the *Arteries*; but, on careful consideration, it is evident that we have not here an example of what is sometimes found; viz., a persisting right aortic arch, but that we have a simple transposition—a complete turning of the heart and large vessels, so that its posterior surface is anterior, and the left ventricle is carried to the right side. It is interesting to note that, although in this case nearly all the viscera had undergone a corresponding change, the lungs were in their normal position, the large three-lobed lung occupying the right side."

The heart and lungs are now in the museum of University College, London. I ascertained that the woman was not left-handed, and there had been no discovery during life of the existence of such an unfrequent arrangement of viscera.

THE PATHOLOGY OF URÆMIA AND THE SO-CALLED URÆMIC CONVULSIONS.

By F. A. MAHOMED, M.D.,

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I HAVE to thank Dr. MacLagan for his criticism of my theory of uræmia, to which, if I may be allowed, I should like to make some reply. Before referring to his arguments in detail, I may state that I was perfectly aware of Traube's theory of cedema of the brain, nor do I claim any originality for the suggestion of *adema* as a cause for the symptoms of uræmia, as will be seen by a reference to my paper, in which I refer to it as a very well known and debated question among pathologists. I was especially anxious to avoid a discussion on mere theories, and to confine myself to the observation of facts; for that reason I forbore to quote any of the old arguments, or refer to the past writings on this subject, but only mentioned the very strong support which this hypothesis had received from the recent observations of Sir William Gull and Dr. Sutton on the hyaline exudation surrounding capillaries in Bright's disease, observations which I cannot but think go far to demonstrate an actual condition of what might almost be called chronic or fibrinous cedema.

Dr. MacLagan divides his objections into general and special, and I will follow his clear and methodical plan and answer them in detail. First, as to the *general* objections.

1. Dr. MacLagan objects to study the pathology of convulsions in uræmia apart from convulsions generally. I think, on the other hand, that we can only form a true theory of the cause of convulsions in those cases in which they cannot be associated with any discoverable lesion, by studying as closely as we are able the cases in which convulsions occur associated with definite and well ascertained lesions; if we can place by future observations the convulsions of uræmia among these, I believe we shall certainly advance our knowledge of the cause or causes of convulsions generally.

2. Objection is taken to my separation of convulsions from all the other nervous symptoms associated with uræmia. This separation is not mine, but is made by disease. It is not a complete one, however; I take it that the conditions of cedema of the brain and capillary hæmorrhage may occur simultaneously in many cases, and therefore the symptoms produced by each must be coincident. Symptoms of localised disease, such as convulsions and transient paralysis, or even aphasia, I should claim as those arising from capillary hæmorrhages more or less numerous; while the more general symptoms of depressed vitality or nerve-force, which resemble those of compression of the brain, are those which I would leave cedema to account for; supposing it to exist, that it should produce both cerebral anæmia and "malnutrition of the nervous centres" are results which few will deny. To return, however, to Dr. MacLagan's objection, it is impossible to treat different symptoms produced by different lesions as due to one and the same cause. Though hemichorea may precede or follow cerebral hæmorrhage causing hemiplegia, would Dr. MacLagan describe hemiplegia and hemichorea as the same disease, due to the same lesions, and decline to consider them apart? or would he not rather take the association of hemichorea with well ascertained lesions as likely to throw more light upon the pathology of that obscure disease chorea than merely leaving it to purely theoretical generalisations?

The *special* objections offered appear to limit themselves to one; namely, that Dr. MacLagan fails to distinguish between the conditions of capillary hæmorrhages and miliary aneurisms. Again, Dr. MacLagan credits me with ignorance of a very generally known condition; but, in this case, he will find that I have particularly, and more than once, referred in my paper to Charcot and Bouchard's miliary aneurisms, and I take especial trouble to distinguish between the two conditions of capillary hæmorrhage and capillary aneurisms: practically, the great

difference between them appears to be that the hæmorrhages produce convulsions, and the aneurisms do not. Why this should be so is not clear, but it is not very difficult to conceive that blood outside a vessel is a somewhat different condition from blood inside a vessel, and that the pathology of the two should be also different.

Finally, Dr. MacLagan sums up the evidence against my supposition under two heads: first, that I do not accept the theory of "malnutrition of the nervous centres" as a sufficient cause for the convulsions in Bright's disease. To this I would reply that there are many other conditions in which malnutrition of the nervous centres occurs, and to a greater extent, without giving rise to convulsions; and that many, if not all, of those conditions in which they are ascribed to malnutrition are conditions in which the term is used in lack of any other discoverable cause for them. Dr. MacLagan must excuse me, however, if I decline to enter into a discussion of the numerous theories of the pathology of convulsions. Secondly, he says that I substitute for malnutrition "an agency whose existence has not been proved, and whose competence to produce convulsions is at least doubtful." This, in some measure, I admit, but bring what I consider powerful evidence in support of it; and I have only to ask all pathologists to put it to the crucial test of *post mortem* observation, by which alone it can be affirmed or denied.

THERAPEUTIC MEMORANDA.

CARBOLIC ACID FOR TINEA TONSURANS.

THE treatment of tinea tonsurans by the local application of carbolic acid, so ably advocated by Dr. Lee, is of older date than Mr. Rhodes seems to suppose; for it has been used, both alone and in combination with sulphur, and also with sulphur and a salt of mercury, for many years, both by the late Mr. Startin, the late Mr. Naylor, and by me, as have also those closely allied bodies creasote and liquor carbonis detergens; and it is a method of treatment very generally recognised, I believe. The application has been made with almost the frequency which Dr. Lee considers essential, and for the reasons mentioned by him. I am bound to confess, however, that these substances, though of indisputable efficacy in the great majority of cases, nevertheless do not seem to have the power of arresting the complaint in those instances in which there exists, as it were, a congenial soil for the development of the disease in some condition of defective nutrition of the skin, often connected with general disposition to low inflammatory action.

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SURGICAL MEMORANDA.

FATAL CASE OF HÆMOPHILIA IN AN INFANT.

HAVING observed in the JOURNAL of July 14th the record of a case of hæmophilia, occurring in the practice of Mr. Mac Cormac of St. Thomas's Hospital, in a man aged 42, I am induced to give the details of the following case in an infant.

On June 23rd, 1877, A. D., aged seventeen months, received a wound, which divided the ala of the left nostril, by falling on the sharp edge of a zinc bath. He was brought to me by his father as soon as the accident happened. On examination, I found he was bleeding pretty freely from the wound, which was stitched up; cold water and pressure by means of a pad of lint and a bandage were applied, whereby I succeeded in arresting the flow of blood; and after waiting some little time without any sign of its recurrence, the patient was sent home. Early on the following morning, however, I received an urgent message to attend at once, as the child was bleeding as badly as ever, and had been doing so for some time. On arrival, I found the patient saturated with blood. I at once removed the dressing, and applied the liquor ferri perchloridi fortior, which for some hours had the desired effect. The hæmorrhage, however, returned during that and the following days in spite of the various styptics which, each in its turn, was tried without success. Seeing that my patient's life was in imminent danger, as a last resource, and after consultation with my partner, the introduction of a hare-lip pin and the application of a twisted suture was resorted to; but notwithstanding this, a continuous oozing was kept up, and eventually the little patient sank from exhaustion on the 28th, five days after the accident. In this case, the hæmorrhagic diathesis was strikingly well marked, and appeared to run in the family of the mother, as she, on two occasions, nearly lost her life from hæmor-