the signs and symptoms of the aortic lesion become much less obvious and may be entirely overlooked until recovery sets in and the bruit reappears.

Table illustrating the Relation of Aortic and Mitral Disease to Malignant Endocarditis.

No.	Sex.	Rheumatic Attack, Mitral and Aortic Disease.	Death from Malignant Endocarditis.
1	M.	In 9th year	In 10th year.
2	M.	In 10th year	In 13th ..
3	M.	In 6th, 8th, 10th, and 12th years	In 16th ..
4	F.	In 12th and 13th years	In 14th ..
5	F.	In 7th and 20th years	In 21st ..
6	M.	In 11th, 14th, 17th, and 18th years	In 19th ..
7	F.	In 12th year	In 16th ..
8	F.	In 9th and 16th years	In 11th ..
9	F.	In 12th year	In 16th ..
10	M.	In 7th year	In 11th ..
11	F.	In 12th and 15th years	In 17th ..
12	F.	In 8th, 13th, and 15th years	In 24th ..
13	F.	In 13th and 17th years	In 27th ..
14	F.	In 7th year	In 12th ..

This group strengthens, I think, the view that I hold that aortic and mitral disease is not of importance only because two valves are affected, but because the lesions point to a tendency to spreading activity and the possibility of malignant development.

As already stated, aortic regurgitation as a solitary valvular lesion is exceptional in childhood, and for the purposes of this article sufficient attention has been given to it under the combined lesion just described. Aortic stenosis and tricuspid endocarditis occur in childhood, but they illustrate no special principles and need not detain us.

MALIGNANT ENDOCARDITIS.

Space will not permit any detailed description of this deadly disease in childhood, which resembles in its behaviour the condition in adult life; but I would repeat here my belief that the rheumatic infection is a frequent and important cause, and will very briefly state the reasons.

There is frequently an antecedent history of rheumatism; it may occur together with such active symptoms as arthritis and pericarditis, and these may be overcome although the valvular lesion may run a fatal course. Both simple and malignant endocarditis may occur in the cardiac valves in the same case, and connecting links between simple and malignant endocarditis may be met with in childhood. Experimental inoculation with the rheumatic infection may produce both forms of endocarditis.

I look upon simple endocarditis as evidence of destruction of the micrococci by the tissue resistance, and malignant endocarditis as proof of the failure of that resistance. I cannot accept the view that septic micrococci have a predilection for damaged and sclerotic valvular tissues, but I think it highly probable that in the necrotic areas of vegetations micrococci may remain latent and spring into virulence. How is it that in written answers we find examinees insisting upon the multiple abscesses found in the viscera in malignant endocarditis? The great feature is the usual absence of such abscesses, and the term arterial pyaemia is as a rule quite incorrect.

It need hardly be added that I do not suppose rheumatism to be the only cause of malignant endocarditis, which we all admit may be produced by various infections. Lastly, I venture to think that more attention directed to the supervision of patients with organic heart disease would throw light upon the all-important factors tending to cause this most dangerous condition.

It follows from the above considerations that I recognize three cardinal processes in endocarditis: (1) The simple; (2) the sclerosing; and (3) the malignant.

NEPHROTOMY COMBINED WITH CAESAREAN SECTION IN THE TREATMENT OF ECLAMPSIA WITH SUPPRESSION OF URINE.

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THE number of patients suffering from eclampsia appears to have increased recently, this complication having occurred at Queen Charlotte's Hospital one and a half times more often during 1917 than in the year before the war. Hence the question of its treatment, important at any time, is now the greater.

Before discussing whether transperitoneal incision of the capsule of the kidney may be a useful addition to Caesarean section, it is necessary to consider in what type of eclamptic patient Caesarean section should be performed. Stated briefly, the indications for Caesarean section are the occurrence of fits or the onset of severe toxæmic symptoms in a primigravida with an undilated cervix, especially if little urine is being secreted and if generalized oedema and cyanosis are present. The advantage of Caesarean section in such cases is that no rapid method of delivery *per vaginam* is possible except at the cost of local trauma and shock that exceeds that of laparotomy. If the view be taken that such cases are better left undelivered and treated on general lines, we are faced immediately with the undesirability of infusing fluid into the veins or subcutaneous tissues of an oedematous patient who is frequently already bronchitic, and the equally undesirable practice of giving large doses of morphine to a cyanosed patient. One of the few drugs that is still available in such cases is veratrine, and this, unfortunately, frequently fails. After Caesarean section the eclamptic seizures rapidly improve in the great majority of cases, but in some there is difficulty in eliminating the toxin, owing to more or less marked suppression of urine.

It is usual for eclamptic patients to pass only a diminished quantity of urine for some days after delivery, and this diminution is considerable in quite a large number. The occurrence of acute suppression of urine in toxæmic patients immediately after delivery, although uncommon, is well known, and is frequently fatal. The pathological explanation generally accepted is that the suppression of urine is due to the thrombosis of the intertubular vessels, with consequent necrosis of the renal cortex. This theory seems to rest on the publication of very full and careful *post-mortem* records of certain classical cases.

Bradford and Lawrence (*Journal of Pathology and Bacteriology*, 1898, vol. v, p. 195) recorded the case of a multipara who had practically complete suppression of urine after delivery till her death on the seventh day. There were none of the usual symptoms of acute uraemia—vomiting, delirium, coma, dyspnoea, and amaurosis being absent. *Post mortem* the convoluted tubules were found to be necrotic and the intertubular arteries thrombosed.

Herringham and Griffith (*Ibid.*, 1906, vol. xi, p. 237) gave details of a patient who, from the time of delivery till her death nine days later, passed only ten ounces of urine. The ordinary symptoms of uraemia were absent till the end. *Post mortem* the kidneys showed cortical necrosis and thrombosis of the intertubular vessels.

Lloyd (*Lancet*, 1909, vol. i, p. 156) recorded the case of an eclamptic patient from whom only an ounce and a half of urine was collected from the time of delivery till her death on the eleventh day. There were slight twitchings from the seventh day onwards, but no fits or other symptoms of uraemia. The *post-mortem* description of the kidneys is that together they weighed seventeen ounces. Microscopic examination showed coagulation necrosis of the cortex; the large arterioles were not thrombosed, although the intertubular vessels were, and the intertubular spaces infiltrated with leucocytes.

It is of paramount importance to consider whether this explanation is correct in the majority of cases, as if primary renal thrombosis occurs and is the cause of the suppression, it is in the highest degree unlikely that any surgical interference will save the patient's life once the symptoms are established. There are, however, reasons for doubting if this explanation is correct in all—or any—cases. In 1909 Weber¹ criticized this pathology, and pointed out that "there is nothing in the description of the thrombi to indicate that they were formed several

days before the death of the patient," and suggested that the thrombosis was secondary to inflammatory changes affecting the secreting tissues. The existence of inflammation in all the classical cases is shown by the presence of small cell exudation. A second criticism that may be brought forward is that it is common knowledge that a large number of cases recover which, in the early stages, are clinically identical with the three classical cases. Yet it is difficult to conceive a thrombosis occurring that is sufficiently widespread to cause almost complete anuria and yet allow of recovery. A third and more important point is that after ten days of a condition clinically identical with the account given of the cases already quoted, the simple procedure of incising the capsule of the kidney at once caused free secretion of urine. It is to be noted that the symptoms present in such cases are nearly always those of "latent uraemia" resembling the phenomena observed in obstructive suppression of urine when the ureters are suddenly blocked by a moving calculus.

It remains to consider what is the condition of the kidney during life in eclamptic or toxæmic patients with more or less marked suppression of urine. Cases provisionally grouped under the terms "eclampsia" and "toxæmia" include widely divergent clinical types, and so it is hardly to be expected that the state of the kidney during life will be uniform if a sufficiently large series of cases be taken. To ascertain the state of the kidney during an eclamptic attack in patients with a diminished output of urine, I have palpated the kidney directly through the abdominal incision after performing Caesarean section in eclamptic patients. In every case I have found the organ swollen, tense, and, in some cases, as hard as stone. Whatever the pathology of the condition, increased intracapsular pressure certainly has been present in the cases in question, and this is, in my opinion, a very important factor in causing suppression of urine; it may be the only factor of vital importance in some cases of acute inflammation of a previously healthy kidney. If increased pressure inside the fibrous capsule of the kidney be the cause of the suppression, it may act through alterations in the blood flow or by pressure on the tubules leading towards the ureter. Of the two, the latter seems to be the more probable mode of action.

It would not require great pressure on these tubules to cause suppression of the flow of urine, as it has been shown experimentally that if the ureter is ligatured the pressure of urine in the ureter rises to 60 mm. of mercury in a manometer and then remains stationary, showing that at this pressure either secretion ceases or reabsorption commences. The swelling of the kidney within its fibrous capsule and the intertubular infiltration may cause compression of the collecting tubules leading towards the kidney pelvis and thus prevent the urine reaching the ureter. Thus suppression can theoretically go on for a considerable time, even if the secreting epithelium is still functioning. The suppression of urine in these cases, on this hypothesis, would be "obstructive" in nature, and we should expect to get the symptoms actually met with—namely, those of latent uraemia. One of the cases recorded below seems to lend support to the mechanical hypothesis, for, in a case of post-eclamptic suppression, as soon as the kidney capsule was incised, urine commenced to reach the bladder in reasonably large quantities. Also sections from small pieces of the renal cortex demonstrate the existence of distension of the tubules (see Figs. 1 and 2), and the distension is more marked in the case of the patient with the more complete suppression of urine.

If increased intracapsular tension be the cause of the diminished flow of urine, and if the diminution in the quantity of urine passed is sufficient to cause delay in the excretion of the toxin causing the nephritis, the indication is to relieve the hypertension before degeneration of the renal epithelium takes place and the terminal thrombosis of the intertubular vessels occurs. Caesarean section for eclampsia is one of the few conditions in which this can be done easily, as at this time the operator is faced with (1) acute inflammation of a previously healthy kidney, (2) general toxæmia causing that inflammation, and (3) a laparotomy incision giving easy access to the kidney. Since I satisfied myself that the renal tension is very frequently raised it has become a routine to treat suitable cases by nephrotomy after doing Caesarean section. As such suitable cases are uncommon even in a lying-in

hospital, my experience is so far limited to six, but the results as regards immediate free diuresis have been good and uniform, as every patient has passed a large quantity of urine from the time of operation.

The method adopted is as follows: The patient being under ether, Caesarean section is performed through a high abdominal incision instead of the usual one below the umbilicus. One that gives good access is a median incision made so that two-thirds of its length are above the umbilicus and one-third below. The Caesarean operation being finished in the usual way, a large retractor of the Doyen type is inserted and the wound retracted upwards and to the right. The flaccid abdominal wall allows of free retraction. The small intestine being packed over to the left, the peritoneum lateral to the ascending colon is incised for five inches, the colon turned towards the middle line, and the kidney exposed. An incision through the renal capsule is made along its convex border for its whole length, and as it is impossible to limit the incision to the capsule, a superficial portion of the kidney substance is cut through. A small drainage tube is passed through to the skin of the loin, the colon replaced, and the abdomen closed. The only difficulty is that of illumination in so deep a hole in a fat patient; in an easy case the nephrotomy does not add ten minutes to the operation.

The performance of nephrotomy some time after delivery requires separate consideration, as it might be expected that the secreting epithelium would be damaged permanently after, say, a week, and that little improvement would result from simple division of the capsule. That this is not a fact, and that the operation may be followed by the most marked improvement, is shown by the two following cases:

CASE I.

[By permission of Dr. G. F. Blacker.] S. O., who had had one child in 1911, was admitted under Dr. Blacker's care at University College Hospital on March 30th, 1912, being then six months pregnant. For the last week the daily quantity of urine had fallen to 8 oz.; it was solid with albumin, and contained granular casts. Intense general oedema present. Optic discs normal. Labour induced, and a dead oedematous fetus delivered on April 1st. Blood pressure 128 mm. mercury. No improvement occurred, and the oedema became so intense that the swollen upper and lower eyelids met, and no vision was possible. In spite of treatment, only 9½ oz. of urine were passed each day. On April 17th four fits followed by temporary coma occurred. On April 18th nephrotomy was suggested and one kidney capsule was incised (by Mr. Trotter) at 8 p.m.; the kidney did not bulge much through the capsule. In spite of this, 20 oz. of urine were passed the next day and about double that quantity on each subsequent day. The oedema rapidly disappeared and the patient was discharged on May 17th. She walked up to the hospital to see me on July 25th in good general health, and on September 25th was noted as being very well although a little albumin was still present in the urine.

This case was probably one of nephritis aggravated by pregnancy and ending in uraemia; the second is one of true eclamptic toxæmia in which increased kidney tension was noted at the time of the Caesarean section and where, after ten days of almost complete suppression, nephrotomy immediately relieved a condition that appeared to all to be approaching a fatal termination rapidly.

CASE II.

L. B., primigravida, aged 25 years, was examined in the antenatal department at Queen Charlotte's Hospital on March 28th, 1918. There were no pre-eclamptic symptoms and no albuminuria. On April 7th at 3 a.m. she had severe headache and vomited repeatedly; at 6.30 a.m. the first eclamptic fit came on, and eleven more occurred before she was brought to Queen Charlotte's Hospital soon after noon. On admission the report of Miss E. Pfeil, the Resident Medical Officer (from whose notes this abstract is made), was that the patient was convulsed, and consciousness was not regained between the fits. Extreme cyanosis and slight jaundice were present. Blood pressure 160 mm. There were slight oedema of ankles and shifting dullness of the flanks. The physical signs were those of a twenty-four weeks pregnancy, cervix closed, and the fetal heart not heard. On passing a catheter an ounce of dark bloody urine, which became solid on boiling, was obtained. In spite of morphine and an enema ten more fits occurred. At 3 p.m. I removed a dead fetus (six months) by abdominal Caesarean section. There was a considerable amount of ascites. The right kidney was very hard, but owing to the early period of gestation in this case, the incision was too low to allow of nephrotomy without considerably extending the incision upwards, and, as the patient was extremely ill, I did not incise the kidney capsule. Two hours after the end of the operation the patient was conscious, and no more fits occurred.

On April 8th, 9th, and 10th frequent watery evacuations of the bowels were obtained, so it is difficult to state with accuracy if any urine was passed. On April 11th the bowels were not opened for twenty-four hours and all urine was collected with the greatest care; only 2 oz. were obtained. For the ten days (April 7th to 17th) though very large quantities of fluid were drunk, the quantity of urine collected in twenty-four hours was never more than 3½ oz. and frequently only a few drachms. During this time she was slightly drowsy but easily awakened, and answered all questions quite rationally. Delirium, twitchings, vomiting, convulsions, and headache were absent. The temperature averaged 99° and never rose above 100°. After the shock of the fits had subsided the pulse-rate averaged 84. The tongue was fairly clean and the breath not foul. She complained only of pain in the eyes, but Mr. Dawney, who examined the discs on April 16th, found them normal. During the first part of this time the oedema was slight, but on April 17th it had increased; her general condition was getting definitely worse, no improvement in the output of urine had taken place, and a fatal termination within, at most, two days, seemed the only end. At noon, under ether anaesthesia (Miss Russell), I incised the right loin and exposed the kidney. It was dark purple in colour and hard. On incision, the kidney substance bulged slightly through the tense capsule. The capsule was freely opened by a T-shaped cut which penetrated the cortex for about one-third of an inch. A large drainage tube was inserted into the oedematous perinephric tissues and the wound closed rapidly by through-and-through sutures. In the sixteen hours preceding the operation she had passed only 2 oz. of urine; in the next eight hours, besides what came from the loin, 6 oz. were passed by the urethra. On the next day, April 18th, 38 oz. were passed; on April 19th, 37 oz.; on April 20th, 50 oz. During the next week the average amount measured daily was 82 oz. She rapidly improved after the nephrotomy and recovered without further symptoms.

The urine was repeatedly examined by Dr. F. E. Taylor; on April 16th albumin was present up to 0.5 Esbach, but diminished in amount till it was reported as absent on April 23rd and following days. Later a trace reappeared. Neither diacetic acid nor acetone was ever found. The amount of urea on April 13th was 0.5 per cent. and on April 15th 0.7 per cent. Casts were repeatedly searched for, but were absent till the day after the nephrotomy, when blood and leucocytic casts were passed in quantity.

HISTOLOGY.

Case I, Fig. 1. A section of a minute piece of cortex removed from Case I shows considerable degeneration of the tissues. Some tubules are lined by cells that are clear, greatly swollen, and whose nuclei can no longer be distinguished. Many cells are disintegrating, and the tubules in places are entirely filled by granular material. Many

FIG. 1.—Case I: Section of portion of kidney removed from nephritis and pregnancy patient during period of partial suppression of urine. Relative size of dilated tubules obtained from section by tracing. Nephrotomy. Recovery.

tubules are still lined by healthy cells that stain well. Compared with a healthy kidney the tubules show general dilatation, but this is not so advanced as in Case II. The glomeruli show no profound change; the size of the clear space round them is rather greater than normal. There is considerable infiltration of the interstitial tissue. No thrombosis of vessels is to be seen.

Case II, Fig. 2. A small piece of cortex at the angle of the T-shaped incision was removed during the nephrotomy on Case II. Sections show that, as a rule, the cells take the stain well and the nuclei are distinct. Some of the cells are degenerate, but no extensive areas of cloudy swelling or necrosis are present. The connective tissue in places is

FIG. 2.—Case II: Section of portion of kidney removed during almost complete post-eclamptic suppression of urine. Relative size of dilated tubules obtained from section by tracing. Nephrotomy. Recovery.

increased in amount, contains round cells, and is slightly oedematous. The blood vessels show no marked changes, and no thrombosis is present. The glomerular cells are normal; each glomerulus is surrounded by a considerable clear space. The most marked changes are in the tubules. These are everywhere dilated, so that the section has a more open appearance than usual; this is due to the size of the tubular lumina, and not to the state of the interstitial tissue. Some of the tubules contain blood and others granular debris. Some cells have partly or wholly desquamated, but in most tubules the majority of the cells are healthy looking and stain well.

CONCLUSIONS.

Suppression of urine in some cases of pregnancy toxæmia is caused by pressure on the collecting tubules due to increased tension inside the fibrous capsule of the kidney. It can be treated successfully by nephrotomy after symptoms have persisted for many days. As a prophylactic measure nephrotomy may be combined with Caesarean section advantageously in suitable cases.

REFERENCE.

¹ *Lancet*, 1909, vol. i, p. 602.

TINEL'S SIGN IN PERIPHERAL NERVE LESIONS.

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Two aphorisms with regard to peripheral nerve injuries appear to have a wide vogue in England at the present time—namely, (1) that an electrical examination of one kind or another is in itself the best, if not the only criterion of the physiological continuity of a nerve, and (2) that when the reaction of degeneration is found to be present at several examinations conducted within a brief space of time, or when there is much pain in the course or territory of a wounded nerve, it is the duty of the surgeon to explore. The reason commonly given is that "no harm can be done by the operation." But there are two serious objections to early operation—the risk of injuring muscular branches which are intact, and the danger of damaging a nerve trunk in process of natural recovery. Of all the lessons