The amount of blood which was lost was enormous, and one could not but be amazed at its quantity as well as at the large size of the opening in the tube. Over and over again I have operated upon tubes distended to an enormous size by pus, and yet in which no rupture had occurred. Certainly at such an early date the cause of rupture could not be ascribed to over-distension of the tube, and when we looked on the everted, ragged edges of the gap we seemed to find clinical corroboration of the evidence brought forward by Herzog and others. They consider that one of the chief factors in the production of early rupture in tubal gestation is the action of the syncytium, which displays greater phagocytic properties, that is, greater penetrating powers, than it does in normal uterine pregnancy. In the latter instance the muscular coat affords an insurmountable obstacle, while in tubal pregnancy it is hardly present at all. So continuously destructive is the action of the syncytial cells upon the wall of the tube that Werth compares the condition to a malignant process, which in every case requires to be treated as such.

With respect to the amount of bleeding which had taken place, this was due in part to the position of the rupture. The nearer it approaches the uterine end of the tube the greater is the haemorrhage.

As to the diagnosis in these cases, it may not be possible to be absolutely dogmatic. When menstruation has been quite regular and no period has been missed, when there have been no symptoms such as nausea or sickness, and when the breasts are virginal in appearance, the practitioner is liable to be thrown off his guard. Rupture of an ovarian, tubal, or appendical abscess, and acute torsion of the pedicle of an ovarian cyst may cause sudden and severe pain followed by shock. But there is then usually no pallor such as accompanies loss of blood, and there is probably a history of previous ill-health or abdominal swelling. The rupture of a gravid tube at a very early date of pregnancy generally occurs in a woman in a state of good health, while the pallor that follows it in serious cases is strikingly apparent. One need, therefore, have no great hesitation in diagnosing as ruptured extrauterine pregnancy a case presenting signs and symptoms similar to those described, even when the proofs of pregnancy are entirely absent.

The treatment consists, of course, in opening the abdomen and securing the bleeding points as soon as possible. But each case requires separate judgement. I would have operated whenever I saw the patient, had we believed that the bleeding was continuing so actively as to render death imminent; but being convinced that it had ceased, even although only temporarily, I am sure that we adopted the best method of treatment—waiting until the shock had passed away, and operating before the recurrence of another haemorrhage. The result was eminently satisfactory. During the operation ether was administered, the technique was carried through as rapidly as possible, and, before the patient was removed from the table, saline fluid was transfused under one of the breasts. For a day or two the pulse was feeble, but after then no anxiety was felt as to the issue of the case. The wound healed without trouble, and the patient is now going about in perfect health.

ON THE MICRO-ORGANISM ISOLATED FROM ACUTE RHEUMATISM,

AND ITS RELATION TO OTHER MEMBERS OF THE
STREPTOCOCCUS GROUP.*

By E. W. A. WALKER,

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THAT acute rheumatism, or rheumatic fever as it is commonly called, is an infective disease, will hardly be questioned at the present day. It conforms in all respects to the type of an acute infective fever. Its rapid onset, with pyrexia; its fairly definite course, apart from complications; its seasonal variation, its apparent endemicity in certain areas, and the occurrence of well-recognized epidemics, as shown by Newsholme, all tend to the conclusion of an infective origin. Moreover, a certain number

of cases have been recorded, though they are comparatively few, in which there appeared to be evidence of transmission of infection from one individual to another. Doubt has been thrown on the value of the evidence in these cases, which occurred chiefly among blood relations; but that such cases should have been recorded at all is, I think, a somewhat striking fact. The disease is clearly one against which the natural resistance of a large proportion of individuals is high, as compared, for example, with such acute fevers as scarlatina or diphtheria, so that under ordinary circumstances they exhibit no susceptibility to it. And it is also one in which the nature and the situation of the lesions are unfavourable to the liberation and dissemination of an infective agent. If also, as I believe to be the case, the infective agent is one of low vitality outside the living body, it is not surprising that the infectiousness of the disease is practically negligible.

Various bacteria have been described by different observers as the causal agents of this disease. Thus, Achalmé in 1891 described an anaërobic spore-bearing bacillus, which he had isolated from cases of acute rheumatic fever. Beside Achalmé's cases this bacillus was obtained from a number of rheumatic patients by Thiroloix, Coyon, and others, and once by Hewlett of King's College. It is, however, now generally admitted to have been an accidental finding; and it is probably identical with the *Bacillus enteritidis sporogenes* of Klein.

In a certain number of cases also staphylococci have been described, but these have been for the most part isolated cases. In some of them the diagnosis of acute rheumatism appears to have been somewhat uncertain, and others of them were clearly cases of pyæmia; thus, one case recorded by Guttman had multiple abscesses in the kidneys, muscles, and pericardium. It would indeed be very curious, if a staphylococcus were in reality the cause of acute rheumatism, that the connexion of so robust and so readily isolated an organism had not long ago been clearly established. And it would be very odd that rheumatic lesions suppurate with such very extreme rarity as is the case were their causal agent an organism so pre-eminently prone to pus formation. These cases are, therefore, generally agreed to have presented either accidental findings, or terminal infections, or cases of mistaken diagnosis.

Within the last decade a good deal of attention has been directed to the possible relation of streptococci, or a streptococcus, to acute rheumatism.

More than thirty years ago—in 1875—Klebs observed cocci ("monadina") in the cardiac valves in rheumatic fever. And Popoff, in 1887, isolated a micrococcus from the blood of a case of rheumatic fever, and found that on injection into rabbits it caused arthritis, endocarditis, and pericarditis. Following Popoff's observations, Netter, Kraus, G. Lion, and Buday have all found streptococci in rheumatic fever. In 1894 Professor Dana isolated a diplostreptococcus from the meninges of a fatal case of acute rheumatic chorea. And Riva also demonstrated streptococci in acute rheumatism. Triboulet and Coyon described a diplococcus in rheumatic lesions; and Apert and Triboulet isolated the organism from 11 cases of rheumatic fever in 1898. Lastly, in 1899, Wassermann, Westphal, and Malkoff obtained a diplostreptococcus from a fatal case of rheumatic chorea, and found that on injection into rabbits it gave rise with constancy to a condition of acute polyarthritis.

In 1900 the subject was seriously attacked by English workers; and in that year Poynton and Paine announced that they had obtained a diplococcus in eleven successive cases of acute rheumatism—five times in pure culture. They described the organism in some detail, and supported their contention as to its causal relation to the disease by the results of animal inoculations, which had yielded in rabbits acute non-suppurative polyarthritis, endocarditis with vegetations, pericarditis, and on one or two occasions a condition which they regarded as comparable to chorea.

Since their original paper in 1900, Poynton and Paine have continued their investigations with the greatest perseverance, and have now isolated the organism on, I believe, some 29 or 30 separate occasions. Von Leyden and Fritz Meyer also claim to have shown the association of a streptococcus with acute rheumatism.

This, broadly speaking, was the state of the case in favour of the constant presence of a diplostreptococcus and

* A paper read before the Oxford Medical Society.

its causal relation to rheumatic fever, when, in association with my friend Dr. R. M. Beaton, I began to interest myself in its investigation. Subsequently I also undertook, with the assistance of Mr. Ryffel, of Guy's Hospital, to work at the subject from the chemical side. The main outlines of the results which we obtained were published in two Preliminary Notes in the BRITISH MEDICAL JOURNAL, but unfortunately my return to Oxford in 1903 led to a pressure of other work which prevented the preparation of the detailed account of our experiments that I had intended to publish, until it was too late to be of any special interest.

In December, 1903, however, Dr. Vernon Shaw published a detailed account of experiments which he had made with cultures of the organism in question obtained respectively from Kral (Wassermann's organism), from Poynton, and from myself, leading him to the conclusion that the various organisms were *identical*, and were the causal agents of a condition in animals which exhibited the salient features of acute rheumatism of man.

Still more recently, in 1904, Professor Beattie found the same organism in two cases of acute rheumatism, isolating it in pure culture from the second case, and obtaining with it positive results in three rabbits.

Dr. Beaton and I had begun to work at the subject in January, 1901, and had addressed ourselves in the first case to the question whether we could with constancy obtain any micro-organism from rheumatic cases. In the result we isolated a streptococcus from 15 cases, from 10 of which we obtained it in pure culture. Of the other 5 cases, in 2 the original cultures were contaminated with the *Staphylococcus albus* from the skin, and in the other 3, which were *post-mortem* specimens, with a motile bacillus which we took to be the *Bacterium coli* but did not work out in detail. In 8 of the cases the organism was isolated from the *living* subject; in the other 7 it was found in the heart's blood *post mortem*.

Having found the organism in question, we proceeded to examine its cultural and microscopic characters, growing it on a number of different media, and comparing it with cultures of an ordinary streptococcus from a case of septicaemia grown on the same media. It at once appeared that neither the microscopic nor the cultural characters on ordinary media afforded any reliable criteria by which this organism could be distinguished from other members of the streptococcus group. Compared with the streptococcus from septicaemia it grew rather more freely, —especially on gelatine at the room temperature—it formed acid more rapidly and in greater amount, and it clotted milk some days earlier than the latter. But we had no hesitation in saying that, so far as its appearance and cultural characters went, the organism would undoubtedly be classed as an ordinary streptococcus if handed without comment to an expert bacteriologist. We therefore proposed to avoid the term "diplococcus" (which had been somewhat emphasized by Paine and Poynton) as one that might conceivably occasion some misunderstanding, and lead to the organism being unrecognized when met with by other workers, and we referred to it for convenience as the *Micrococcus rheumaticus*. At the same time we noted that, as stated by Poynton, this organism, when growing in the tissues of an animal, tends to lie in pairs, or in quite short chains; and that in cultures on the ordinary media, and especially when only recently isolated from the body, it commonly presents the same arrangement. This, however, can very frequently be noted in the case of other streptococci also, and is therefore of comparatively small importance. On the other hand, we found that when grown in milk, or in fluid media of unusual alkalinity, the micrococcus often extended itself into chains of considerable and, it might be, even of remarkable length.

Up to this point, therefore, we could not *distinguish* the micro-organism from other streptococci. But we had found a streptococcus in fifteen cases of acute rheumatism.

We next proceeded to test its pathogenic action, using three different specimens (one from an acute rheumatism with endocarditis, a second from a chorea dying of heart failure, and the third from the urine of an acute rheumatic chorea), and inoculating altogether 17 rabbits. The three specimens employed had been obtained in pure culture in the original cultivations, and in our experiments they gave the following effects in the rabbits: Fever and acute arthritis in all cases, and in some cases in addition

pericarditis or endocarditis, or both. After large doses death eventually ensued, but never pyaemia. With smaller doses the arthritis and suspected cardiac lesions, if any, recovered. After recovery a fresh inoculation usually caused a recurrence of the condition, and in one rabbit we produced at intervals four successive attacks. It has been quite properly objected by Cole that we did not publish the protocols of our animal experiments, and I have explained to you how that was prevented. But Dr. Shaw, using our organisms, as well as others, obtained polyarthritis, pericarditis, vegetative endocarditis, visceral infarctions, and in two cases an acute iritis; and not only published his protocols in full but also exhibited the specimens at the British Medical Association meeting in Oxford; and a number of them have, I believe, since been placed in the museum of the Royal College of Surgeons.

It appears, therefore, that the organism which we had isolated was capable of producing in animals the morbid lesions of acute rheumatism, just as did Wassermann's and Poynton's organisms; and that it did not, even when administered in doses which produced a fatal septicaemia, ever lead to the formation of pyaemic abscesses, as is frequently the case in ordinary streptococcal septicaemias.

Cole of the Johns Hopkins Hospital, writing in 1904, based a strong criticism of the whole position on experiments in which he had shown that other streptococci from various sources could on injection cause arthritis and endocarditis in rabbits. This was, however, already quite well known; and while we had attached a good deal of weight to the fact that the condition which we produced in rabbits, and still more the condition which we saw in two monkeys inoculated by Shaw, appeared to us to be more like acute rheumatism and less like any ordinary streptococcus septicaemia, as well as to the fact that among all the rabbits inoculated by Paine and Poynton, Shaw, and ourselves, pyaemia had never been noted, even in the fatal cases, yet we had endeavoured to make it clear in our first note that up to this point the micro-organism with which we were dealing could not, in our opinion, be specifically distinguished from other members of the streptococcus group.

At this point, therefore, having found an organism, and having shown that it was capable of producing lesions similar to those of acute rheumatism, Beaton and I endeavoured to attack the question whether the micrococcus was or was not distinguishable in any way from ordinary streptococcal forms.

Now Marmorek had shown that if a fluid medium in which streptococci of human origin had been grown were filtered free from micro-organisms, and again employed as a culture medium, bacteria in general could flourish in the fluid, while human streptococci were entirely unable to do so. This appeared to afford the test for which we were seeking, and on applying it we found at once that the rheumatic micrococcus grew abundantly in the fluid, whilst several other streptococci which we tested refused to grow. Accordingly we stated our opinion that "if the result be confirmed by other workers, the specificity of the *Micrococcus rheumaticus* will be established."

Pursuing the subject in my later note published with Mr. Ryffel, I stated four points of apparent difference which we had observed between this organism and other streptococci that we had been able to examine, and these differences we claimed to be "strongly suggestive of the specific nature of the micro-organism concerned." These were as follows:

1. A greenish-brown discoloration produced in a blood-agar culture, due apparently to a reduction of haemoglobin to haemochromogen.
2. The growth obtained on using Marmorek's test.
3. The faculty of growing in very alkaline media.
4. The production of considerable quantities of acid, and we also noted its very rapid haemolytic action on red corpuscles.

But grave doubt was thrown on the value of Marmorek's test by the work of Meyer and of Aronsohn in 1902; and in 1903 Schottmüller showed that various streptococci can produce the colour change in blood above referred to. The acid production, therefore, remains *alone* at present as a distinctive characteristic of the streptococcus which we obtained from rheumatism. But I am not aware that up to the present the production of the particular acids in

question, in anything approaching the same quantity, has been recorded in the case of any other streptococcus.

Having ascertained that the acids formed belonged to the fatty acid series, Ryffel and I grew the rheumatic micro-organism for definite periods in highly alkaline fluid media of known alkalinity and then determined their volatile fatty acids. We found considerable, and approximately equal, quantities of formic acid and another fatty acid (which afterwards proved to be acetic acid), together with a very small amount of higher fatty acids, which were solid at the room temperature. And the amount of acid thus accounted for corresponded closely to the total change in the reaction of the culture fluid. A litre of culture several times yielded us about $\frac{1}{2}$ gram of formic acid. These analyses I repeated independently and confirmed here in Oxford on my return; and quite recently I have obtained similar results with a culture of Paine and Poynton's organism, which they kindly sent me for comparison (though the amount of acid is less).

I have not examined very many other streptococci for formic acid production, the method being very long and tedious; but those which I have examined (five different strains) only produced very small amounts of the acid. I therefore feel entitled to maintain the statement made by Ryffel and myself that the formic acid production "may constitute a means of differentiation between the rheumatic micro-organism and other members of the streptococcus group." And I hope to be able to pursue the subject to a more definite conclusion.

The method which I am now employing is as follows: The culture is made alkaline and evaporated to dryness by gentle heating. Absolute alcohol is added to the precipitate and subsequently acidified with phosphoric acid.

The acid alcohol extract is poured off (leaving peptone and other substances behind). It is then made alkaline with NaOH and the alcohol distilled off.

The new precipitate is extracted with ether after acidification with H_3PO_4 . The ether extract is shaken up with strong NaOH solution, and the ether driven off by heat.

The remaining solution is acidified with dilute sulphuric acid, and the fatty acids separated by distillation. The total acids are then determined by weighing in the form of sodium salts, and the amount of formic acid by the reduction of corrosive sublimate to calomel.

Following up this line of inquiry, Ryffel and I proceeded next to examine the urine in cases of acute rheumatism. And here again we found the presence of a considerable amount of formic acid. Ryffel's figures showed quantities of formic acid up to 0.2 gram (and in one case 0.25 gram) in a day's urine; while the total fatty acids of the urine in health (among them formic acid) amount to no more than 0.05 gram per day, according to von Jaksch. The effect of treatment with salicylates was to cause a rapid fall in the formic acid excretion. Unfortunately the work was interrupted at this stage, before we had been able to decide the essential question whether other febrile illnesses were or were not also accompanied by a similar excretion of formic acid. But my friend Dr. Hertz has lately undertaken the investigation of this point, and also the examination of the familiar acid sweats of rheumatic fever.

Summarizing the case in favour of the micro-organism in question as the probable cause of acute rheumatism in man, it may be stated that this organism has been isolated in pure culture from subjects of the disease, both during life and on *post-mortem* examination, by a succession of observers on a relatively large number of occasions; that it has been seen microscopically in rheumatic lesions—in the synovial membrane of joints in the cardiac valves, and in the meninges in cases of acute rheumatic chorea; and that it was both seen in a rheumatic nodule, and cultivated from it in pure culture by Paine and Poynton. On injection into animals it produces morbid lesions similar to those of acute rheumatism. It has the appearances and general cultural characters of a streptococcus. But it produces in considerable amount an acid, formic acid, which is not known to be produced in like amount by streptococci from any other source. And this appears to afford a specific distinction.

On the other hand, various objections have been raised against the acceptance of this micro-organism as the cause of rheumatism.

A considerable number of competent observers have

entirely failed to find any micro-organisms in acute rheumatism. And here I must refer especially to the recent careful investigation carried out by Bulloch and Thompson, to which I attach great weight, and which gave entirely negative results in 14 cases examined during life, and about 20 cases examined after death. Cole, Philip, and others also have similarly failed to find any micro-organisms in the cases which they examined.

Accordingly it is suggested that the organism which I have discussed was either a contamination or a terminal and agonal infection, or a secondary infection.

The first suggestion is an obvious and convenient argument, and can only be met by endeavouring to multiply the number of observers, and the number of positive results. The other two are open to a more direct discussion; and they require a very serious consideration.

Against the terminal infection theory is the fact that the organism has been isolated *during life* in a number of cases, and that these patients were not dying at the time of examination.

The third suggestion is that the organism was the occasion of a secondary infection superposed on the rheumatism. And it is pointed out, quite justly, that in the *post-mortem* examination of cases with pericarditis, or endocarditis, or both, the number of positive results is higher than in the cases in which these lesions are absent, the conclusion being that the micro-organisms in question may, perhaps, be the causal agent in these further stages. The position which one takes with regard to this suggestion must, I think, depend entirely on one's own conception of acute rheumatism. If pericarditis, endocarditis, subcutaneous nodules, and chorea occurring in association with rheumatic fever, are all due to a secondary infection with this streptococcus; then I should willingly admit the view in question. At the same time, I must confess that if these conditions be not acute rheumatism, but a further infection with an independent causal agent of their own, I do not know what remains to us of the disease acute rheumatism, except the fever, and perhaps the arthritis—neither of which, according to leading clinical authorities, is necessarily a marked feature of typical acute rheumatism of children.

There remains one further view, namely, that rheumatic fever is an "attenuated" septicæmia due to debilitated pyogenic cocci—a view which is strenuously held by several German workers, and notably by Singer.

But this again is, I think, sufficiently answered by the clinical evidence and by the definite opinion of the most experienced clinicians that acute rheumatism is a definite entity, distinguishable and distinct from ordinary septicæmias; and I find it very hard to picture to myself the way in which an "attenuated" micro-organism is supposed to act when it causes high pyrexia and rapid death in acutely fatal cases, such as undoubtedly occur from time to time.

Accordingly, while I am well aware that the case in favour of the *Micrococcus rheumaticus* is *non-proven*, and appears in some respects, perhaps, less favourable at the moment than it did four or five years ago, neither do I think that the evidence against it is at all conclusive; and for the present, at any rate, I see no reason to abandon positive results in favour of purely negative evidence. Accordingly, I am inclined to claim this micro-organism as a distinct variety of streptococcus.

Andrews, who has lately applied the ingenious tests devised by Gordon, in an exhaustive attempt to define the outlines of a classification of streptococci, found that the only two specimens of *Micrococcus rheumaticus* which he had been able to obtain for examination belonged respectively to the salivary and faecal types in his classification—organisms likely to be found in terminal infections. But this statement cannot be regarded as entirely conclusive, since he does not appear to have tested the haemolytic power of the two strains in question. And he says in his paper that "such cases of salivary and faecal streptococci as I have tested showed no haemolytic power." On the other hand, the *Micrococcus rheumaticus* exhibits very striking haemolytic action in test-tube experiments.

From the clinical standpoint acute rheumatism is a disease *sui generis*, and is, therefore, doubtless due to a single definite infective micro-organism. But before this can be regarded as in any way established probably the whole question must be reinvestigated *ab initio*. First, it

must be shown that the micro-organism can by suitable methods be obtained from a long series of cases, which must be chosen with due regard to their acuteness and their freedom from suspicion of complications; and secondly, it must be clearly demonstrated that the organism itself is a distinct and definite variety of streptococcus.

A CASE OF RETROPHARYNGEAL FIBROMA.

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SIMPLE tumours growing in or behind the walls of the pharynx seem to be of such rarity that I think the following case is worth recording. I have succeeded in finding less than half a dozen in the literature similar to my own, while neither the College of Surgeons Museum nor those of the London hospitals contain a like specimen.

HISTORY.

A lady, aged 31, was sent to me by Dr. Stone, of Northampton, on August 3rd, 1906. Since Easter, 1904, she had been unable to breathe through her nose and had had some difficulty in swallowing. In May, 1906, she had two attacks of epistaxis; she had never been deaf. She did not think that during the last twelve months, at all events, the tumour had increased in size. On looking into her mouth, behind the right side of the posterior pharyngeal wall was a pyriform swelling, firm, regular, and well defined; the mucous membrane over it was stretched and thin but not involved. It reached to just beyond the middle line; it extended upwards to the lower border of the atlas, while its lower limit was determined by the upper border of the fifth cervical vertebra. It bulged forward the soft palate and the right posterior faucial pillar and filled two-thirds of the nasopharynx. A finger could be swept all round it except along its outer border. Grasped with the finger and thumb it was freely movable laterally on the vertebral column, while the mucous membrane could be pinched up over it.

OPERATION.

A six weeks' course of iodide and mercury had no effect upon it, and on October 30th I operated. Chloroform being given, the head was brought well over the edge of the table and Mr. Whitehead's gag was inserted. With the soft palate drawn gently forward with a small pair of tongue forceps, a longitudinal incision was made through the mucous membrane over the tumour, and the latter was then readily enucleated with the finger, assisted by two or three snips of scissors, curved on the flat. The haemorrhage, brisk for an instant, stopped almost immediately, and the mucous membrane was then sutured by two medium catgut sutures mounted on Mr. Arbuthnot Lane's cleft palate needle holder. Her convalescence was uneventful, rectal feeding being enforced for the first forty-eight hours.

The tumour when removed was the size of a hen's egg, and the following is the report of the Clinical Research Association, who examined it microscopically: "This tumour is an angio-fibroma with a more or less definite capsule; the vessels are chiefly capillary, but there are many large venous channels at the periphery. The stroma is richly nucleated in places, but it is forming fibrous tissue, and we do not observe any definite indications of sarcoma in the sections."

The first description of fibrous tumours in this region was published, in so far as I can ascertain, by Mr. Syme¹ in 1856. He mentions having operated on 4 such cases, but all of them originated in the neighbourhood of the faucial pillars rather than in the posterior pharyngeal wall. In 1857 Dr. Busch² recorded 3 cases of retropharyngeal tumour, and his authority is referred to in the only detailed account I could find of them in any text-book—namely, that in v. Bergmann's *System of Practical Surgery*. His first case was that of a man, aged 34, who gave a fourteen years' history of alteration in voice; for eight years he had been conscious of a mass behind the angle of the left lower jaw, and for six months had noticed a tumour in his mouth. This was the size of a goose's egg and well defined. Fearing haemorrhage, Dr. Busch first tied the left external carotid, and then, without an anaesthetic, split the soft palate and enucleated the tumour after incising the mucous membrane. On section, he says, the growth was a sarcoma with a good connective tissue capsule or a soft fibroma, with some portions of cartilage and some fatty degeneration. From the length of the history and the ease with which it was shelled out it would appear to be similar to my own case.

His second case occurred in a man, aged 70, who gave only a year's history. The mucous membrane was ulcerated over the tumour, and although this was enucleated as

readily as the first, and was like it on section; the involvement of the mucous membrane suggests malignancy. The third case he mentions was undoubtedly a sarcoma. Another similar tumour was reported by Dr. Ficano³ in 1895, but unfortunately I have been unable to verify the reference I found to his article.

Other benign growths have been described by Dr. Roe,⁴ who in 1894 removed a fibro-lipoma from the pharyngeal wall, and by Dr. Frederick Taylor,⁵ who in 1876 had a case of a retropharyngeal lipoma, the specimen of which is in the Guy's Hospital museum.

With regard to the origin of these tumours, Dr. Busch describes them as growing from the pharyngeal aponeurosis. They should, therefore, be styled "pharyngeal" rather than "retropharyngeal." I confess that, before reading his article, I myself thought my specimen sprang from the connective tissue of the retropharyngeal space.

The accompanying photograph was taken unfortunately after the tumour had been in formalin for some two months, and had shrunk somewhat.

I saw the patient again in February. There is no sign of anything like a recurrence of the tumour.

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EPITHELIOMA OF THE LARYNX IN A YOUNG MAN.

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This case appears to be worth recording, as it presents several very unusual features.

The patient was a hawker, aged 27. He came to the throat department of St. George's Hospital in June, 1906, complaining of gradually increasing difficulty in swallowing, together with shortness of breath. These symptoms had been noticed for about two months; the dysphagia was not extreme, for he could take a normal quantity of soft food; there was some inspiratory stridor on deep inspiration, but the voice was very little affected, being only slightly hoarse. He was unable to work on account of the dyspnoea which came on with active exertion, but otherwise the general health was fairly good; he denied syphilis and no signs of the disease were to be discovered. The cervical glands were not enlarged, but a slight fullness could be detected about the right ala of the thyroid cartilage.

On examination of the larynx a large rounded swelling was seen to occupy the right lateral wall; internally it filled a large part of the lumen of the larynx and concealed both vocal cords, while externally it bulged into the right pyriform fossa and vallecula. It was situated below the level of the right aryteno-epiglottidean fold, which projected above the mass and appeared in the mirror to bisect it. The diagnosis appeared to me to lie between gumma and perichondritis of the thyroid cartilage. I therefore ordered mercury and potassium iodide, the latter in doses of 30 grains three times a day well diluted with water, and warned him to come to hospital at once should the dyspnoea get worse.

Under this treatment the dysphagia improved, but the tumour did not alter appreciably in size, and the patient reported several attacks of dyspnoea; the last of these was so severe that he was admitted to the hospital on the night of August 28th, and tracheotomy performed. I then gave intramuscular injections of benzoate of mercury on alternate days in doses of $\frac{1}{2}$ to $\frac{1}{4}$ grain, and at the same time I clipped away at several sittings a considerable part of the growth projecting into the larynx; in this way the dyspnoea was relieved so that he would wear a plug continually in his tracheotomy tube, but the main mass of the tumour appeared slightly larger and the swelling in the neck over the thyroid cartilage was more apparent; antisyphilitic treatment was, therefore, omitted. Microscopical examination of the fragments clipped away