested state of the venous system as is produced by the spasm of the glottis in eclampsia is able to produce the kidney complication.

This seems to me to be the state of the case as it at present stands, and further than this our present knowledge will scarcely allow us to proceed—that is to say, so as at once to decide; and it is for this reason that I have considered it better to call the attention of this Society, in order that, by the co-operation of some of its members, we may the earlier obtain sufficient data by which we may hope to clear up the question, than to wait till I had single-handed collected a larger number of cases—a work of more difficulty to those engaged in consulting practice than to those who are likely to see the cases at their outset.

Now in order to decide on the first point, namely, whether the convulsions themselves produce the kidney complication, we ought to inquire how far the non-renal convulsions can produce it. I have seen it stated that it is by no means rare to find albumen in the urine after an ordinary epileptic convulsion. In confirmation of this it is difficult to find sufficient evidence in English authors, though it would be an easy matter in those who have charge of epileptics. In those cases which have come under my own observation there has been no albumen following the attack. It is very desirable, indeed, that researches were made in this direction. *A priori* it does not seem difficult to believe that irritation to the cerebro-spinal system should cause it, since we know that injuries to the spinal column do produce marked changes in the urine; though how far nephritis may result one can hardly say. At any rate, it is a point which one would think would not be difficult to clear up.

With regard to the second explanation, there are some facts which may seem to give weight to it, namely, the cerebral symptoms which, in some cases, precede the attack a few days. But it is true we cannot detect the occurrence

of any abnormal symptom up to the moment of the convulsion in a large proportion of cases of puerperal convulsions: I mean even in those cases where we find copious albumen after, with acute desquamative nephritis. Yet these cases, it must be borne in mind, are by no means uncommon; they probably form half, and these are important in their bearing upon the whole question, because it is by no means improbable but that they belong to the class of those in which no albumen is found at first, but is after the convulsion. At least, we must not at present class them with those in which albumen has been detected before the convulsion. At any rate, in them we have, from *apparently perfect health*, convulsions and acute nephritis, which soon subside in the favorable cases. If these cases are not closely watched by the microscope, and for many days, the acute condition of the kidney-attack may be overlooked. And in many of these, which have run on to almost complete suppression of urine, with blood-discs, &c., although albumen was indeed present at the commencement, yet it was in a very small quantity compared with that which followed; and it must be recollected that the urine which was examined was passed after the convulsions. Therefore, is it not a fair question to ask, If the urine could have been examined before the attack, would it not have been free from albumen?

It is certain that the explanation given by some, namely, that the anæmic condition of the brain is the cause of puerperal convulsion, will not hold good in these cases; because there is no anæmia, neither simple, nor in connection with Bright's disease, of old or recent standing.

The occurrence of albumen in the urine in pregnancy has been by some confidently explained by the pressure of the uterus on the renal veins, whereby congestion of the kidneys ensues, and, as a consequence, an oozing of serum into the tubes, with a detention in the blood of the ingredients they should excrete.

It has been asserted by a German author, that as many as twenty-five per cent. of pregnant women have albumen in

the urine. This statement has frequently been repeated in England.

But is this really the case? One would think that such a question could be most readily answered; but I have not been able to find more than fifty cases on record, and those are given by Dr. Lever :* in *none* was albumen present. To these I can add another fifty cases investigated by the gentlemen attending Guy's Hospital Maternity: in *only one* was albumen present, and in this there was a clear history of old disease of kidney. Thus we have one hundred cases taken at random, and in only one do we find albumen in the urine. This, at least, must make us hesitate to believe that uterine pressure alone does cause albumen in the urine during pregnancy. But there are other considerations which must make us doubt the value of this explanation. The first is, that albumen is found at a stage of pregnancy so early that the uterus has not yet reached the renal veins so as to compress them. Again, if pressure were the cause, we should expect to find albumen in urine in cases of advanced ovarian enlargement. But I am not able to find any confirmation of this; and, as far as the cases which have been treated in Guy's Hospital can be taken as a test, there is no evidence in favour of the pressure theory. And here again it might be pointed out how easily most of these facts may be satisfactorily ascertained by the joint labour of many of the Fellows of the Obstetric Society, whereby a large number of cases might be examined.

In the above remarks I have not alluded to those cases of puerperal eclampsia where *no* albumen has been found during their whole course, because they are exceedingly rare. Cases are on record, and doubtless are to be now and then met with. But unless the urine has been frequently examined for twenty-four hours, we cannot be certain there is none: some of the cases might easily be overlooked, so slight is the quantity and so short its persistence. It is therefore necessary to examine three or four times

* 'Guy's Reports,' loc. cit., supra.

during the first twenty-four hours after the first convulsion.

It may be here remarked, that those cases where the albumen is slight are generally of the milder type, and much mixed up with hysteria, so that they are classed under the head of "Hysterical Eclampsia." But in nearly every case where the eclampsia is at all distinctly marked, we almost invariably find albumen, and even the more marked characters of acute renal disease. It is probably only because the insensibility not being so severe, the emotional system is not completely subjugated by it; nor is it under the proper regulating power of the mind.

Before concluding these remarks, I may again be allowed to urge the importance of examining the urine as early as possible; and, if obtainable, the urine passed before the first convulsion should be obtained: after this it should be tested twice a day, to ascertain the progress of the complaint. It is not easy in practice to secure the urine at so early a period. It may be mixed with that of other persons; but here the difficulty would not be very great, because if the mixed urine had no albumen, neither patient nor the other was affected; and if albumen were found in it, then it would not be difficult to obtain that of the non-pregnant person.

I have subjoined four cases in which the urine when first obtained had no albumen, but which afterwards had a large amount. This number may appear small, but it formed half of all the cases where the quality of the urine was capable of being ascertained early; and it is to be remarked that in those where albumen was found, from the first there was also anasarca. I therefore confidently expect that, if care is taken to ascertain the state of the urine beforehand, we shall find a still larger proportion of cases which are free from albumen up to the first moment of the attack; because, as before noticed, probably many of those cases which give albumen, but which have not been examined until after the fits have fairly set in, will be found free at the very beginning, for the urine bears all the characters of that of acute desquamative nephritis.

I need scarcely remark, that observers should be very careful not to allow blood to be introduced into the urine in conducting these inquiries.

Case 1.—Mrs. C—, about twenty-five years old, primipara, was taken with eclampsia at the seventh month of pregnancy. The attacks were repeated nearly every half-hour, and were of a very severe, strangling character. The liquor amnii was drawn off, and delivery soon took place. The attack abated soon after, and then entirely ceased; much stupor continued, but this went off. About the sixth day after, she was taken with pneumonic symptoms (secondary?), and died the tenth day.

The urine which was passed just before the attack was entirely free from albumen or other abnormal state. Twelve hours after, it was highly charged with it, with waxy and epithelial casts, of high colour and specific gravity. Next day the urine, which was drawn off, became less in quantity, of higher colour, much albumen, blood-corpuscles, and copious mixture of albumen, with uric-acid crystals. This state continued for six or eight days, the albumen gradually disappearing before she died.

There was no symptom of anasarca previous to the first attack; but she had had some unpleasant sensations in the head for a day or two before, with partial loss of sight, noises in ears, &c.

Case 2.—Mrs. C—, 1865, was attacked at the early part of her first labour with puerperal eclampsia; this was repeated with great frequency, and delivery was ultimately accomplished by craniotomy. The attacks after a time gradually subsided, and the next day she became sensible. The urine was drawn off by the catheter during the early part of the case. It was examined, but not a trace of albumen existed. The urine required to be drawn off again next day, and for three or four days. On the first occasion after delivery, and upon each subsequent occasion, albumen was found in large quantity. When it was tested after the cessation of the

lochia, there was no albumen: it was not examined by the microscope. I am indebted for these facts to the observation of Mr. Burton, of Lee, with whom I had seen the case after delivery. This patient had beforehand no symptom of œdema, nor other ailments.

Case 3.—Mrs. —, 1860—primipara—was delivered by the forceps for slight contraction of the pelvis. While the instruments were being applied, she had an epileptiform fit, which was twice repeated after labour. The urine drawn off immediately after the first attack had so small a trace of albumen, that it was doubtful whether it possessed it at all. Nine hours after, its presence was strongly marked. Twenty-eight hours after the first attack, no trace of albumen was detectable. She recovered quickly. She was free from any abnormal symptom before the confinement.

Case 4.—A. B—, 1866, a well-formed girl for her age, fourteen and a half years, being in labour at full term, was taken with puerperal convulsions of great severity when the head was near the outlet. Forceps were applied, and she was easily delivered. The urine was drawn off after the placenta was expelled: it was examined; no albumen existed. The fits recurred with excessive frequency, with deep coma between; pulse 130 per minute, very weak. This continued notwithstanding treatment, and she gradually became worse, dying thirty-six hours after labour.

The urine was drawn off a second time, twelve hours after the first, and then a copious quantity of albumen was very apparent. I am indebted to Mr. White, of Tooley Street, for the notes of this case, which I treated with him.