Time of Operation.

The best time for operating on the patient is during the first stage of labour before the membranes have ruptured, as cervical dilatation ensures free drainage. In most instances, to suit my own convenience, I operate before labour begins. The patient is prepared in the usual way for an abdominal operation, and if a vaginal examination has been made recently a few antiseptic douches should be administered.

Site of the Abdominal and Uterine Wounds.

For many years I have operated on my gynaecological cases either through an incision in the rectus sheath or through a transverse wound. More recently I have adopted the incision through the rectus sheath in cases of Caesarean section, owing to the fact that a weak wound is liable to be obtained by a middle-line incision, as the abdominal wall in this situation is attenuated by the large gravid uterus. It is a matter of importance as to whether the right or left side is chosen. In my opinion the incision ought always to be made on the right side, as the uterus usually lies towards the right, and therefore the left margin of the organ approaches the middle line of the abdomen. Over a year ago I was performing the operation in the country, where the lighting equipment was miserable, and the uterus was exposed through an incision in the left rectus sheath. As soon as the uterine incision was made profuse haemorrhage occurred from the lower angle of the wound, and it was found that some large veins in the left broad ligament had been severed; great difficulty was experienced in arresting the haemorrhage. The left border of the uterus in this case was situated near the middle line, and if the incision had been made on the right side of the abdomen the above unpleasant complication would have been avoided. Two knives are always used by me—one for the abdominal and the other for the uterine wall—and I regard this as an important detail in my technique as it prevents infection of the uterine tissues if the skin be not sterile.

Every surgeon who has had a large experience in dealing with cases of repeated Caesarean section has probably on many occasions been hampered by numerous and dense adhesions in the region of the former scar or scars. The attachment of the uterus to the abdominal wall can often be diagnosed before the abdomen is opened, owing to movements communicated to the uterus being accompanied by dragging on the abdominal wall. If attached in the usual way the operation in these circumstances may be dangerously prolonged, and I have known a patient in the practice of an accomplished obstetric surgeon succumb from shock following a tedious operation of this description. In dealing with these cases I strongly advocate the evacuation of the uterine contents through transverse incisions in the abdominal and uterine walls. By so doing the surgeon can avoid the labyrinth of adhesions, and as the uterus is usually firmly fixed to the parietal peritoneum the organ will remain there in a state of ventro-suspension. Sometimes this attachment of the uterus to the parietes favours incomplete retraction, and on several occasions I have observed fairly profuse *post-partum* haemorrhage in such cases. As a rule I make the transverse incision a short distance above the upper extremity of the old scar; this will usually be above the level of the umbilicus and near the fundus of the uterus. On opening the abdomen the free portion of uterine wall near the fundus is severed transversely, and the child is extracted.

Treatment of the Uterus.

Before opening the uterus I insert four large swabs, so that two are situated laterally and the other two at the upper and lower angles of the abdominal wound. The swabs are not removed until the wound in the uterine wall has been closed. After the uterus has been opened the operator should feel for a leg and drag the limb through the wound. If he discover that he has seized a hand he should at once release it and find a leg, as the child should be delivered as a breech. As the limb is often greasy, the nurse who is attending to the dressings should keep a swab in readiness to pass to the operator to prevent his hand from slipping during the extraction. Immediately the child is delivered the uterus should be drawn through the abdominal wound on to the abdominal wall. There the organ should be turned inside out, so that the placenta and membranes can be stripped from their attachment. For this purpose a large pledget of gauze will be found most useful. In most instances the membranes in the region of the cervix are the most difficult to detach, and it is highly desirable that they should be entirely removed, as

even a small portion overlying the os may prevent the lochia from escaping for several days.

When the extraction of the placenta and membranes has been accomplished the inner wall is thrust back into position in order that the uterine wound may be closed. The method which I now adopt is to begin by inserting three interrupted sutures of silk; one suture is placed in the middle of the wound and the other two midway between the first suture and the upper and lower angles of the wound. Each suture passes through the entire depth of the uterine wall, with the exception of the inner layer. These sutures are firmly secured, and the remainder of the wound is closed with similar sutures of catgut. Finally a continuous suture of catgut is used throughout the entire length of the wound. The three sutures of silk are a safeguard in case the catgut becomes absorbed too rapidly.

Closure of the Abdominal Wound and After-treatment.

As soon as the wound in the uterus is closed the four large swabs are withdrawn and the abdominal incision is closed in layers. For many years I have used anchor sutures to support the wound and to keep the small gauze dressing in position. No other covering is applied to the wound. Before the patient leaves the operating table the surgeon should observe whether blood is escaping from the vagina or not; if it is not, he should grasp the uterus and compress it through the abdominal wall. Dilatation of the cervix may sometimes be necessary to remove an obstructing portion of membrane.

In my surgical practice I have for many years allowed my patients every liberty of movement as soon as they become conscious after the operation. They are encouraged to lie on their side and to sit upright in bed in Fowler's position. By so doing drainage is promoted, flatulence is diminished, convalescence is shortened, and the liability to thrombosis is lessened.

ECLAMPSIA: EVOLUTION AS A CAUSATIVE FACTOR.

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BEFORE proceeding in detail with the subject-matter of this paper I must briefly summarize a paper dealing with the general causation of the toxæmias of pregnancy read by me at the recent South African Medical Congress held at Cape Town (October, 1921).

1. A normal pregnancy is one in which all the changes that take place incidental to that condition are necessary for the purpose of gestation—that is, all the changes brought about must be functional.

2. As with almost all pregnancies there are concomitant functionless changes apart from frequent pathological-like manifestations, it is evident that almost all pregnancies are not quite normal.

3. To explain this anomaly in nature—that such an important physiological process as gestation should almost always be abnormal—I advanced the view that in the process of evolution of the various vital functions of the body the placental form of reproduction was evolved last and comparatively late. While all the other vital functions are now mature and harmoniously correlated to each other, gestation is still in some degree developmentally immature, hence its many physiological imperfections. In consequence of this tardiness in the process of evolution of the placental form of reproduction, the pregnant state approaches in some degree primitive conditions; gestation is, as it were, a physiologic atavism. This is evidenced by the following facts:

A. That in the pregnant woman the secondary feminine characteristics often become less marked; she approaches the masculine type in many respects—that is a recession towards the primitive state, in which secondary sex characteristics were not as yet markedly differentiated. This metamorphosis towards masculinity is shown by the frequent occurrence in pregnancy of:

- (a) Overgrowth of hair on the body, especially on the face.
- (b) Coarsening of the features.
- (c) Thickening of the skin.
- (d) Hypertrophy of some of the bones.
- (e) Enlargement of the hands and feet.
- (f) The stability of calcium metabolism.

B. Another result of this physiologic atavism of pregnancy, and one fraught with even more important and far-reaching

consequences is the "survival" of the state of hyperpituitarism in pregnancy. In this respect also the pregnant woman approaches the condition of primitive man—the Neanderthaler giant—who has been shown to have been in a condition of marked hyperpituitarism.

Hyperpituitarism in Pregnancy.

The pituitary body has been shown to increase greatly in size and in weight during pregnancy. Not only does the anterior lobe increase in bulk, but remarkable histological changes take place in the kind and number of cells. New kinds of cells—the neutrophil cells, the "pregnancy cells" (called by Bell the "emergency secretory cells" of the pituitary body)—now make their appearance in enormous numbers, and, from the way they assume a lobulated or an adenomatous arrangement, suggest a glandular hypertrophy.

This marked glandular hypertrophy must mean also augmentation of the secretory function. We may therefore reasonably assume that the increase in the amount of the "hormone of growth," as Cushing calls the pituitary, is responsible for stimulating the growth and development of the foetus (probably by altering the metabolism of the woman from the usual calcium instability to one of calcium retention); for the expulsion of the foetus (by its infundibulin) when mature; and, by its galactagogue action, provides for the sustenance of the foetus after expulsion. At the same time it produces in the woman certain changes: some functional, such as the growth of the uterus, the increase of the pelvic girdle; some functionless; and some morbid, the relation of which to hyperpituitarism can best be studied clinically.

The analogy between acromegaly—a condition admittedly due to hyperpituitarism—and certain abnormal cases of pregnancy has been pointed out by many authorities.

I have shown (in the paper on toxæmias of pregnancy mentioned above) how practically all the manifold changes—functional, functionless, and morbid—that take place in the pregnant woman are very much like those produced by hyperpituitarism. Here I will endeavour to show the relationship of this hyperpituitarism in pregnancy to eclampsia.

Hyperpituitarism and Eclampsia.

In studying the sequence of the various cardinal symptoms of eclampsia, as well as of the other toxæmias of pregnancy, the following important facts seem to stand out quite clearly—namely, that as the physiological requirements for the process of gestation from the commencement to the end of pregnancy are stimulated by the hormone from the anterior lobe, the pathological changes likewise manifested from the beginning to the end of pregnancy are due to overactivity of that part of the gland. Again, as the physiological requirements for the latter stage of pregnancy are supplied by a stimulus from the posterior lobe—for example, infundibulin for the expulsion of the foetus—the pathological conditions peculiar to the latter stages of pregnancy are similarly the resultants of the overactivity of the posterior lobe, superadded on those morbid changes which are due to the hyperactivity of the anterior lobe.

These facts not only explain why eclampsia is almost always a disease of advanced and the end-period of pregnancy, but they also furnish us with an etiological and physiological basis for the usual clinical divisions of the symptoms of eclampsia into prodromal, pre-eclamptic, and eclamptic; for, as will be seen, the prodromal symptoms are due solely to the overactivity of the anterior lobe, the pre-eclamptic symptoms are the result of the overactivity of both the anterior and posterior lobes, while the eclamptic symptoms are due to oversecretion of the posterior lobe only.

A. Symptoms Due to Anterior Lobe.

Prodromal.—The prodromal symptoms are due to overactivity of the anterior lobe, and are mainly subjective symptoms of the same nature as those met with in early pregnancy and also in early acromegaly; these are:

In Eclampsia:

- (1) Nervous irritability and excitability and a certain degree of nervous unbalancing.
- (2) Vomiting and epigastric pain.
- (3) Headaches.
- (4) Tendency to drowsiness and sleep.
- (5) Cramps and twitchings of muscles.

In Acromegaly:

- (1) Similar symptoms to those recorded under eclampsia (1) are met with in early acromegaly.
- (2) As in eclampsia, vomiting and epigastric pain are also symptoms in some cases of acromegaly.
- (3) Cushing thinks the "pituitary headaches" are due to distension of the glandular envelope. Blair Bell ascribes them to increased secretion, and cites cases where the internal administration of the anterior lobe extract often caused severe headaches.
- (4) Somnolence is a symptom in early acromegaly.
- (5) Cramps and twitchings of muscles (as in eclampsia).

Pre-eclamptic.—The pre-eclamptic symptoms are due to hypertrophy of both parts of the gland. Those due to the anterior lobe are *objective* symptoms, which are caused by the hypertrophy and increase in the size of the anterior lobe producing pressure symptoms on neighbouring centres and nerves; thus we have as a result of pressure on the optic tracts or of the chiasma symptoms of disturbed vision, such as flashes of light, coloured lights, commonly called spangles, photophobia, hemianopsia, amblyopia, amaurosis, and even complete temporary blindness, while pressure on the third nerve is responsible for diplopia and even external strabismus.

B. Symptoms Due to Posterior Lobe.

The pre-eclamptic symptoms caused by overactivity of the posterior lobe are due to its well-known pressor action on the blood vessels and the consequent rise of blood pressure. This furnishes us with the most important and an early danger signal of an impending attack; hence we must constantly watch for a pulse with a high tension and an accentuated second heart sound.

As regards changes in the urine, this is not such a constant feature of eclampsia as raised blood pressure; but generally the urine is diminished, has high specific gravity, contains albumin, blood, and hyaline, epithelial, and granular casts. My explanation for these morbid changes in the kidney is that, as Herring and Schafer have shown that the extract of the posterior lobe has a distinct stimulating effect on the renal secretory cells, one may assume that a prolonged overstimulation with a sudden absorption of a great quantity of the stimulus would produce the opposite effect of depression of the secretory cells of the kidney (there may possibly be also a condition of dyspituitarism). In this connexion Thaon's record of haematuria produced as a late result in animals injected repeatedly with infundibulin is very suggestive.

Eclamptic.—The violent tonic and clonic convulsions in eclampsia are entirely due to an overstimulation of the posterior lobe producing, by its well-known pressor action, contractions and spasms of the blood vessels of the brain, thus causing anaemia and oedema of that organ. Dührssen suggested this spasm of the blood vessels of the brain, kidney, and liver as an explanation for the convulsions in eclampsia. The causative factor producing this spasm of the vessels he thought was an "irritation proceeding from the uterus," but the specific pressor action of the posterior lobe on the arteries being well known the primary cause for this spasm is quite evident. In this connexion one may mention Bell's experiments; he produced twitching of the muscles in animals subjected to repeated injections of infundibulin. Another interesting point to note is that in eclampsia the pupils are widely dilated, as opposed to the contracted pupils in epilepsy. Now Cramer has shown that infundibulin produces dilatation of the pupils in the enucleated eye of the frog.

PATHOLOGICAL ANATOMY.

As the most prominent and fatal symptoms of eclampsia are, according to the view propounded in this paper, due to an excessive secretion of the posterior lobe, we would expect the *post-mortem* findings in eclampsia to show such morbid changes in the various organs as may fairly be ascribed to that cause; especially should we find evidence of irritation in the posterior lobe. That this is the case is evident from the following:

Morbid Changes in the Pituitary.

1. Blair Bell finds in the pars anterior a predominance of basophil cells, colloidal substance in the pars intermedia and in the cleft, and an extensive invasion of the pars nervosa by the cells of the pars intermedia. The same author remarks on his findings: "This invasion of the pars nervosa may lead to an excessive production of infundibulin."

2. Clifford White, who examined carefully *post mortem* all the ductless glands of a patient who died two days after

labour from no evident cause (a case probably of eclampsia without convulsions), finds similar conditions in the pituitary as Bell. In the pars anterior he also finds "large cells whose protoplasm stains feebly with any stain—exhausted secretory cells (?). The pars nervosa has been invaded by the large eosinophil cells from the anterior lobe."

Brain.—*Post-mortem* findings in the brain in eclampsia are "moderate oedema of the convolutions, sometimes with anaemia, sometimes with congestion." These changes may reasonably be brought about, as explained before, by sudden spasm and contractions of the blood vessels of the brain.

Liver.—"The most typical changes *post mortem* are albuminoid degeneration, with haemorrhagic and anaemic necrosis. These focal necroses occur near the small portal vessels." These changes are produced by spasm of vessels as explained above. Hepatic degeneration with marked necrosis of liver cells has been produced in animals experimentally by Carro by continuous injections of infundibulin. This spasmodic necrosis is probably the causative factor also of the condition of acute yellow atrophy of the liver in pregnancy.

Kidney.—"The most common changes in the kidney in a majority of cases are those of an acute nephritis, and this is hard to distinguish from an acute parenchymatous nephritis." The cause of these changes in the cells of the kidney I explained above.

I think I have adduced sufficient evidence to show that eclampsia—in common with all the other so-called toxæmias of pregnancy—is brought about not by some accidental external agent, nor is it the result of some morbid derangement producing poisons or toxins from either the foetus, uterus, or intestine, as most of the theories advanced on the toxæmias of pregnancy assume; but that it is essentially a physiological process *overdone*—due to an excess, rather than a perversion, of an essential product.

Why nature should, in the process of gestation, so often overshoot her mark, and thus defeat her own aim, is due to the comparatively short evolutionary experience she has had of this, her newest experiment in reproduction—the placental form.

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CONTINUOUS TREATMENT IN SURGICAL TUBERCULOSIS.*

BY

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IN recent years so much work has been done with regard to the treatment of tuberculosis of the bones and joints, and in both medical and lay papers so much has been written, that further insistence on principles now firmly established and fully recognized may seem unnecessary. The work of Sir Robert Jones, of Sir Henry Gauvain at Alton, and of Calvé, Calot, and others at Berck-sur-Mer and elsewhere, and the discussion at the Annual Meeting of the British Medical Association in 1921, have shown clearly what can be effected by organized and continuous treatment in this direction. But it is doubtful whether in this part of the country we have followed in the footsteps of our leaders as closely as we might. Perhaps on close examination it may be found that although we have acknowledged our belief in continuity of treatment in surgical tuberculosis, this belief has never been translated into action, and, as we are told, "Faith without works is dead." Those of us who are surgically minded are still too apt to regard an operation as the whole treatment in this disease, instead of a mere incident, and an incident which continuous treatment has made less frequent and of diminishing importance. Others, who, having been impressed by

certain unsatisfactory results following operation, have decided to do without it, have made no serious or continued attempt to establish the conditions necessary to avoid operation. These mistakes appear to have arisen because we do not continually bear in mind that surgical tuberculosis is a disease whose duration is measured in years and not in months, and that during the whole of the active period and for some time after we cannot afford to relax our vigilance for a moment. If we do, we run the risk of lengthening the necessarily long duration of the disease, and of permitting the existence and increasing the severity of deformity. Relaxation of continuous supervision in the early stages leads often to rapid spread of the disease, to abscesses which might have been prevented, to deformity difficult or impossible to correct when the condition becomes obsolete, and to the necessity for serious and often mutilating operations. Those who advocate early operation in these cases insist on careful and continued after-treatment. How much more, then, is it necessary if the condition is too advanced for complete eradication of the tuberculous lesion? When such an operation is apparently successful, how unpleasantly often does the return of disease, or at the very least deformity, show that after-treatment has been too brief! How seldom, in spite of all that has been written, does recovery take place without leaving the patient a cripple!

The necessarily long course of the disease makes continuous in-patient treatment in general hospitals almost impossible, and leads almost inevitably to periods of neglect of, or loss of interest in, treatment. During such intermissions the disease breaks out afresh into activity or deformity increases. Once it is recognized that deformity can be prevented, and when present can be minimized only while disease is active, the present disconnected methods will have to give way to a continuous supervision.

In Bradford there are no fewer than six institutions which deal with surgical tuberculosis. Of these, three act entirely independently of the rest. They are hospitals whose constitution precludes long periods of in-patient treatment. The other three are, in theory, linked, but since no one of them was primarily established to deal with surgical tuberculosis, they suffer from the defects of overlapping and lack of co-ordinated surgical supervision. They are a sanatorium to which, along with phthisis, cases of active surgical tuberculosis can be sent; a cripple school where, amongst other conditions, surgical tuberculosis believed to be obsolete can have the benefits of such treatment as is possible for a finally fixed deformity; and a special hospital where, with other orthopaedic cases from this school, the large percentage of cases of supposed obsolete tubercle which prove to be active can have emergency treatment. The last two institutions contain the germ of an ideal scheme and afford at the present time the only attempt to solve the problem of continuous treatment of surgical tuberculosis in the city.

To get the best results it should be possible for all children suffering from this condition to obtain systematic treatment during the whole period of active disease and for some time afterwards. If we include the infants' and children's clinics and exclude the special hospital fed from the Cripple School we have seven separate "nets," the gaps between which are filled by the medical practitioners of the city. The active cases thus "caught" in many instances escape from the net before they are "cured" (if I may venture to continue the metaphor) and are picked up again if seriously crippled at the medical inspection of school children. They may thence be sent to the Cripple School, but too late to prevent or cure deformity. Hence the weak point in Bradford is the provision for active or acute cases.

The Cripple School has accommodation for about two hundred children, of whom some 40 per cent. are or have been cases of surgical tuberculosis. The cases of active disease can and do have the benefit of recumbent, postural, and other forms of active treatment—extensions, plaster, and ultimately of properly made and fitted celluloid jackets and other appliances. By a wise provision such cases are resident from Monday to Friday in each week, and for the week-end or during periods when active surgical interference is required are transferred by ambulance to the special hospital linked to the school. In suitable cases they spend their week-ends at home. Under these favourable conditions I have been able to keep cases of spinal caries recumbent for two years and to watch them through the critical period of their gradual return to active life. And all this has been possible without interruption of their education. An extension of this scheme to include admittedly active cases is all that is needed to put

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