50° C. It grows well on ordinary sugar and glucose agar. It does not grow on Sabouraud's maltose agar at any temperature. On agar and glucose agar the growth is a pale yellowish tint. It also grows in broth and on potato. It liquefies gelatin with the production of a greenish tint.

Many sugars are fermented by it.

In raffinose, acid and gas are produced.

In inulin—negative.

In dulcitol—negative.

In maltose, acid and gas are produced.

In mannite, acid and gas are produced.

In litmus milk—alkaline, no coagulation.

In salicin—negative.

In glucose, acid and gas are produced.

In saccharose, acid and gas are produced.

In lactose, acid and gas are produced.

Scales from seborrhoeic scalp have been planted out upon 8 per cent. glucose agar and incubated at 27° C., and this organism has been found in association with others.

Implantations after scarification have been made upon the skin, using the spore of *Malassez* as a control. At the end of fifty-six hours no lesions have appeared in the case of Dr. Benedek's culture, whilst the usual follicular papular lesion has occurred with the implantation of the spore of *Malassez*.

Dr. Benedek's organism appears to be a sporing bacillus, and unrelated to any yeast forms.

Benedek's claims are clearly too fantastic to gain any wide acceptance. He dispenses entirely with seborrhoea and with Darier's keriosis. In his opinion there is no hereditary element in seborrhoea, only an infection with a pathogenic fungus—that is to say, there is no pre-existent soil on which the fungus will thrive, but a soil that is made by the toxins of his fungus.

Further, from the observations made in the laboratory of St. John's Hospital, it would appear that the organism is not a yeast at all. Even if it were an ascomycete, as he claims, there exists no known pathogenic ascomycete, the only possible near relation to a true yeast being the blastomycetes of Gilchrist.

Finally, he states that he regards the spore of *Malassez* as a harmless saprophyte. In all the fungous infections of the epidermis that I have mentioned at the beginning of this paper the proof of the causal relationship of the fungus in each case is not the fact that it can be cultivated, but that it can be easily seen in the scales produced by the disease. In the stained scale of pityriasis capitis or seborrhoeic dermatitis one can always see the spore of *Malassez*, but never any other fungus. This fact, I think, is the strongest possible evidence in favour of its etiological significance.

SUMMARY AND CONCLUSIONS.

From dandruff a fungus can be cultivated which, when implanted on the skin, produces a condition clinically indistinguishable from seborrhoeic dermatitis. It can be recovered from the artificial lesion by culture, and may be seen in a scraping of the lesion. The fungus appears to be a species of the monilia type of fungus, though differing, as demonstrated by MacLeod, from other known pathogenic species of this genus in definite respects. I believe it to be the spore of *Malassez* for the following reasons:

1. It resembles the spore of *Malassez* morphologically.

2. The spore of *Malassez* is always found in dandruff, and is the only fungus that can be seen inside the scales. It may therefore be regarded as the cause of pityriasis capitis, for the same reason that *Microsporon furfur* is accepted as the cause of pityriasis versicolor. Pityriasis capitis or dandruff has been regarded, since Unna, as the starting point of seborrhoeic dermatitis, and the breeding ground of the disease. The spore of *Malassez* is therefore concerned in the production of seborrhoeic dermatitis.

3. The cultivated fungus is capable of accurately reproducing the disease, not merely a crusted or scaly lesion, but one which is primarily follicular, papular, and ultimately squamous, and which, in many cases, cannot be distinguished from those already present.

4. There are marked individual differences in susceptibility to this fungus. Those with seborrhoeic dermatitis, particularly in the more acute forms, being very sensitive to it, whilst others, especially normal subjects, show a minimal response to it.

I will conclude by saying that if, as I think, the observations of MacLeod and myself which I have just related bring the groups of dermatoses that I have discussed into line with one another, the facts only substantiate the view

held by Unna, Sabouraud, and many others, that in essence seborrhoeic eczema is a fungous disease.

BIBLIOGRAPHY.

- Acton and Panja: *Indian Medical Journal*, 1927, 62, p. 603.
Benedek, T.: *Dermatologische Wochenschrift*, April 27th, 1929, p. 611.
Idem: *Arch. Derm. and Syph.*, vol. 156, p. 184.
Idem: *Dermatologische Wochenschrift*, 1929, vol. 88, p. 585.
Castellani, A.: *Arch. Derm. and Syph.*, 1928, vol. 17, p. 194.
Gougerot and Gancéa: *Bull. Soc. Franç. de Derm. et de Syph.*, July 2nd, 1914, p. 338.
Hudelo and Montlaur: *Bull. Soc. Franç. de Derm. et de Syph.*, June 4th, 1914.
Kingery and Thienes: *Arch. Derm. and Syph.*, 1925, vol. 11, p. 186.
MacLeod, J. M. H., and Dowling, G. B.: *Brit. Journ. Derm. and Syph.*, April, 1926, p. 139.
Pellier: *Ann. de Derm. et de Syph.*, 1912, vol. 5, p. 563.
Schamberg, J. F.: *Arch. Pediat.*, 1915, vol. 32, p. 617.
Shelmire, B.: *Arch. Derm. and Syph.*, December 6th, 1925, p. 789.
Templeton, H. J.: *Arch. Derm. and Syph.*, 1926, vol. 14, p. 270.
Whitfield, J. H.: *Brit. Journ. Derm.*, 1908, vol. 20, p. 274.

DIATHERMY EXTIRPATION OF THE PHARYNGEAL TONSILS.

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THERE are many sufferers from chronic septic intoxication, apparently from the tonsils, for whom complete tonsillectomy of the ordinary kind is unsuited. People with cardiac disability, or with exophthalmic goitre, for example, belong to this class, and we must also include patients whose nervous dread of "cutting" renders the operation an ordeal they dare not face. For cases like these surgical diathermy is now being tried, and it bids fair to become the method of choice. Its action can be limited or extended according to desire; the wounds it gives rise to remain aseptic, and there is only a trifling amount of soreness during the separation of the diathermy sloughs. Furthermore, tonsils of all sizes can be extirpated, whether they happen to be small stumps hidden behind the faucial pillar or large hypertrophied tonsils touching each other in the middle line. It is merely a matter of time and patience.

Method.

Tonsils can be diathermized easily and comfortably under local anaesthesia, and, at least until they are reduced to their last remnants, surface anaesthesia is quite sufficient. The surface of the tonsil is painted over once or twice with pure adrenaline solution (1 in 1,000) in order to blanch it, and then with equal quantities of cocaine hydrochloride solution (10 per cent.) and adrenaline solution (1 in 1,000). Towards the end of the treatment, when the bulk of the tonsil has disappeared and we are nearing the capsule, infiltration of the peritonsillar region, and perhaps also of the pillars of the fauces, with 2 per cent. novocain or beta-eucaine solution is advisable in addition to the surface anaesthesia.

For active electrode I usually employ either a short, curved, blunt point, or a short needle, according to the part of the tonsil to be attacked, the indifferent electrode being applied as usual to the arm or back. The blunt active electrode is more useful in the early, and the needle more useful in the later, stages.

At the first séance the blunt electrode is passed into the supratonsillar fossa, the point being inserted as far as the capsule of the tonsil, to which this blind pouch reaches. Then, with a curved vulcanite guard protecting the anterior pillar of the fauces, the current is turned on at zero, and slowly raised in strength until coagulation appears in the tissue around it. The current is then switched off.

Our object in selecting the supratonsillar fossa for the first coagulation is that, as the pathological changes in ordinary peritonsillar abscess show, septic absorption is probably more active here than anywhere else in the tonsil. Further, a large number of crypts open into this cul-de-sac, and these we destroy straight away.

This region, then, having been put out of action, first in the one and then in the other tonsil, the surgeon may or may not proceed further at the first séance. If the patient is nervous and the tonsil small he will probably limit himself on this occasion to a single application of the

diathermy. But if the patient is stout-hearted there is no reason why a little more should not be diathermized.

Subsequent to the operation, as the effect of the anaesthetic wears off, the patient will feel a little soreness, which will continue for a few days. But, particularly if the surgeon is careful to avoid the pillars of the fauces, little or no complaint is made, and the patient is able to submit to the treatment from start to finish without being away from work for a single day.

The first séance is succeeded by others at intervals of about a week or ten days, each time one or two or more areas of about 1 cm. in diameter being coagulated. The length of the course depends, naturally, upon the size of the tonsils and the amount the surgeon feels called upon to coagulate each time. I find the duration of my treatment to be from two to four months. Towards the end of the time there will remain to be disposed of a few isolated mounds or clumps of lymphoid tissue in the recesses of the tonsil bed, and adhering to its capsule and to the faucial pillars. When this stage is reached infiltration anaesthesia may be required, and the needle electrode is then handy for getting at these less accessible remnants.

In general, we avoid too extensive a coagulation at any one séance, chiefly because of the risk of bleeding during separation of a large, deep slough. In view of the possibility of this accident the patient should remain within reasonable reach of the operator throughout, so long as there are any sloughs to come away. Personally, I have not yet seen serious haemorrhage in these cases, but I know that it does occur, and, indeed, circumstances being as they are, an occasional haemorrhage is to be looked for. But there can be no doubt that the risk is minimized if the surgeon contents himself with modest advances and restrains his own and his patient's desire to bring the treatment to an end as early as possible.

By the foregoing method, carried out with care, patience, and perseverance, one can destroy all the tonsil tissue in the pharynx without seriously taxing the patient's fortitude, and with a minimum of scarring.

One interesting observation has been made, and that is, that patients quite frequently report improvement in their remote symptoms—rheumatic pains and so on—immediately after the first applications of the diathermy. The explanation probably is that the diathermy sterilizes the tonsil of bacteria beyond the area coagulated and so cuts off the supply of toxins.

There can be no doubt, in my opinion, that diathermy coagulation will come to be the method of choice for removing tonsils in adults. It is much superior to London paste and the galvano-cautery, and it may even challenge comparison with the ordinary surgical operation, particularly in delicate and nervous adults. It is, of course, unsuitable for children.

ADDISON'S ANAEMIA:

THE OCCASIONAL FAILURE OF LIVER THERAPY.

BY

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It has lately become almost a platitude that a case of anaemia which does not respond to treatment by means of a liver diet should not be diagnosed as Addison's anaemia. It is therefore of interest to consider the following three cases, on clinical grounds diagnosed as Addison's anaemia, which failed to respond to liver therapy. With these cases were simultaneously treated others of the same disease which gave a response to liver in every way comparable with that described by Minot and Murphy. Moreover, the methods of diagnosis and treatment employed in both the successful and unsuccessful cases were identical.

CASE I.

The patient, a single woman aged 58, was admitted on March 20th, 1928, with a twelve months' history of sensory changes with loss of power in the hands and legs. She had a sister suffering from anaemia, and the only important fact about her previous

medical history was that she had been operated on for gall-stones in 1925.

Physical Signs.—The tongue was clean and glazed, but not sore; there was subjective and objective evidence of involvement of the nervous system; achlorhydria was present; the Wassermann reaction was negative. The van den Bergh test gave an indirect positive reaction of 2 units. The blood count was as follows: red blood corpuscles 3,460,000 per c.mm., haemoglobin 77 per cent., colour index 1.26, white blood cells 4,000 per c.mm. Megalocytosis and anisocytosis were present.

Treatment.—On the above evidence a diagnosis was made of subacute combined degeneration of the cord, accompanied by a moderate degree of Addison's anaemia, and treatment with 8 oz. of liver a day and dilute hydrochloric acid was instituted.

Progress.—The treatment was continued for over six weeks, but the blood picture remained unchanged and there was no improvement in the nervous symptoms. The patient was discharged on May 5th, 1928, and stopped taking liver and hydrochloric acid. On December 18th her blood was examined again, and we were astonished to find that the anaemia had spontaneously disappeared and she now had a normal blood count. There was no concurrent amelioration in the nervous symptoms. Further observation on this case was not possible, but an inquiry made in March, 1929, elicited the information that the patient was alive and in much the same condition as when we saw her in 1928.

Commentary.

Clinically this case was one of Addison's anaemia with subacute combined degeneration of the cord, though confirmation of this diagnosis could be obtained only by post-mortem examination. The occurrence of a spontaneous remission afforded proof that the bone marrow was capable of responding to a suitable stimulus, but the conclusion appears inevitable that the liver could not provide such a stimulus in this case.

CASE II.

A man, aged 51, by occupation a miner, was admitted on October 5th, 1928, complaining of progressive weakness for twelve months. The patient had one brother suffering from some variety of anaemia, but otherwise his family history and personal medical history afforded no significant data.

Physical Signs.—The patient showed evidence of intense weakness and wasting; he was drowsy and febrile. There was no evidence, subjective or objective, of involvement of the nervous system; achlorhydria was present; the Wassermann reaction was negative; and the van den Bergh test gave an indirect positive reaction of 5 units. The blood count was as follows: red blood corpuscles 1,150,000 per c.mm., haemoglobin 25 per cent., colour index 1.2, white blood cells 2,400 per c.mm. The urine had a specific gravity of 1022, and contained a trace of albumin. Megalocytosis and anisocytosis were present.

Treatment.—The patient was given 8 oz. of liver each day, with liver extract to supplement the deficiency when he was unable to take the full dose. Dilute hydrochloric acid was given as well.

Progress.—The patient went steadily downhill, became comatose, and died on October 24th. On October 15th the blood count had dropped to 1,000,000 red blood corpuscles per c.mm., with haemoglobin 23 per cent. From this date onwards the quantity of liver extract was doubled, and arsenic in the form of sulphostab was administered as supplementary treatment.

Post-mortem Examination.—The liver, kidneys, and spleen gave a marked iron reaction; the haemolymph glands were prominent, and the bone marrow showed the megaloblastic reaction typical of Addison's anaemia. There was no evidence of any complicating disease.

Commentary.

This is a case in which the diagnosis of Addison's anaemia was confirmed by post-mortem examination, and the only reason that can be advanced to account for the apparent failure of liver therapy is that the anaemia was a severe one. It may be suggested that the bone marrow was so overwhelmed with toxins that it had become too far damaged to respond; on the other hand, many patients with low blood counts are known to respond well to liver therapy.

CASE III.

This patient, a man aged 45, by occupation a collier, was admitted on October 24th, 1928, complaining of progressive weakness for ten months, loss of weight and sore tongue, and gastro-intestinal disturbances.

Physical Signs.—The patient was weak and showed signs of wasting; his tongue was clean and glazed, his spleen palpable, and evidence was obtained, both subjective and objective, of involvement of the nervous system. The Wassermann reaction was negative; the van den Bergh test gave a positive indirect reaction of 5 units. The blood count was as follows: red blood