show that there must be some such point when the new cases exactly balance the recoveries.

Of course, in practice, the number of *Anopheles* and of the bites given by them are sure to vary with rainfall and temperature—that is, with season; but this must be understood to be the case; and the simple equations given do not pretend to contain any more than a first approximation towards the facts. The mathematical method possesses the advantage of showing, not only the elements which contribute to a result, but also how and how much each element does so—a thing which can seldom be expressed in words alone without much circumlocution.

This work, first given in my report on the prevention of malaria in Mauritius,³ was repeated and improved in Sections 26, 27, 28 (pp. 151–164) of my book^{4, 5}. The former was examined mathematically by H. Waite (*Biometrika*, vol. vii, No. 4, October, 1910); but his argument contained a flaw which I pointed out to Professor Karl Pearson, though Waite endorsed my work. Section 28 of my book just mentioned leads to a problem of repeated operations (iteration), but I did not solve it until 1918 (see my paper¹⁰).

A second edition of my book was required early in 1911, and I determined to add a fuller treatment of this mathematical study in a final addendum to the work. This constituted Section 66 of the second edition.⁵ This section consists of thirty-five pages, which contain nineteen sub-headings and fourteen sub-sub-headings. It continued the theme much more exactly, and referred not only to malaria but to similar happenings. The section was called "Theory of Happenings." The problem before me was as follows: Suppose that some kind of event happens in every unit of time to a given proportion of a population, while at the same time the same population is undergoing constant changes due to births, deaths, immigration, and emigration, at one rate for the affected and at another rate for the unaffected, what will be the numbers of the affected and the unaffected portions of the population respectively at the end of the period considered? Also, suppose that some of the affected portion are constantly reverting in unit of time, then again what will be the numbers of the affected and the unaffected groups at the end of a given period covering t units of time?

These problems led to two simultaneous equations in the finite calculus. The equations were solved and integrated by that calculus, and the solutions were examined in detail. As well known, the finite calculus possesses the advantage that the unit of time taken may be large or small. When it is infinitesimal the finite calculus merges into the infinitesimal calculus, as shown in subsection (7, ii) and the following paragraphs. I also dealt with happenings that were repeated in the same individuals, and went on to examine metaxenous (or heteroxenous) diseases—that is, diseases which are common to two kinds of hosts. The rest of the paper deals with many details, and confirms my previous quantitative studies of malaria in the same book, and also gives some calculated constants; then it goes on to deal with the infinitesimal treatment of the subject. The integrations were obtained and are given; but of course I should like to have dealt with the theme even more copiously than I have done. Unfortunately the paper contains many misprints and omissions, and the author himself finds considerable difficulty in understanding parts of it.*

Some years later I thought it advisable to submit the same investigation to the Royal Society in the form of the infinitesimal calculus only. Not being so hard pressed for time, I was able to develop the subject considerably, and to introduce a special section for the kind of happenings which are connected especially with infectious diseases—namely, happenings which do not fall at a constant rate on a population, but one in which every infected person tends to infect a given number of healthy persons in his turn. What would be the respective numbers of the affected and the unaffected groups of the population in this case? Here also I was able to solve and to integrate the differential equations in twenty-six pages containing seven sub-headings and twenty-five sub-sub-headings; but of course both papers require some mathematical knowledge.

* We are preparing corrected "pulls" of Section 66, and will be glad to supply copies on application.

The Royal Society published the paper⁷ in full, and was also kind enough to suggest Dr. Hilda P. Hudson as an expert mathematician to help me. Next year we brought out two succeeding parts of the same paper⁸ containing twenty-eight pages, six sub-headings, and twenty sub-sub-headings. Errata for Part I are corrected at the beginning of Part II.

All the equations must of course be examined from many points of view, and, though we could not consider different diseases in detail, we hoped that our paper would enable more precise studies to be made in the future. Unfortunately Dr. Hudson was called away for war work, and we have not touched the subject again until quite recently. We are now beginning to consolidate and summarize our previous studies and to develop them by means of the finite calculus, the infinitesimal calculus, and by the use of integral equations.

I should add that my paper⁷ demonstrated that the sick-to-healthy communication of infectious diseases and the exhaustion of susceptible persons were enough to explain the rise and fall of the characteristic bell-shaped curves of most epidemics—a point on which some doubt was being thrown at that time.

My bibliography contains references to several works, some of which have been examined by Lotka, but all of which I have not read at present. They seem to reach their conclusions by dropping various constants, which, of course, always renders equations more simple but diminishes their general applicability, epidemics of many diseases being modified by small factors which are often overlooked.

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THE RELATION BETWEEN RECURRENT ALBUMINURIA, CHRONIC NEPHRITIS, AND TOXAEMIA OF PREGNANCY.

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THE renal disorders associated with pregnancy have been described as "chronic nephritis," in which the renal disease is the primary fault, and "pregnancy toxæmia," in which the renal disease is entirely secondary to the toxæmia, and as a rule no overlapping has been allowed between these two groups. It has generally been assumed that a patient with pregnancy toxæmia recovers her full kidney function soon after the toxæmia ceases—that is, at the termination of pregnancy, and it follows, therefore, that chronic nephritis should not occur as a result of pregnancy toxæmia. It has also generally been stated that there is no special liability for albuminuria to recur with subsequent pregnancies.

The biochemical counterpart of the belief that albuminuria occurring during pregnancy is due either to pre-existing renal disease or to an acute toxæmia in a patient with previously healthy kidneys is evidenced by the continued efforts to find some biochemical test to distinguish the "nephritic" from the "pregnancy toxæmia," and of recent years a very large number of such tests have been put forward.

Clinicians are continually faced with the difficulty of fitting cases into one or other of these hard-and-fast classes, and biochemists have consistently admitted the uselessness of their tests in classifying the borderline cases. Is it not possible that the fault lies, not with our clinical observations or chemical tests, but with our arbitrary classification of all cases into "nephritic" and "pregnancy toxæmia" types—two watertight compartments in which no overlapping is allowed? Is it not also possible that the belief that "pregnancy toxæmia" does not give rise to chronic nephritis is also false? Surely we have had sufficient experience of the completely unsatisfactory position to which these beliefs have led us to examine what evidence there may be for changing them.

First with regard to the question of chronic nephritis resulting from pregnancy toxæmia. Recently several observers have published series of cases showing that chronic nephritis does sometimes follow as the result of an attack of pregnancy toxæmia. Caldwell and Lyle¹ found the incidence to be 8 per cent. after eclampsia. Gibson² found definite evidence of chronic nephritis in 5 out of 14 patients who had had eclampsia some years previously; and of 12 patients who had suffered from "pre-eclamptic toxæmia," chronic nephritis was found subsequently in 2. In all these patients, Gibson points out, there was nothing to suggest any renal disease antedating the toxæmia. He quotes Koblanck, who found that 6.5 per cent. of patients had chronic nephritis as a result of pregnancy toxæmia. Harris³ found 3 out of 27 eclamptics had chronic nephritis when examined one year after delivery, and the same observer found that 60 per cent. of patients who had suffered from "pre-eclamptic toxæmia" showed definite signs of chronic nephritis when examined one year afterwards. In none of these cases was there any history of renal disease before the pregnancy. James Young⁴ has recently given the incidence of chronic nephritis as 3 per cent. following eclampsia, and 8 per cent. following "albuminuria."

These series are surely of more value than the unsupported statements that chronic nephritis does not occur, and, in spite of the frequency with which such statements are made, as far as I am aware there is no published series in support of them. It is sometimes argued that the patients who showed signs of chronic nephritis afterwards were really suffering from chronic nephritis before their pregnancy, but in a series which I published recently⁵ care was taken to exclude all cases in which there was any suspicion of pre-existing renal disease. In a group of 37 such patients who had albuminuria with their pregnancy, 14 per cent. were found to have undoubted signs of chronic nephritis when examined at long periods after delivery.

We must therefore admit the possibility of chronic nephritis as a result of pregnancy toxæmia, and it follows that because a patient may be found to have chronic nephritis after a pregnancy with albuminuria she was not necessarily suffering from "nephritic" as opposed to "pregnancy toxæmia" during her pregnancy. She may have been suffering from either, or indeed from both.

The question of the frequency with which albuminuria recurs with subsequent pregnancies has also received attention of late, and here again we are forced to modify views that have held sway for a very long time.

James Young⁶ has published a series in which 31 per cent. recurred, and if macerated foetus, abortion, or accidental hæmorrhage (in the absence of albuminuria) are considered as signs of toxæmia, the incidence of recurrence in his series reached 44 per cent. More recently the same writer⁴ has published a larger series with a recurrence rate of 57 per cent. Included in such series may be a certain number of cases of definite chronic nephritis, which will, of course, show a recurrence rate of 100 per cent. In a series of 28 cases which I published⁵ recurrent albuminuria

was noted in 19. In 21 cases there was every reason to believe that the patients were perfectly healthy before their first pregnancy with albuminuria, and in this group 12 (57 per cent.) had albuminuria with a subsequent pregnancy. All these patients were free from obvious signs of renal disease in the intervals between pregnancies.

As far as I am aware there are no published series in support of the statement so commonly made that albuminuria of pregnancy seldom recurs.

We are therefore justified in modifying two time-honoured misconceptions about pregnancy toxæmia. We must admit, first, that pregnancy toxæmia sometimes causes definite chronic nephritis (probably in something like 10 per cent. of cases); and secondly, that of all patients who suffer from pregnancy toxæmia and who are apparently free from renal disease afterwards, something like 50 per cent. have albuminuria with subsequent pregnancies.

If these clinical facts are admitted, we are in a better position to consider the difficulty which arises when we try to place all cases of albuminuria occurring during pregnancy into one of two pathological groups. That these two groups—the "nephritic" and the "pregnancy toxæmia"—exist is very probable, and the evidence for regarding the kidney damage in the latter group as secondary has been admirably stated and defended by James Young. But the difficulty is that we have four clinical types and only two pathological groups to contain them.

The four clinical types are as follows:

1. Patients with chronic nephritis preceding the pregnancy. These patients, of course, continue with signs of renal deficiency after pregnancy. They form the "nephritic" group.
2. Patients presumably healthy before pregnancy who develop albuminuria during pregnancy, but in whom all signs and symptoms of renal disease disappear after delivery, and do not recur with a subsequent pregnancy.
3. Patients presumably healthy before pregnancy who develop albuminuria during pregnancy, and in whom all signs and symptoms of renal disease disappear after delivery, but recur regularly with subsequent pregnancies.
4. Patients presumably healthy before pregnancy who develop albuminuria during pregnancy, but in whom all the signs and symptoms of renal disease persist permanently after delivery. (These patients are really members of the second group, who, as a result of their first pregnancy toxæmia, develop chronic nephritis, and thereafter become members of the first group. It is not therefore necessary to introduce a new pathological class to explain this group of cases.)

It is the cases in the third group that cause so much difficulty in classification, since although they are apparently healthy in the intervals between their pregnancies, yet they fairly consistently show recurrence of the albuminuria with every succeeding pregnancy. If these cases were rare we might explain their occurrence as the result of chance, but when we realize that they form about half of all the cases of albuminuria, it is quite obvious that something more than chance is operating in favour of recurrence.

There is manifestly some difference between those cases in which the albuminuria recurs with succeeding pregnancies, and those in which subsequent pregnancies are quite normal.

James Young has suggested that this difference is due to some unknown factor, some inherent idiosyncrasy to pregnancy, in the former group. He suggests that these patients are really (as opposed to "apparently") free from all renal defect in the intervals between pregnancies, and looks upon every attack of albuminuria with pregnancy in such patients as a primary toxæmia. If this is the case it is difficult to explain why many of these patients do not start this sequence of abnormal pregnancies until late in their child-bearing life. If the peculiar fault is inborn, one would not expect a series of albuminuric pregnancies to be preceded by a series of normal pregnancies in any particular patient. In the series which I published⁵ it was pointed out that amongst patients with recurrent albuminuria the first abnormal pregnancy occurred after one or more normal pregnancies in 40 per cent. of cases. In many cases, therefore, we have evidence that this tendency to recurrence is not inborn but acquired.

What is the nature of this "predisposition," and how is it acquired?

The first attack of albuminuria, if the patient is previously healthy, we must look upon as being due to "pregnancy toxæmia," regarding the kidney damage as purely secondary. If the patient is fortunate this secondary kidney damage recovers completely, and the patient then embarks on a second pregnancy. For this patient to get a second attack of albuminuria she must be extremely unlucky (since the chance of a 3 per cent. risk of toxæmia sufficiently severe to cause symptoms occurring on two occasions is less than one in a thousand). Yet we know that half of the women who apparently recover completely from their first attack of albuminuria with pregnancy actually do have recurrence. Both these types—those who have a recurrence and those who do not—seem to start off in the same way, and in their first albuminuria are indistinguishable from one another. We have seen that the assumption of an inherent idiosyncrasy on the part of one patient causes certain difficulties in explaining recurrent albuminuria occurring after previous normal pregnancies, and, furthermore, since both these types of patient are identical in their behaviour during their first albuminuria, it is difficult to see that one can have an idiosyncrasy that is not shared by the other.

Is it not better to assume that both these patients are previously healthy women—that both have been unfortunate in striking a 3 per cent. chance of "toxæmia"? So far they are identical, but beyond this each runs a different course. In the one the secondary renal disorder recovers completely, leaving the patient free to have a perfectly normal pregnancy next time. In the other the renal damage does not clear up completely, but persists in an amount insufficient to cause symptoms until the onset of the next pregnancy. In other words, this acquired predisposition to albuminuria is simply a low-grade renal insufficiency. The disease is an "occult nephritis," demonstrable only by the most delicate test of renal function that we possess, and that test is pregnancy.

Further evidence in support of this standpoint is obtained from the following series of cases, which occurred in the practice of the Guy's Hospital Obstetric Department, and I am indebted to the obstetric surgeons at that hospital for permission to publish these figures.

Fifty-one patients were studied during at least two pregnancies, the first being associated with albuminuria. Of these, four were suffering from chronic nephritis before their first pregnancy with albuminuria, and will not be considered further.

This leaves 47 patients, all of whom were presumably perfectly healthy before their first attack of albuminuria. They were considered healthy on the following grounds: medical history, urine examination, cardio-vascular examination, and occasionally on the results of biochemical tests. No patient was considered healthy unless there was positive evidence of the absence of albuminuria, and of other signs, at the beginning of pregnancy.

Of these 47 patients, signs and symptoms of chronic nephritis persisted for a year or longer after labour in 6 cases—that is, definite chronic nephritis developed in 13 per cent. Amongst these 6 cases the average time between when the albuminuria was first noted and when pregnancy was terminated was nine weeks.

This leaves 41 patients, none of whom showed any signs or symptoms of renal disease in the intervals between successive pregnancies. They have been divided up as follows: Twenty-seven patients in whom the first albuminuric pregnancy was terminated either by induction or spontaneously within three weeks of the time that albuminuria was first noted; albuminuria recurred with the next pregnancy in 11, giving a recurrence rate for this class of 40 per cent. Fourteen patients in whom the first albuminuric pregnancy was terminated either by induction or spontaneously at some time later than three weeks after the albuminuria was first noted; albuminuria recurred with the next pregnancy in 10, giving a recurrence rate for this class of 70 per cent.

In this series it is seen that the duration of the albuminuria with the first abnormal pregnancy was strikingly long in those patients who subsequently developed frank chronic nephritis, and the deduction is that a prolonged toxæmia is more likely to cause permanent damage in a previously healthy kidney than is one

that lasts only for a short time. This point was also brought out in the series which I previously published, where it was shown that the average duration of the first albuminuria was greater in those patients who subsequently developed chronic nephritis than in those who did not. It has also been noted in a slightly different way by Harris and by James Young. Both these writers noticed a higher incidence of chronic nephritis following albuminuria without convulsions than following eclampsia, and both accept the explanation that although the "toxæmia" in cases of eclampsia is very severe, it is often only of relatively short duration.

It is by studying the last two classes that we have an insight into the etiology of recurrent albuminuria. All patients in both these classes were healthy before their first albuminuria—all were free from obvious stigmata of renal disease between their pregnancies—yet some were "predisposed" to albuminuria with subsequent pregnancies, while others were not. The important point brought out by these two classes is that it shows that the incidence of this "predisposition" is influenced by the duration of the first "toxæmia."

In other words, the etiology of the "predisposition" to recurrent albuminuria is similar to that of frank chronic nephritis in these cases. This is further evidence that the "predisposition" is in fact merely another term for chronic nephritis. If the predisposition is inherent, or inborn, or independent of the first attack of toxæmia, it is difficult to see how early induction of labour in a first attack can influence the probability of a subsequent attack, yet the figures quoted show that this influence is quite definite.

We can thus regard all first attacks of albuminuria in previously healthy patients as due to a primary toxæmia—any renal insufficiency being purely secondary. Of ten such patients one will suffer such marked kidney damage that she will continue to show signs and symptoms of chronic nephritis so long as she lives. Of the remaining nine patients, all of whom are apparently healthy in the intervals between pregnancies, only four have really recovered their kidney function completely. These four subsequently have normal pregnancies. The other five show a recurrence of the albuminuria fairly consistently with all subsequent pregnancies, because although their kidneys are not sufficiently damaged as the result of their first toxæmia to show signs in the ordinary way, yet, when put to the strain of pregnancy, they show signs of insufficiency. They are, in fact, permanently suffering from "occult nephritis."

As an objection to this view James Young suggests that these patients with recurrent albuminuria conform to the "toxæmic" type clinically, rather than to the "nephritic" type. As far as biochemical observations are concerned the same criticism has been used.

My own experience is that these cases conform accurately to neither type; rather have they some things in common with both types. Thus in cases of recurrent albuminuria it is quite common for the foetus to be macerated and for the albuminuria to take a long time to clear up in the puerperium. These characteristics are rather suggestive of a "nephritic" element. On the other hand, such a patient may actually develop eclampsia, which we accept as evidence of "toxæmia." The chemical estimations in the blood and urine are usually indecisive in such cases.

We can only explain all this conflicting evidence—clinical, etiological, and chemical—by assuming that these patients with recurrent albuminuria are really suffering from both "toxæmic" and "nephritic" albuminuria at the same time. The toxæmic element is supplied every time by the pregnancy (and this may be a "normal" pregnancy in the sense that there is not sufficient toxæmia to cause albuminuria in a patient with healthy kidneys), and the "nephritic" element is supplied every time by the occult nephritis which has resulted from the first "toxæmic" pregnancy.

The objection that it is difficult to see how kidney damage that can pass undetected by all our tests of renal function can yet be a serious factor during pregnancy, is merely another example of the unreliability of our renal function tests; and in the face of evidence there can be

no fundamental objection to regarding pregnancy as the most delicate test of renal function that we possess.

The value of this view of the etiology of albuminuria is twofold. In the first place it gives us a working basis for trying to improve our results. If the recurrent albuminuria is really caused by an occult nephritis due to the first toxæmic pregnancy, plus a subliminal toxæmia due to the present pregnancy, it is by paying more attention to the first toxæmia that we may hope to prevent the occult nephritis, and so avoid at least one important factor tending to cause recurrence subsequently. It has been shown that the length of time the initial toxæmia lasts is of considerable importance in the causation of this occult nephritis; but that this is not the whole question is obvious from the fact that, even when it lasts for only three weeks or less, the recurrence rate is still 40 per cent. We must study carefully these initial attacks of toxæmia to try and discover other factors which tend to cause occult nephritis if we hope further to reduce the incidence of recurrent albuminuria. In the second place this view enables us to explain the difficulty that is so frequently encountered of placing certain cases of albuminuria into one of the two pathological groups, either by clinical or by chemical tests. Many cases belong to both groups at the same time, and we should not be surprised, therefore, when we are unable to class a particular case as either "nephritic" or "toxæmic."

A chronic nephritic who becomes pregnant is suffering from albuminuria which is almost entirely "nephritic" in origin, only a very small element being "toxæmic." At the other extreme is the healthy patient who develops albuminuria during pregnancy. She is suffering from "toxæmia" with no primary "nephritic" element. Between these two extremes come all shades of combinations of the two elements, and many of the recurrent albuminurias can be regarded as a fairly equal mixture of the two pathological processes.

CONCLUSIONS.

Toxæmia of pregnancy can be regarded as the cause of all first attacks of pregnancy albuminuria in previously healthy patients. The renal damage in these cases is purely secondary to the toxæmia.

As a result of this toxæmia a certain proportion (about 10 per cent.) develop frank chronic nephritis, and bear the stigmata of this disease, whether or not they become pregnant again.

A further proportion (about 40 per cent.) recover their kidney function completely after delivery, and subsequently have normal pregnancies without albuminuria.

The remainder (about 50 per cent.) recover their kidney function only so far as to be free from signs of renal disease in the intervals between pregnancies. If they subsequently become pregnant they suffer with recurrent albuminuria.

Recurrent albuminuria may be looked upon as due partly to a subliminal pregnancy toxæmia, and partly to a pre-existing occult nephritis caused by the first toxæmic pregnancy.

Pregnancy itself is the most delicate test of renal function that we possess.

If this view is accepted the difficulties of placing certain cases of albuminuria into one of two pathological groups are easily understood. Many cases of albuminuria of pregnancy are due to a mixture of both the "nephritic" and the "toxæmic" elements.

Although the incidence of chronic nephritis and of recurrent albuminuria can be favourably influenced by earlier induction of labour in the first attack of toxæmia, this measure by no means solves all the difficulties. We have still to find out the other important factors in the first attack of toxæmia which tend to produce permanent renal damage.

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CAUSES OF SPINAL COMPRESSION.

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THE following four cases of spinal compression seem worthy of record in view of their etiology and symptomatology; thus in the third and fourth cases tuberculosis and syphilis gave rise to the condition—a comparatively infrequent occurrence. The clinical histories of the patients are given below.

CASE I.

A boy, aged 12, first began to complain of weakness of the legs and difficulty in walking in March, 1927. This was followed in April by increasing stiffness of the legs, until he was confined to bed and was unable to walk. The patient had also noticed that for eight months he had been slowly losing the sense of feeling in the legs. The previous medical and family histories were uneventful.

On examination (January 7th, 1928) the patient was found to be quite normal mentally, and no gross abnormality was noticed as affecting the cranial nerves, or the motor or sensory systems in regard to the arms. The lower limbs showed a tendency to involuntary flexion when handled; there was weakness, increase of tone, and very marked loss of power in both legs. The knee and tendo Achillis jerks were markedly exaggerated; the plantar responses were extensor and Oppenheim's reflex was present; the superficial abdominal reflexes were absent; and ankle and patella clonus were present. Sensation to light touch was impaired to the level of the fourth rib, and to a three-inch brush to the level of the umbilicus; heat and cold perception was affected over a similar area. The joint and vibration senses were impaired over the lower limbs. The fundi were normal. No changes were noticed in the other systems.

The Wassermann reactions of the blood and cerebro-spinal fluid were negative, as was also an x-ray examination of the spine. The Queckenstedt test showed a complete block, while the fluid removed from the cisterna magna and the lumbar region gave evidence of stasis of the cerebro-spinal fluid when examined chemically.

Lipiodol injected into the cisterna magna came to rest at the level of the second dorsal vertebra, and was crescentic in shape. A laminectomy revealed the presence of an extradural tumour at the level of the third dorsal vertebra. This was removed; the patient made an uneventful recovery, and is now able to walk about in a normal fashion. The tumour on section proved to be an endothelioma.

CASE II.

A woman, aged 53, first began to be ill with a dull aching pain at the lower part of the spine six years ago. Two years ago this pain grew worse, and tended to pass round the lower part of the abdomen, and down the legs to the knees. Six months ago the legs grew weak, and tactile sensation began to diminish. The patient became unable to walk.

On examination on January 24th, 1928, the patient was found to be quite normal mentally; there was no gross abnormality affecting the cranial nerves, and the motor and sensory systems in the arms were not involved. The legs showed loss of power and a slight increase of tone, but no wasting; the knee and tendo Achillis jerks were sluggish, there was no ankle clonus, and the plantar response was extensor on both sides. There was loss of sensation to pain, touch, heat, and cold to the level of the umbilicus. The fundi were normal, and the other systems showed no changes. The blood count was quite normal, and the Wassermann reactions of the blood and the cerebro-spinal fluid were negative. An x-ray examination showed some osteo-arthritis of the dorsal spine. The Queckenstedt test revealed a partial block, while examination of the fluid removed from the cisterna magna and lumbar regions gave evidence of stasis.

Lipiodol injected at the cisterna magna came to rest at the level of the eleventh dorsal vertebra. A laminectomy revealed the presence of an intramedullary tumour at the level of the eleventh dorsal vertebra. Removal was impracticable, so the dura was incised to effect a spinal decompression. The patient is now having x-ray treatment, and shows slight improvement.

CASE III.

A man, aged 27, first complained in May, 1928, of loss of the use of the right leg, followed in two or three days by loss of the use of the left leg. About fourteen days later the paralysis became complete, with total anaesthesia to the level of the umbilicus. The patient had suffered from Pott's disease since the age of 5.