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TWO CLINICAL LECTURES ON ADDISON'S DISEASE,

WITH NOTES OF THREE CASES, IN ONE OF WHICH SUPRARENAL EXTRACT PRODUCED MARKED IMPROVEMENT, AND IN WHICH THE SUPRARENAL BODIES WERE FOUND AFTER DEATH TO BE ABSENT AND REPLACED BY MASSES OF FAT.

Delivered at the Edinburgh Royal Infirmary

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LECTURE I.

A FORTNIGHT ago we had the opportunity of investigating together a very interesting case of Addison's disease, and I propose to-day to redirect your attention to some of the points which the case illustrates, and to describe to you a very remarkable case which has recently come under my notice in private practice. It was a typical case of Addison's disease, in which most marked improvement resulted from the administration of suprarenal extract, and in which, on *post-mortem* examination (death having taken place from an intercurrent attack of influenza) the suprarenal capsules were found to be absent, and replaced by masses of fat. The notes of the first case are as follows:

CASE 1. Typical Addison's Disease (profound Asthenia, Pigmentation of the Skin and Buccal Mucous Membrane): Very Great and Rapid Emaciation: No Vomiting, Abdominal Pain, or Diarrhoea: History of a Severe Blow on the Small of the Back: Death: Both Capsules Extensively Affected with the Typical Fibro-caseous Change: Extensive Deposit of Recent Tubercle in the Peritoneum: Old Tuberculous Lesion at the Apex of the Left Lung.

W. M., a miner, aged 50, was sent to me by Dr. Rorie, of Cowdenbeath, and was examined before the students at the Edinburgh Royal Infirmary on June 30th, 1895. His chief complaints were weakness and loss of flesh. He stated that these symptoms had developed after an attack of influenza which he had had three months before.

On examination, we found that he was markedly emaciated. Indeed, he stated that he had lost no less than 4 st. in weight in the course of nine weeks. The debility and asthenia were extreme. His expression was one of great lassitude. The heart sounds could hardly be heard, and the heart's impulse could not be felt; the pulse was small and extremely soft; the appetite, he said, was poor; the tongue was slightly furred on the dorsum, and red at the edges and tip. He stated that there had been neither diarrhoea nor vomiting, and that he had no cough and was not short of breath.

I examined him very carefully, and, with the exception of the extreme feebleness of the heart's action, was unable to detect any physical signs indicative of localised organic disease. The lungs seemed absolutely healthy. There were no indications of phthisis or tubercle, which from the very rapid loss of flesh and the history of influenza at once, of course, suggested themselves as possible causes of the condition. There was no diabetes—another condition which was carefully excluded. And here let me say that in any case in which marked emaciation and debility are rapidly developed without any obvious cause, the possibility of diabetes mellitus should always be kept in view. With the exception of the loss of appetite there was nothing to suggest disease of the stomach. Malignant disease of the stomach is another condition which should always be thought of when debility and emaciation are rapidly developed without any obvious

cause. Physical examination failed to reveal any evidence of organic disease either in the stomach or peritoneum.

Having failed to detect any evidence of organic disease in the lungs and stomach, and having excluded diabetes mellitus, the next thing which suggested itself to my mind was Addison's disease. Extreme debility and cardiac asthenia are, in my experience, the most constant and characteristic symptoms of Addison's disease. Further, it must be remembered that in some exceptional cases of Addison's disease there is very marked and rapid emaciation. This is a point which I wish particularly to emphasise, for it is not generally recognised; indeed, it is contrary to the teaching of some of the best authorities on Addison's disease. I will refer to it again later.

Thinking, then, that the case might be one of Addison's disease, I next directed attention to the condition of the skin. The face, neck, and backs of the hands and wrists were undoubtedly dark in colour, but not darker than one might expect in a man who had been much exposed to the sun and weather.¹ The skin of the body, especially of the back, also seemed to me to be somewhat darker than it usually is in most healthy people; but if so, it was not sufficiently marked to enable one to be sure that it was pathological. It became necessary, therefore, to try to ascertain whether the skin was actually darker than it had been before the commencement of the illness. The patient was first questioned, but he was unable to give any definite information on the point. I then asked my house-physician to bring in the man's wife, who had come with him to the hospital, and I asked her whether she thought the patient's skin was darker than it had been before the attack of influenza. She answered, "I could not say." I then told her to look carefully at the patient's back and tell me whether she thought it was darker than it was before the commencement of the illness. She said, "I could not say, I never saw his back before." I then said, "That is a very remarkable statement. Do you mean to tell me that you never saw your husband's bare back before?" The supposed wife then said, with some show of indignation, "I am not his wife at all!" It appeared that the house physician had brought in the wrong woman. When the real wife was introduced she told us that she did think that the skin was distinctly darker than it used to be.

I next carefully examined the buccal mucous membrane, and detected a very distinct pigmented patch on the inner side of the left cheek. This discovery at once clinched the diagnosis. The presence of this pigmented patch on the buccal mucous membrane, taken in conjunction with the profound asthenia which was present, showed to my mind without any doubt that the patient was suffering from Addison's disease. I at once, you will remember, committed myself to a positive diagnosis of Addison's disease, and gave directions that the patient should be admitted to the infirmary.

And here an interesting point, illustrative of the extreme asthenia which was present in this case, occurs. We were then, you will remember, seeing patients in the relief wards in Lauriston Lane. After I had left the hospital the patient was directed by the nurse to go across to the main building; his wife had by this time left him to return home by train. In going across to the hospital wards—a distance of about 50 yards—the patient had evidently been overcome by the asthenia, for he was found some little time afterwards lying in the centre path, quite conscious, but so feeble that he was unable to get up. He was helped to the medical wards and admitted under the care of one of my colleagues—for I have unfortunately as yet no male beds of my own. Not-

¹ Dr. Rorie's letter given below shows that the patient had been exposed in this way.

withstanding the most assiduous care and treatment, he died a few days afterwards. My colleague informed me that he refused to take the minced suprarenal gland which was prescribed for him, and that he remained in a prostrate, semi-delirious condition, the head symptoms being highly suggestive of some form of intoxication.

Before describing the *post-mortem* appearances, let me add certain particulars regarding the clinical history which I subsequently learned from Dr. Rorie, who, as I have already told you, recommended the patient to consult me at the hospital, but who had not sent me any particulars of the patient's case nor expressed any opinion as to the nature of the disease from which he was suffering. In answer to my inquiry regarding the early history of the case, Dr. Rorie kindly wrote me on July 7th—after the patient's death—as follows:

I am afraid that I cannot tell you much more concerning the case of Addison's disease about which you wrote, but I have inquired further, and got the following particulars from his wife:

The patient's children are all healthy. He himself is one of a family of eight, having four brothers and three sisters. One sister died three days after himself, of cardiac dropsy. The others are alive and well. There is no history of tubercle in the family.

His wife states that he has been "failing" for three years. About three years ago he received a severe blow over the lumbar region through the slipping of an iron "pinch," and has since complained of tenderness in the left side. At the time of this accident he was laid off work for a week. He has since worked steadily till three months ago, when I was first called to attend him for a sharp attack of influenza. He insisted on returning to work far too soon, and after a day or two returned home, and has not worked since. At that time his face and hands were discoloured, but the bronzing became much more marked after he stayed above ground, as during the late hot weather he lay about outside most of the day. He was in a dull listless state, refusing to go to infirmary or to be treated at home.

As regards the condition of the peritoneum found at the *post-mortem* examination, he has never had any temperature or pain beyond a slight tenderness on palpation. His wife states that twice during the past twelve months he has had a "bloody diarrhoea," with severe pain lasting some three hours, and making him lie down by the roadside. He was a fairly temperate man.

At the *post-mortem* examination which was made by Dr. Muir the diagnosis of Addison's disease was fully confirmed. Both suprarenal capsules were markedly enlarged and extensively diseased; they were as typical examples as one could wish to see of the characteristic fibro-caseous change which is the usual lesion in Addison's disease. In addition to the lesion of the suprarenal capsules, the peritoneum was thickly studded with recent miliary tubercles. There were one or two old fibrous tubercles at the apex of the left lung. The right lung and the other organs were healthy.

The presence of such well-marked tuberculous lesions in the peritoneum is of considerable clinical interest and pathological importance. I did not elicit anything, either in the previous history or in the condition of the patient, to lead me to suspect its presence. I examined the abdomen with great care, thinking that the case might be one of cancer of the stomach; but the patient showed no evidence of suffering or discomfort while the abdomen was being palpated. The absence of pain and tenderness may perhaps have been due to the extreme asthenia and depression which were present.

As I have already stated, the lesion of the suprarenal capsules was typical and characteristic. Now there is every reason to suppose that this fibro-caseous lesion, as it has been termed, is tuberculous in nature. And in this particular case that opinion is, I think, amply confirmed by the existence of the extensive deposit of recent miliary tubercles in the peritoneum and the old tuberculous lesion at the apex of the left lung.

Let me now refer to some of the clinical symptoms:—

The Extreme Asthenia and Feebleness of the Heart's Action.—This is the most constant and important symptom of Addison's disease, although it varies considerably in degree in different cases, and indeed very often notably from time to time in the same case. It is the one symptom which is never altogether absent in well-marked cases of the disease; usually, too, it is the first symptom of which the patient makes complaint. The weakness of the heart's action was in this case very apparent on physical examination. As I have already told you, the heart sounds were so faint that they could hardly be heard, and the heart's impulse could not be felt. As I have stated elsewhere,² in many cases of Addison's disease the heart is markedly atrophied. In more than one

case which I have examined *post mortem* I have been much impressed by the smallness of the heart, and I am disposed to think that this atrophy is a feature in the pathology of the disease which has not, so far, received anything like the attention which it deserves.

The Great Diagnostic Importance of Pigmented Patches on the Buccal Mucous Membrane.—For the purpose of diagnosis the pigmentation of the skin, and especially the pigmentation of the mucous membranes, is by far the most important symptom, or rather sign, of the disease. It must, however, be remembered that discoloration of the skin *per se*, that is to say, without asthenia and other constitutional symptoms, is not sufficient to warrant a diagnosis of Addison's disease. The pigmentation of the skin is in no way peculiar; it is not pathognomonic; it may be due to a variety of other conditions. But the pigmentation of the buccal mucous membrane is of far greater diagnostic importance. A few cases have, indeed, been reported (all, so far as I know, cases of phthisis or tuberculous disease) in which pigmentation of the buccal mucous membrane, identical with that of Addison's disease, was present during life, and in which the capsules were found, to the naked eye at least, to be healthy after death. But such cases are exceedingly rare. When one meets with pigmentation of the buccal mucous membrane in a case in which the constitutional symptoms of Addison's disease are also present, one can have no hesitation whatever in giving a positive diagnosis of Addison's disease, provided that there is no obvious disease in any of the other organs or tissues capable of affording a satisfactory explanation of these symptoms.

In order to warrant a positive diagnosis of Addison's disease three factors are, in fact, necessary, namely (1) the presence of the characteristic constitutional symptoms (the more important are asthenia, vomiting, pain in the abdomen and back, and, in some cases, marked emaciation); (2) pigmentation of the skin, and, still more, pigmentation of the mucous membranes; and (3) the absence of any evidence of local organic visceral disease, other than the lesion of the suprarenal capsules, to account for the condition. When these three factors are present, in other words, in typical cases of the disease, a positive diagnosis may be confidently made. The recognition of typical cases of Addison's disease is, in fact, no more difficult than the recognition of other well-marked clinical entities. It is the atypical and complicated cases in which the diagnosis is difficult or impossible, but I do not propose on the present occasion to consider the diagnosis of the atypical cases. You will find the whole subject very fully discussed in the article on Addison's disease in my *Atlas of Clinical Medicine*, vol. i, p. 49. Let me also direct your attention to the two plates which illustrate the article. They are faithful representations of a case which I had under close observation for several years, drawn by a very able artist, and are the most beautiful illustrations of the disease with which I am acquainted.

Emaciation.—As I have already told you, in most cases of Addison's disease there is no marked loss of flesh; the muscles, indeed, are usually wasted, but the body fat is generally well preserved. The late Dr. Greenhow, who was a great authority on Addison's disease, held the view that extreme emaciation was never developed as the result of Addison's disease. And in one case about which I had occasion to consult him many years ago, he went so far as to say that the fact that the patient was emaciated led him to think that the case was not one of Addison's disease. The circumstances under which I had occasion to elicit this opinion were somewhat amusing, and I may as well detail them to you.

In the year 1877 I reported in the *BRITISH MEDICAL JOURNAL* a remarkable case of lymphosarcoma in which one suprarenal capsule was entirely destroyed, but in which there were no symptoms of Addison's disease. A distinguished teacher of physiology, now an equally distinguished lecturer on the principles and practice of medicine, wrote an interesting criticism of my paper, in which amongst other things he said that it is only the peculiar quasi-tuberculous change in both capsules which produces the symptoms of Addison's disease. In answering this criticism, I was able to cite a case which had come under my own observation, in which pigmentation of the skin and other symptoms suggestive of Addison's disease were associated with disease of one supra-

² *Atlas of Clinical Medicine*, vol. i, p. 62.

renal capsule only, and to quote two cases detailed in Dr. Greenhow's work in which one capsule only was diseased. I further was able to point out (and this was a very curious fact, and for the purposes of my controversy a very useful and amusing one) that one of the two cases recorded by Dr. Greenhow had actually been seen during life and diagnosed as a case of Addison's disease by the very gentleman who criticised my paper. If any of you care to refer to this controversy you will find it in the *BRITISH MEDICAL JOURNAL*, 1877, vol. i, pp. 8, 99, 256, 325, and 384.

My object in referring to this episode is, as I have already told you, that Dr. Greenhow, whose opinion I asked as to the nature of the second case, advanced as his chief argument against that case being one of Addison's disease the fact that the patient was markedly emaciated. Now, while I admit that in most cases of Addison's disease there is no emaciation—at all events, so far as the body fat is concerned—I know, from cases which have come under my own observation, that most marked and rapid emaciation, without any associated visceral disease to account for it, is in some cases developed. I believe that in the great majority of cases of Addison's disease the muscles, including the heart muscle, become atrophied; indeed, as I have already told you, I am disposed to think that the atrophy of the heart muscle is one of the most important clinical and pathological facts connected with the disease. In the present case the patient lost no less than 4 st. in weight in a period of nine weeks. I am disposed to think that this great loss of weight was chiefly due to the disease of the suprarenal capsules; it can hardly, I think, be satisfactorily accounted for by the very recent deposit of tubercle, with practically speaking little or no peritonitis, which was found in the peritoneum after death. But, as I have just told you, the present is not the only case of Addison's disease which I have met with in which profound emaciation has been present. In another case, which came under observation at this clinic some years ago, the patient lost 3 st. in weight during a period of six months. That case, like the present, was one of great interest—and it was the recollection of the facts of that case which led me, after I had excluded some of the conditions which are more common causes of asthenia and emaciation, to look for the symptoms of Addison's disease in the present case.

And I may take this opportunity of pointing out to you that the diagnostician is largely aided in quickly and rapidly arriving at a satisfactory opinion as to the nature of the cases which come before him by a knowledge of the pathological probabilities and possibilities which certain groups of clinical symptoms present. He knows, as the result of his past experience, and I may also add of his past mistakes—and this is a very important aid to knowledge—that certain symptoms when met with in combination, probably point to certain diseased or pathological conditions; and he then proceeds, as you saw me do in this case, to look for these different conditions—to exclude some and to look for additional symptoms and signs indicative of others—until he finally arrives at a definite opinion. Those of you who are in the habit of attending this clinic know that we first elicit the facts of a case, and that I then ask you to suggest a diagnosis. In many cases we have, as you so frequently have heard, a dozen different diseases suggested. We then proceed to discuss the different opinions which have been advanced. Many of them are, as you know, very quickly disposed of. It is unnecessary to say that some of the opinions which are expressed will not hold water. But the most erroneous opinions are often very valuable, for they make you think for yourselves; they necessitate our covering a lot of ground, and contrasting the symptoms and signs in a great variety and number of conditions. You learn far more by making mistakes in this way, provided always that you are told why and how you are wrong, than you would do if I were merely to state my own opinion. You get that in the end. I don't say that it is always right, for all of us sometimes make mistakes. But you get it, and, what is far more important, you always also get my reasons for it. This method of teaching is, in my opinion, most useful; and we often, as you know, also find it not only interesting but amusing. I am no believer in *ex cathedra* teaching. It is for this reason, and because I know how much one learns by one's mistakes, that I encourage you to express your opinions freely, whether they are right or whether they are

wrong. I would almost say that it is better to have a wrong opinion, provided only that you can give some grounds for holding it, than to have no opinion at all.

But this is a digression. I was about to detail to you a case of Addison's disease, in which emaciation was a prominent symptom. The leading particulars of the case are as follows:

CASE II.—*Case of Addison's Disease Characterised by Profound Asthenia and Feebleness of the Heart's Action, Localised Pigmentary Deposits over One or Two Vertebral Spines and on the Buccal Mucous Membrane; Great Emaciation; Diagnosis Verified on Post-mortem Examination; Simple Atrophy of both Suprarenal Capsules.*

A man, aged 36, came to this clinic complaining of progressive weakness and debility of six months' duration. The asthenia had developed gradually, had not been preceded by any illness, and could not be attributed to any obvious cause. The patient stated that, prior to the present illness, he had been robust and healthy. On examination, he was found to be extremely prostrate, distinctly anæmic and much emaciated. He stated that he had lost three st. in weight during the six months that he had been ill, and he was by no means a big made man. The red corpuscles numbered 3,500,000 per cubic millimetre; they were normally shaped, and formed *rouleaux* in the ordinary manner; the white corpuscles were not in excess. The tongue was clean, the appetite poor; the digestion was said to be fairly good. There had been no vomiting, no diarrhoea, no loss of blood, and no pains in the abdomen or back. The most careful examination failed to reveal any evidence of visceral lesion or localised organic disease; but the heart sounds were remarkably feeble, the heart's impulse altogether imperceptible, and the pulse extraordinarily small and weak. The cause of the anæmia, emaciation, and extreme prostration was obscure. I felt myself unable to make a positive diagnosis. I stated this to the students, and directed the patient to go into the side-room to put on his clothes; but as he was walking away from me, and when a good light fell full on his back, I noticed (what I had not observed before) two or three brown discolorations over the spines of some of the dorsal vertebræ. It immediately flashed across my mind that the case was one of Addison's disease.³ I at once called the patient back, and carefully examined the mucous membrane of the mouth. A small brown discoloration was seen to be present on the inner side of the left cheek just opposite the angle of the mouth. The discoloration was quite characteristic, and I immediately committed myself to a positive opinion that the case was one of Addison's disease. The patient died shortly afterwards in the hospital, and at the *post-mortem* examination both suprarenal capsules were found to be reduced to small masses of fibrous tissue. There was no other lesion in the body. The case was one of the rare cases of Addison's disease in which the capsules, instead of being affected with the typical fibro-caseous tuberculous change, had apparently undergone a process of simple cirrhosis and atrophy.

History of Injury.—The fourth point to which I would wish to allude is the fact that the patient received a severe blow on his back about three years before his death (that is to say, according to his wife's statement, shortly before the weakness, which was no doubt the first symptom of Addison's disease, began to develop). This is a point of some interest, for it confirms the view which is generally held, that blows and injury to the back are the exciting cause of the disease in a certain proportion of cases; and this is just what one might expect if the lesion of the capsules is tuberculous. There can be no doubt that local injury favours the production of some tuberculous lesions, such for example as tuberculous tumours of the cerebellum.

I may add, with regard to the treatment of this case, that before the patient's admission to hospital, Dr. Rorie had recommended the administration of suprarenal gland tissue, but the patient had refused to take it.

The cases which I have now related to you present many features of clinical and pathological interest. I have referred to the more important, but I have said little or nothing, you

³ I had previously met, with another case of Addison's disease in which the pigmentation was limited to the skin over some of the vertebral spines. Here again was another illustration of the advantage of previous experience.

will observe, with regard to treatment. The reason of this is that I had the opportunity of seeing each case on one occasion only. I had no opportunity of watching them and of personally carrying any treatment into effect.

REMARKS ON THE SURGICAL TREATMENT OF MALIGNANT DISEASE OF THE TONSIL.

By DAVID NEWMAN, M.D.,
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At the beginning of last year I showed at the Laryngological Society of London two cases of malignant disease of the tonsil treated by operation, and which are now well, more than six years thereafter. In describing these cases reference was made to statistics collected four years ago,¹ but since that time so many new cases have been recorded that it is well worth inquiring into the present position of surgery on the subject. Before doing so a short account of my two last cases may be given.

CASE I. Carcinoma of the Left Tonsil Removed by Incision after Splitting the Left Cheek; no Glands Involved. No Recurrence within Five Months.—The patient, a farmer, aged 69, consulted me on July 24th, 1896, and stated that during the last six weeks he had suffered from pain in the throat, especially in the region of the left tonsil, but sometimes the pain extended to the left ear and to the angle of the jaw on the same side. He had some difficulty in opening his mouth, but no swelling could be detected externally, nor was there any enlargement of the lymphatic glands. There was no dysphagia nor laryngeal trouble, and the patient's general health was remarkably good. On examining the tonsil it was found to be occupied by a firm hard swelling the size of a hazel nut, which was strictly limited to the tonsil itself, and did not extend even to the pillars. The surface, however, was fungating, and partly covered by a mucopurulent discharge. On August 3rd, a portion of the tumour was removed for microscopic examination, and on being demonstrated to be a carcinoma, immediate operation was advised, and was performed at the Sandyford Nursing Home the following day. With the assistance of Dr. E. A. Gibson and my house surgeon, Dr. Galloway, I split the cheek from the angle of the mouth to the ramus of the inferior maxilla. The skin and muscles were first divided, and when all bleeding was checked, the mucous membrane was opened except close to the tonsil, where it was pushed inwards and backwards with the finger; in this way the tonsil was easily freed, and by cutting with blunt pointed scissors the growth was completely removed. The patient recovered rapidly, and returned home on August 18th. Reported well December 20th, 1896.

CASE II. Epithelioma of the Left Tonsil removed by Lateral Pharyngotomy, with Division of the Jaw in Front of its Angle.—D. S., aged 54, was sent to the Royal Infirmary on April 23rd, 1896, by Dr. James Paton, of Greenock, who informed me that the patient was suffering from malignant disease of the tonsil. He said that he enjoyed good health(?) until six weeks previous to admission, when he began to suffer from pain in the throat, which had steadily become worse. While resting the pain was not severe, but during deglutition it was sharp and lacerating; the dull constant pain was referred to the root of the tongue and the faucial region, while a more acute pain extended to the ear. There was some difficulty in swallowing and considerable increase in the saliva, so that the secretion tended to collect, and when accumulated it flowed from the angles of the mouth. The voice was very little altered, only so far as the sounds were impeded by the pain produced by movements of the muscles of the mouth and pharynx. Examination of the pharynx revealed a tumour the size of a pigeon's egg occupying the left tonsil; the surface was very irregular, ulcerated, moderately hard, and showed a few small bleeding points. The growth was almost limited to the tonsil, but on very careful examination it was found to involve the anterior part of the posterior pillar, and extended down to, but did not involve, the base of the tongue. The anterior pillar was free, and there was no enlargement of the lymphatic glands. The larynx was normal. Thoracic and abdominal organs were normal. On April 30th a small portion of the tumour was removed for microscopic inspection, and when, on immediate examination, it was found to be an epithelioma, the tumour was excised at once by performing a lateral pharyngotomy, in which the lower jaw was divided at its angle on the left side. The patient made a good recovery, and is now well (December).

The question of how to gain access to the tonsil has been frequently discussed, and certain advantages have been claimed for each method.

It is not necessary to discuss many of the older and less efficient modes of operating, such as removal by chemical caustics and escharotics, by curetting, the ligature, the *écraseur*, or by electrolysis—as they have, very properly, been given up.

OPERATION THROUGH THE MOUTH.

Within recent years the galvano-cautery has been employed in the removal of malignant tumours of the tonsil *per vias naturales*. A preliminary tracheotomy may be performed or not according to circumstances, but this is a point which will be discussed presently. Two questions require to be considered: (1) Is it possible totally to remove a malignant tumour of the tonsil without an external incision? (2) In

making an external incision, is it advisable to incise the lower jaw or remove a portion of it?

Seven cases of cancerous disease of the tonsil have been recorded in which growths have been completely removed through the mouth without any external incision.

Wolff, Körte, McIntyre, Watson Cheyne, and Watson of New York, have each recorded one case in which this operation has been successful, and I have two cases in which a cure was effected. These cases show at least that it is possible completely to excise a cancerous tonsil without any external incision, but while this is so a large number of cases might be brought forward to prove that such an operation, while apparently successful at the time, completely fails to eradicate the disease. This is not to be wondered at when, for the purpose of avoiding hæmorrhage, the surgeon employs the wire *écraseur* or the galvano-cautery, or sometimes is even contented with removing the tumour piecemeal. From the very nature of the growth, and the great tendency to invasion of surrounding parts, not only must the entire tumour be removed, but also sound tissue in juxtaposition to it. There is no objection to using the electric cautery in place of a knife, so long as one is certain that the incision is being made through normal structures, but anyone will admit that it is much easier to distinguish between healthy and diseased tissues when cutting with a knife or scissors than when dividing with a cautery. The advantage gained from the avoidance of hæmorrhage by use of the cautery is greatly over-estimated. It is a question of risks: on the one hand the danger of hæmorrhage, on the other the likelihood of leaving remnants of malignant disease. Ligature of the external carotid artery has been employed to control bleeding, but if the disease has extended so far as to require such a procedure, then removal of the disease through the mouth is not likely to be efficient. It is only in cases where the disease is very limited that it can be removed *per vias naturales*, as, for example, in my cases operated on in 1890 and 1891,² who are still alive.

LIGATURE OF THE EXTERNAL CAROTID.

When, however, the disease is more extensive, and a very radical removal is demanded, the question of ligature of the external carotid may be considered. Mr. Watson Cheyne³ advises the division of the operation into two stages, on the ground that while giving a bloodless wound it avoids the danger of secondary hæmorrhage from ligature of the artery, and he says: "Failing this division of the operation into two stages, I am inclined, much against my will, to give up the preliminary ligature of the external carotid artery in most cases, and either tie its branches or control the bleeding by temporary compression of the artery during the removal of the tumour."

The advantages of ligature are more evident in cases where in the operation the lower jaw is not divided.

DIVISION OF THE LOWER JAW.

This leads us to the point: Is it necessary to divide the lower jaw in operating for malignant disease of the tonsil, and in what cases should section of the bone be employed? In Case I, where the tumour was small and hard, not larger than a hazel nut, and strictly limited to the tonsil, all that was required in order to have complete control over bleeding points was to split the cheek back to the ramus of the jaw. In Case II the tumour was the size of a pigeon's egg, was found to involve the anterior part of the posterior pillar, and extended down to the base of the tongue, so that the operation was more extensive than in the other case, but while it demanded division of the lower jaw, it did not require the ascending ramus to be removed.

The advantages of division of the jaw are at once manifest. In giving the surgeon more room to operate he has not only greater control over hæmorrhage, but he is also better able to completely eradicate the disease. On the other hand, the disadvantages are that in some cases there has been a difficulty in getting osseous union between the fragments, so that a false joint may result. There is a danger of necrosis of the ends of the bone, also of septic infection from the compound fracture communicating with the mouth. I have noticed, however, that compound fracture of the jaw, whether pro-