chloroform produced a rapid lowering of the excessive arterial pressure.

At 8 P.M., the patient was still insensible, although much quieter; temperature 102.3. The os uteri was high up, and just admitted one finger into the cervical cavity. I then decided to commence the induction of premature labour, which I did by rupturing the membranes with a stylet.

At 11 P.M., the fits had not recurred; she was somewhat more sensible, and occasionally answered questions when addressed. The labour had not made any advance. Temperature 99.2; pulse 108. At 9 A.M. on the 4th, she had no more convulsions, and was much more sensible. The urine was very slightly albuminous; specific gravity 1015. At 5 P.M., the os was high up, and not more dilated; the vertex was not presenting, but the presentation could not be fairly made out. No pains had occurred. At 9 P.M., the obstetric resident was summoned, and found her in a wild, excited, semiunconscious state, with both feet of the child protruding from the vulva, the cord not pulsating. Chloroform was given, and in a few minutes the head was extracted. No more convulsions occurred; and on the 8th, the urine contained no albumen nor casts; specific gravity 1020. She recovered rapidly.

It is to be noticed in the course of this case that a considerable improvement followed the use of chloroform; but a still more decided benefit from the diminution of nerve-irritation obtained by the reduction in size of the uterus at the escape of the liquor amnii, although labour did not come on until twenty-four hours later. The temperature had continued to rise after the use of chloroform, but fell three degrees in three hours after the rupture of the membranes. The chief reason, however, for which I bring it before you, is because it shows that high arterial tension existed at the time of the convulsions, and before chloroform was administered. Now, it has been shown by the use of the sphygmograph, that the same state of the vascular system exists in chronic, and to some extent even in acute Bright's disease. It is attributed to a resistance to the circulation of the blood, either directly the consequence of its altered quality, or due to a spasmodic contraction of the small arteries, set up by its irritating effect. Now, in several cases of uremia, arising in the course of chronic or acute Bright's disease, I have found that while the temperature was diminished, the pulse became rapid; but, at the same time, the sphygmograph showed that it remained non-dicrotic, and bore a pressure sometimes greater than before, and considerably greater than normal. As a rule, when the pulse becomes rapid, it becomes dicrotic and compressible. Thus it seemed, that during the uremic state, there was a still further enhancement of that elevated arterial tension which is usual in Bright's disease, and the condition in the case of puerperal eclampsia just related was somewhat similar. It is possible, therefore, that in this circumstance may be found some explanation of the beneficial action of chloroform, which in that case produced, as it usually does, arterial relaxation. It probably acts by soothing the irritability of those nervous centres which excite the spasmodic action of the vaso-motor nerves, and either through their instrumentality, or by other means, produce convulsions and coma. A diminution of arterial tension is also one of the results of bleeding, and perhaps it is in this way that venesection has proved beneficial, as well as by causing that diminution of temperature which is known to be at least a temporary effect of the abstraction of blood. The dictates of fashion have now declared decisively against bleeding in puerperal convulsions, and no doubt it is true that, in chloroform, there has been found a less injurious means of relaxing arterial spasm, and probably in the application of cold a better mode of reducing the temperature. Nevertheless, it seems probable, that in those cases in which venous congestion has become continuous, and the temperature is rising to a high point, venesection may still be found useful to relieve the strain upon heart and lungs; while, at the same time, it tends to lower temperature and decrease arterial tension. If time allow, however, a considerable part of the same effect, including that of the removal of some of the morbid matter in the blood, may be obtained by free purgation.

With the increase of arterial tension in puerperal eclampsia may be associated the fact that such an increase of tension in minor degree is a normal, or, at any rate, a common condition in pregnancy. This is indicated sphygmographically by an increase in the tidal wave in the pulse, and in the pressure required for its development. It seems to be attributable to the presence in the blood of some irritating material, so that its behaviour is like that of blood which kidneys, deficient in excretory power, have failed to purify. I think that this condition throws light upon the causation of nephritis in pregnancy. It has often been described as being due to mechanical pressure; but, although this no doubt takes some part in producing the result, yet there are several reasons for considering that it is insufficient in itself to account for the whole. For the nephritis, and even the convulsions, are sometimes found to occur as early as the third or fourth month of pregnancy,

when pressure could produce little effect; and experience shows also that in cases of ovarian or other tumours, which may produce even greater pressure than pregnancy, albuminuria is very rare, and nephritis still rarer. Again, heart-disease may produce a still greater venous congestion, and albuminuria may result; but the kidneys do not show the signs of inflammation which are found in fatal cases of puerperal eclampsia. It would seem, then, that the nephritis of pregnancy is due in part to the presence in the blood of some deleterious material, which also causes an elevation of arterial tension. It has been shown by Mr. Mahomed, in a paper read last year before the Royal Medical and Chirurgical Society, that a similar elevation of tension occurs in scarlatina, and forms a stage antecedent to that of albuminuria.

It is extremely unlikely, however, that in puerperal cases this injurious material in the blood should be developed so suddenly as to produce directly and independently both eclampsia and a fully developed nephritis within the space of a few hours. And the facts brought forward by Dr. Hicks, showing that the appearance of albumen in the urine in some cases does not precede the eclampsia, or even follow it after an interval of an hour or two, do not, when rightly considered, prove that nephritis is not the cause of the convulsions. For, although albuminuria is the chief available sign of kidney-disease, it is not necessarily, either according to logic or experience, its first effect. Thus we know that chronic degeneration of the kidney may exist for some time, and produce decided effects upon the system, before any albuminuria occurs; and, again, cases are known of scarlatinal dropsy, in which the dropsy precedes by some hours, or by even a day, the appearance of albumen in the urine. As, therefore, in that case, the failure in the excretion of water precedes the albuminuria, so, in the case of puerperal nephritis, may the loss of power to excrete solid matters.

I would draw, therefore, the final conclusion that there is much ground for the conjecture that the primary cause, both of puerperal eclampsia and of albuminuria, is the presence of some injurious matter circulating in the blood; but that there is strong evidence to lead us to believe that it is not sufficient by itself to produce the convulsions, until nephritis has actually commenced, although in a few cases it may not yet have reached the stage of albuminuria. It is to be remembered, at the same time, that now and then, although very rarely, cases of puerperal convulsions occur in which no albumen can be found in the urine either before or after the fits. The pathology of these would seem to be entirely different, and to be more nearly allied to that of ordinary epilepsy.

CASE OF CONGENITAL SYPHILIS, WITH ASCITES, IN A CHILD THREE MONTHS OLD: DEATH: POST MORTEM EXAMINATION.

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O. A. II., a boy aged three months, was brought to me at the Hospital for Sick Children in Great Ormond Street on April 24th, 1874. The child's face showed the yellow "bistre" tint very conspicuously; it had snuffles and much acrid discharge from the nose, which was excoriated. The buttocks and genitals were covered with a dark-red dry erythematous eruption. The legs and feet were highly oedematous, and the abdomen distended to a great size with fluid; the navel being very protuberant. The liver was large. The child was much emaciated, but this was so masked by the oedema that it had not been specially noticed by the mother. It was ascertained that the latter, a most respectable healthy-looking woman, had had two miscarriages, and four years ago had given birth to a stillborn child, which the doctor stated must have been dead for a month.

The child was said to take its food well, and supposed to be in fair health, but for the dropsy and the "thrush", or eruption on the genitals and buttocks. It was ordered a grain of grey powder every four hours, and mercurial ointment was directed to be rubbed in night and morning. When next seen, at the expiration of a week, it was reported to be better in every way. The oedema had decreased in a remarkable manner, and the ascites was also less. The extremely emaciated condition of the child, with the skin hanging in loose folds on its arms and legs, was now rendered very manifest by the subsidence of the dropsy. The same treatment was continued with apparent advantage, the dropsy regularly diminishing until the limbs were free from oedema. There was no purging, nor were the stools green, but the bowels were regular and the motions natural. On May 8th, however, a fortnight after its admission, it was seized with convulsions, and died the same evening.

Post Mortem Examination.—The body was extremely emaciated; the abdominal walls were very thin, with hardly any subcutaneous fat; fat had also almost entirely disappeared from the omentum. The muscles were pale and wasted. The lower lobes of both lungs were almost entirely empty of air, either atelectatic or collapsed, or fibrous, not congested. The upper portions were fully crepitant, and natural in appearance. The right side of the heart was distended with soft black clot; the left contracted and empty. On opening the abdomen, it was found to contain a large quantity of clear straw-coloured serum, free from flakes. The gall-bladder, cystic, hepatic, and pancreatic ducts were firmly matted together, and to the duodenum and transverse colon, by old firm adhesions. There were no signs of peritonitis elsewhere. There was no recent lymph. The liver was about the natural size, or a little enlarged, and its surface had a peculiar mottled appearance, marked out into lobular divisions by lines of lighter colour. It was irregular to the touch, the elevations being broad and flat, about the size of a split-pea, and but slightly projecting, yet perceptible enough to the finger. The interlobular markings were very distinct on section. The whole organ was extremely hard and leathery; the finger could not be forced through the tissue, even with hardest pressure. The right lobe was more extensively involved than the left, although the change pervaded the whole organ with tolerable uniformity. There were no patches or nodules of deposit. On the surface of the spleen there were one or two thickened opaque patches, and the Malpighian bodies were unusually conspicuous on section; otherwise the organ appeared natural. The kidneys were apparently quite healthy, as also were the mesenteric glands. The brain was not examined.

Microscopic examination of thin sections of the liver hardened in chromic acid disclosed most marked structural changes. The lobules were flooded by small fibro-granular deposit, which penetrated everywhere between the liver-cells, pushing them apart and pressing on them on every side. The gland-cells were, for the most part, shrunken and atrophied, and of all shapes and sizes, less than natural, in many places no larger than the normal nucleus. Here and there the liver-cells were little changed, yet pressed asunder from each other by the intruding element. In most parts, however, few liver-cells of normal appearance could be seen, the lobules being almost entirely occupied by the fibro-granular deposit and atrophied gland-cells. The cells generally were granular and turgid, and more rounded in form than the normal. Scattered about the most affected districts were patches or islets of granular deposit of varying size, often occupying a space equal to a whole lobule or more, and here no gland-cells at all could be discerned. In some of them, the material had the appearance of fibrillation; and the fibrous character was more distinct still at the free margin of a section where cells still remained imbedded. The infiltration was not confined to the interlobular districts. It pervaded the whole organ, flooding the lobules and swamping the cells. It was, however, much more abundant in some parts than in others, its distribution being irregular and patchy, yet no portion being entirely free.

Portions of two thin sections of liver, shewing fibro-granular infiltration of congenital syphilis. Above the line A. B. form a part where the change is less advanced than in the portion below the line, where the liver-cells have almost entirely disappeared. A group of normal liver-cells is represented at the side for the purpose of comparison.

In the lower lobes of the lungs, which, to the naked eye, had the appearance of being collapsed or atelectatic, the microscope disclosed other changes. Thin sections exhibited uniformly increased proliferation of the epithelial-like cells of the alveolar walls, which were in parts

thickly beaded by them. In other parts, the septa were thickened, and the air-chambers narrowed by the addition of delicate fibro-nuclear material, developed from the alveolar wall. With this were mingled in varying degree the epithelial-like cells, resembling those seen lining the alveolar wall elsewhere. In places, the cavities of a number of contiguous air-cells were completely blocked up by the fibro-nuclear material, which in some parts resembled fibrin containing a few large alveolar cells, and others of smaller size. Microscopic examination of the kidney showed what seemed to be an unusual irregularity of size in the convoluted tubes of the cortex, with some blurring of certain parts, as if by granular material; but this condition could not be satisfactorily made out. Sections of the spleen showed nothing abnormal except a general cloudiness of structure. This might be due to granular infiltration, but its actual existence could not be demonstrated.

The appearances observed in the liver correspond, to a great extent, with the morbid changes described by Gubler in one form of infiltration of the liver observed by him in syphilitic infants, and the local peritonitis with similar conditions pointed out by Sir James Simpson as one of the consequences of intrauterine syphilis. The changes in the lung differed from those met with in ordinary catarrhal pneumonia by the smaller number of large cells, and excess of simple fibro-nuclear proliferation. Similar fibrosis of the lung is described by Cornil and Ranvier as occurring in congenital syphilis; and I have observed it in two instances in adults. In these, however, the change was much more extreme, and accompanied by nodules of gummatous deposit.

It will be seen that while the ascites is explained by the condition of the liver (which Gubler found to be in such cases almost impervious to injections by the portal vein), and by the local peritonitis causing pressure on the portal vein, the general dropsy is not accounted for by the lesions which were ascertained.

And yet marked general oedema is a not unfrequent accompaniment of extreme congenital syphilis.

DIPHThERITIC PARALYSIS:

ITS NATURAL COURSE, PATHOLOGY, TREATMENT, AND RELATION TO PARALYTIC AFFECTIONS FOLLOWING FEVERS.*

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PARALYSIS DURING AND AFTER SMALL-POX.

Various affections of the nervous system occur during the course of, or as *sequela* of small-pox. Sometimes the nervous centres are implicated; at other times, we have more or less transitory conditions of paralysis, such as are met with in diphtheria. According to Gubler, who has collected and commented upon a very interesting series of cases, the paralytic affections (as also the convulsions) which occur during the period of invasion or at the onset of the disease, are less dangerous than those which supervene at its termination or during convalescence. The paralytic affections, he remarks, which declare themselves at the period of desquamation, are more protracted and serious than those which declare themselves at the onset of small-pox, and those which occur at an advanced period of convalescence are of still longer duration and of a more intractable character. They are sometimes incurable when they become localised in the lower extremities. The following statement of Gubler is true and of great importance in relation to the whole subject now before us. He says: "Secondary paralysis following measles, scarlatina, and small-pox, are essentially connected with general asthenia; they disappear when it disappears under the influence of a regimen suited to restore the strength of the patient and recuperate his economy."[†]

Gubler quotes a very interesting case of paraplegia supervening during the incubation of small-pox, reported by Dr. Raoul Leroy d'Etiolles in the chapter of his work on paralysis of the lower extremities, which is devoted to the consideration of paraplegia in severe fevers. On the appearance of the eruption, the paraplegia ceased. Numerous abscesses formed, and there was a pellicular phlegmasia of the mouth and nasal fossæ. This state was followed by paralysis of the veil of the palate, difficult breathing, pneumonia, and death. At the necropsy, no lesion of the various centres could be discovered.

* Partly read before the Medical Section at the Annual Meeting of the British Medical Association in Norwich, August 1874.

[†] Gubler (Adolphe): Des Paralysies dans leurs Rapports avec les Maladies Aiguës, et spécialement des Paralysies Asthéniques, Diffuses des Convalescents. (*Archives Générales de Médecine*, 1866, vol. i, p. 551.)