in the head and all over, the throwing off of the bed-clothes, the constant crying, as I presumed, from aches and pains, might be symptomatic of rickets, I have looked out for other corroborative symptoms, but have failed to meet with them.

Whenever I meet with cases presenting such symptoms now, I always make particular inquiries as to where the patient is living, and almost invariably find that the house is in a low-lying region of the town, that it has been, in fact, exposed to malaria, but not in the concentrated form which produces ague.

Now, we are not positive as to what malaria is. We do know that heat is required for its development, together with moisture and decaying vegetable matter; and we are apt to consider those patients who have lived in India for a prolonged period, or who have lived in very marshy districts, as being almost the only patients liable to suffer from ague, because ague in its severe form is only met with generally in such patients. I believe that we frequently have cases of this disease to treat which we never recognise at all; patients who suffer from slight chills, heats, and sweats, and who grow thinner, weaker, and paler every day; patients who are supposed to suffer from other complaints, and who perhaps do, but who mainly suffer from mild intermittent fever with its consequences. The malaria to which they have been exposed has been but mild, and the attack of fever is consequently mild. These cases are also most frequently met with in those parts where great alterations are being made, where the soil is turned up in large quantities, and exposed to the air. Many of our best authorities point out the fact that attacks of ague are frequently met with in places where excavations for building or other purposes have been made; and I feel satisfied that I have seen scores of cases of mild intermittent fever in patients who have been exposed to malaria from excavations, and in those who live in houses but recently built. The spleen in these cases is generally more or less enlarged, oftentimes enormously increased in size.

I saw a case a short time ago, in consultation with a medical friend of mine, which drew my attention more particularly to the fact of the frequency of this disease, owing to the weak state to which the patient was reduced, and the difficulty we found in forming a proper diagnosis.

This little boy had been ailing for about two months. He seemed to become paler, thinner, weaker, less inclined for exertion or amusement, every day, and yet nothing tangible could be found to account for all this. The medical man had looked carefully for phthisis, pulmonary or abdominal, for rickets, and other diseases, and could not find anything to account for the gradually increasing weakness. The mother and the child now went away to visit some friends, thinking that the change would do the patient good. In a few days after its arrival, the child was taken suddenly ill, and was in danger, suffering from bronchitis, under the care of the medical man for about three weeks. Towards the end of this time this medical gentleman found that the spleen was enormously enlarged. After a stay of one month, the mother and child returned home. The family medical attendant was now sent for, and was surprised to find the spleen enormously enlarged, extending down as far as the crest of the ilium. When I saw this child I found the spleen enlarged as above stated, firm to the feel, and tense; the edge could be distinctly felt and grasped. The patient was very anæmic, thin, and flabby; quite unable to stand. Loud anæmic murmurs could be heard, but there was not any valvular disease of the heart; no lung-mischief; no tabes mesenterica; nor rickets. The medical man said that cases of ague were rarely seen in the neighbourhood, and he was inclined to think that the enlargement was due to some other cause; but, as the town was almost on a level with the river, as certain fields around and in the town were occasionally submerged in winter, as the small country town visited by the mother and child was also built on land on the same level almost as the river, and as upon inquiry we found a distinct history of pallor, followed by heat and perspiration, daily, we decided to give small doses of quinine with the tincture of iron, to be gradually increased if found suitable. The child rapidly improved under that treatment, and is now fat, lively, and eats well.

In all these cases, as might have been expected, I have found quinine act like a charm. Children, and grown-up persons, too, who had been ailing for weeks, and in some cases months, have, under its influence, rapidly improved, when other remedies did not seem to have the slightest effect.

As these patients invariably are extremely susceptible to the malaria, I have always recommended removal to higher and healthier parts.

In bringing this subject before the members of this Section, I have had two objects in view: first, to lay stress on the fact that, during the last three years since I have been in the habit of looking

for such cases, I have found them, and frequently successfully treated them; secondly, to point out that cases which would otherwise appear very serious, and which would no doubt become serious in due course, if the proper remedy were not prescribed, or removal recommended, become easy of cure, as soon as the cause has been ascertained.

THE PROGNOSIS OF CHRONIC NON-SUPPURATIVE INFLAMMATION OF THE MIDDLE EAR.

Read in the Section of Otology.

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WHILE this paper, from the nature of the subject, can contain little that is novel to the experienced otologist, it may yet serve a good purpose by eliciting in discussion the views held by British aurists upon a question of immense importance. Before attempting to form a prognosis in any given case, it is absolutely essential to have in the mind's eye a clear view of the pathological conditions which may be present in chronic non-suppurative inflammation of the middle ear. Let us then glance at the possible factors of deafness (accompanied or not by tinnitus or vertigo). I think that most of those present will agree with me when I say that in every case of the disease under consideration, one or more of the following conditions are present. 1. Obstruction of the Eustachian tube; 2. Swelling of the tympanic mucous membrane, due to infiltration of lymphoid cells; 3. Accompanying the last-named condition there must, of necessity, be present more or less free exudation. As a result of disease or abuse of treatment, the tympanic membrane may have become flaccid, so that from loss of tension it is unable to fulfil its function. These factors of deafness are all susceptible of treatment. We must now, however, turn to the consideration of two other pathological conditions which are by no means so amenable to remedies, namely: 4. Thickening of the parts within the tympanum, and the formations of adhesions where their existence is detrimental to hearing; 5. Secondary disease of the labyrinth.

Starting from these premisses, let us glance separately at the various data which influence our prognosis. In calling attention to the different points, it must be understood that it is not alleged that a definite conclusion can be arrived at without welding together the information gleaned from various methods of examination. At the same time it is necessary, in a paper like this, to consider the results of each method separately. Having given this explanation, to guard against misapprehension, I shall proceed on the lines I have sketched.

Hereditary tendency to chronic non-suppurative tympanic disease, a syphilitic or an arthritic taint, are all of more or less unfavourable significance. Hereditary deafness is often of that form known as proliferous catarrh, and the same may be said of that produced by syphilis. A gouty or rheumatic tendency I have often found to exist in those who are afflicted with symptoms pointing to a like pathological condition of the tympanum. In the latter case it is probable, judging by analogy, that the joints of the ossicles are the parts most affected.

Tinnitus, slight at first, but gradually increasing in amount and duration, until it is well-nigh constantly a source of extreme annoyance, and paracusis Willisii (hearing better in a noise), are most unpromising symptoms. Both point to the presence of what Roosa has termed proliferous inflammation, a term which embraces many various pathological conditions, all of which are, however, characterised by the presence of adventitious fibrous tissue or even bone, the result of chronic inflammatory changes. The latter symptom we are, in all probability, justified in ascribing to impaction or ankylosis of the stapes.

After obtaining the history of a case, it is my custom to apply the tuning-fork test. Even if the result point to affection of the sound-perceiving apparatus, I do not consider myself justified in abandoning the case on this account alone, if a subsequent examination reveal chronic middle ear-disease. In the first place, it is probable that a change of tension in the labyrinthine fluid, resulting from physical and remediable changes in the middle ear, may cause a temporary loss of function in the auditory nerve. Again, if, in a case of chronic otitis media, we have pathological changes in the internal ear dependent upon tympanic disease, and, provided deafness be not absolute, it follows that treatment directed to the latter may prevent aggravation of the former, and, by improving the

sound-conducting apparatus, enable sonorous vibrations more easily to reach the defective, but not impotent, auditory nerve.

Our next step is ocular inspection of the drum-membrane. It is only in rare cases that changes in the substance of the membrane, sufficient to account for marked impairment in hearing, are met with. Sometimes, it is true, we can see it to be thickened and altered, that good hearing-power is out of the question. Again, it may be atrophied and flaccid, moving with every respiration, or perfectly transparent. Patches of opacity may exist as the remnants of an acute process in years gone by, which has left perfect hearing-power. The irregularity of the malleus, described by von Troeltsch as common in dry catarrh, is by no means always absent in health. The bright spot may be altered in a variety of ways without causing any symptoms, and I believe that its significance is much overrated. There is, however, an appearance which is often met with, and which, I do not doubt, is familiar to all present, although I have not seen it exactly described in works on otology. I allude to a streaky—one might almost say stringy—opacity in a membrane little altered in transparency except where these opaque fibrous-looking lines occur. This condition is common in those insidious cases in which the hearing goes from bad to worse, or at best remains bad.

What, then, are we to consider as distinguishable conditions of the membrane which render the prognosis favourable? So far as my little experience goes, I should say that in cases in which the drumhead is distinctly indrawn, but not fixed in this position by adhesions, we may have fair hopes of effecting improvement, although, of course, a positive prognosis can only be given after using further methods of examination. Again, the appearance of fluid behind the drum-membrane renders the prognosis very hopeful; for exudation can be removed, and its very presence shows that the mucous membrane lining the cavity has not altogether passed from the stage of cellular infiltration to interstitial thickening.

To sum up; the knowledge we gain by inspection of the drum-membrane is in a large proportion of cases only to be utilised in conjunction with other methods of examination. Thus, if it be opaque in certain parts from interstitial thickening, and if there be deafness, we assume that the opacity is a visible sign of thickening in other parts essential for the act of hearing; while, if it be opaque without the presence of deafness, we make no such assumption.

Turning next to the examination of the naso-pharynx, I need hardly allude to adenoid vegetations and nasal polypi, both of which conditions are often causes of curable deafness. Chronic naso-pharyngeal inflammation, however, requires some further remarks. It is universally admitted that, in a large proportion of cases, tympanic disease is the result of extension of a catarrhal process along the Eustachian tubes. In such cases we may, I believe—other things being equal—consider that the condition of the pharyngeal mucous membrane may be taken as approximately representing that of the tympanum. If, then, that be a pharyngitis characterised by unorganised moist swelling and excessive secretion, we may, *a priori*, assume a corresponding condition of the middle ear as part factor of deafness; while, if we find an inflammation of the naso-pharynx which has gone on to the deposit of fibrous tissue, then we are justified in suspecting the existence of thickening or adhesions within the tympanic cavity. The absence of all sign of pharyngeal mischief in a case of chronic non-suppurative middle ear-disease is also an unfavourable prognostic sign.

I shall next call your attention to what I believe to be the most important aid, alike to diagnosis, prognosis, and treatment. I allude, of course, to the air-douche. Auscultation during this process will enable us to decide whether the air passes through a wide or narrow tube, and also whether the latter contains any free exudation. The absence of secretion, as denoted by a dry sound, naturally leads one to fear that the pathological condition has passed from the stage of swelling and excessive secretion to that characterised by the deposit of fibrous tissue. Inspection of the membrane while compressed air is driven through the Eustachian tubes may also furnish valuable information as to the presence of adhesions. The same knowledge may, of course, also be gained by using the pneumatic speculum.

It is of great importance to note accurately the effect of the air-douche upon the hearing-power of the patient. The factors of deafness which are liable to be influenced by its agency are:

1. Obstruction of the Eustachian tube; so much of the deafness as is due to this cause alone being at once completely, though temporarily, banished.

2. Excessive secretion, causing the presence of exudation within

the tympanum. The action of the air-douche will probably scatter the secretion, thus relieving the clogging of the ossicles and membrane. We must remember, however, that this process of excessive secretion must necessarily—except, perhaps, in cases of hydrops *ex vacuo*—be associated with swelling of the mucous membrane, due to hyperæmia and infiltration of lymphoid cells—a condition amenable to treatment.

3. Adhesions delicate enough to be influenced by the current of air, and so situated as to be stretched or ruptured by its action.

4. Perhaps I should here mention another possible factor of deafness, which is only, however, a secondary result of disease or abuse of treatment, namely, a flaccid condition of the drum-membrane. In such cases, temporary improvement—which may sometimes, by suitable treatment, be maintained—is produced by the use of the air-bag.

If inflation of the middle ear produce improvement, however temporary, we are entitled to hold out hope to our patient, because we have a curable lesion to deal with—always remembering that the prospect of benefit is directly proportionate to the duration of such temporary improvement. If, on the other hand, there be, after several injections of air, no change, then we must suspect the existence of an incurable factor of deafness, viz., the presence of adhesions or thickening in situations where they cannot be acted on by the therapeutic means at present recognised as safe and efficient. In arriving at this conclusion, however, we must take into consideration the various other points to which I have alluded, more especially the condition of the naso-pharyngeal mucous membrane, and the phenomena yielded by the Eustachian catheter and auscultating tube. In calling this factor of deafness incurable, I am aware that the way to criticism is opened, for cases of interstitial thickening are just those for which fluid-injections *per tubam* are advocated. While not prepared to deny altogether the efficacy of this plan of treatment, I cannot help thinking that it has been much overrated. How we can hope to cause disappearance of organised tissue by such highly diluted solutions as alone it is safe to inject into the middle ear, I fail to understand. A solution of iodide of potassium, of the strength of ten grains to the ounce, would not be expected, even by the most sanguine believer in its deobstruent action, to cause the disappearance of an urethral stricture. Why, then, should it remove a similar pathological condition from the tympanum? If those who obtain good results from tympanic injections would record a few cases in which repeated injections of air altogether failed to relieve deafness, but in which tympanic injections succeeded, then I think there would be fewer sceptics as to their therapeutic value, while their *modus operandi* would still remain a mystery.

In this paper, inspissated mucus has not been mentioned as a factor of deafness in chronic non-suppurative inflammation of the middle ear, because most of the recorded cases seem to me to have been more or less subacute. It is difficult to conceive, in the first place, why this tendency to drying up should exist in a part whose air-supply must first pass through a portion of the respiratory tract; and, in the second place, how inspissated mucus can tend to remain long in a cavity where all the physical conditions seem in favour of absorption.

I have endeavoured to allude, shortly to the various points to be considered in forming a prognosis. It will, however, be understood that the various factors of deafness which have been discussed are often associated, and the different points of value in prognosis can only be utilised in combination.

CASE OF ACUTE BILIARY CIRRHOSIS,

CLINICALLY SIMULATING ACUTE YELLOW ATROPHY OF LIVER,
FATAL IN TWENTY-FOUR DAYS.

Read in the Section of Pathology.

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JAMES P., aged 33; shoemaker, living in Clifton, married, having two children, was admitted to the Bristol Royal Infirmary on March 20th, 1883, with jaundice.

He stated that he had been quite well up to thirteen days previously. He was attacked in the night with diarrhoea, which was followed by pain across the epigastric, and right and left hypochondriac regions. The pain was severe, but it did not give rise to vomiting. He was not able to go to work on the day after, as the pain still continued; but two days afterwards the pain abated,