epilation dose, about 6 to 7 H. We distribute it over six or seven sittings, which take place at intervals of two to three days. It would appear that unfiltered rays of 6 to 7 H. hardness are more efficient than strongly filtered hard rays.

We may note in passing that Popoff and Teskoff, experimenting on the hastening of the regeneration of wounds, found that $MgCl_2$ was especially efficient, particularly at intervals of one to two days.[3]

Cases where the lesion is situated in folds or angles of the skin are not suitable for this method—for example, at the upper end of the naso-labial folds, near the alae of the nose, in the angle behind the lobes of the ears, and similar situations. After the excision of such a lesion a cavernous wound results, the inner surface of which cannot be reached in its entire extent by the *x* rays. Very extensive lesions are also unsuitable.

Herr Franz Freund recommends the previous *x*-raying of wounds (*loco citato*), because, in his opinion, the beneficial effects of *x* rays on fresh wounds is due to the fact that they hinder inflammation and the development of the exudation of leucocytes. But I cannot see that this proposal would improve my method, because I consider it a somewhat doubtful procedure to operate on an area of the skin which has been *x*-rayed shortly beforehand, as such a skin sometimes reacts to every form of trauma with an *x*-ray dermatitis. Further, I do not consider it reasonable previously to *x*-ray a portion of skin which is to be immediately excised.

Bearing on this point the communication of Richard Sparmanns is noteworthy.[4] He states that in Malays, who have a very strong tendency to excessive hyperplasia of the connective tissue, and who are liable to keloid formation, the prophylactic *x*-raying of the area before the operation, or *x*-raying immediately after the removal of the stitches (that is, where the open wound is not *x*-rayed), is very often useless.

In conclusion, I thank you for the honour which your Association has done me by its kind invitation, and I am very sorry that it is impossible for me personally to be present. I am very grateful to my eminent colleague and old friend, Dr. Lancashire, for delivering this paper in my absence.

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THE CAUSES AND TREATMENT OF PERSISTENT NASAL CATARRH, PARTICULARLY IN CHILDREN.*

BY

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THE term "nasal catarrh" has of late years become a popular expression covering a large variety of pathological processes. It differs from that other popular disease "a cold in the head" in being chronic, although to be sure nasal catarrh may start with a cold in the head, and its course is generally punctuated by recurrent bouts of the same malady. Translating this into medical phraseology we may say that a cold in the head is an acute rhinitis, and nasal catarrh a chronic rhinitis, both being characterized by a discharge of mucus or muco-pus from the nose, and by inflammatory swelling of the turbinals, particularly of the inferior, leading to nasal obstruction. In the acute disease the symptoms are more severe, and the headache, malaise, and muscular languor betoken the presence of toxaemia. It is, besides, an infectious disease; at least in certain circumstances it behaves as such. (Paroxysmal rhinorrhoea is, of course, out of bounds in the present discussion. It is not nasal catarrh, although popularly it goes by that name.)

Rhinitis, whether acute or chronic, is due to an infection of the nasal mucosa. But when we ask by what micro-organism it is induced we are presented with an embarrassment of riches, since almost any of the *pyogenes* may be present, alone or in company. But inasmuch as the pneumococcus is the most constant, it is natural to infer that that organism is probably the pioneer of the invaders. The fact, however, that during the early hours of an acute catarrh the nasal secretion contains but few bacteria of any kind has led to the surmise that the first onslaught is made by an ultramicroscopic organism, and that those usually found present are secondary infections.

From the clinical, as distinct from the bacteriological, point of view, we may divide our cases into two groups: first, those in which the catarrh can be traced to a local cause or focus; and secondly, those in which no such local cause can be detected.

The chief local causes in children are, first and foremost, adenoids, with or without hypertrophied inferior turbinals and tonsils, and, secondly, nasal sinus suppuration. In adults the same local causes exist, although with a different relative frequency, sinus suppuration being more, and adenoids less, common than in children, while, in addition, deviations of the nasal septum lead on to nasal catarrh frequently in adults and seldom in children.

Our discussion refers particularly to nasal catarrh in children, and at once the mind turns to the all-important if well-worn subject of adenoids.

What is the relationship between nasal catarrh and adenoids? Are they both expressions of one and the same infection? Or does the one induce the other? And, if so, which is the primary? Here we find ourselves up against the old but still unsolved problem of the cause of adenoids. In endeavouring to find a way out of this maze of questions all we have to guide us, as far as I know, is clinical observation. Bacteriology is silent, and experimental pathology is not very helpful. But let us see what we can gather from our observation of the disease.

First of all, I have been informed by Dr. Alice Vance Knox, who has a large experience of welfare work, that nasal catarrh is one of the earliest of life's maladies, appearing quite frequently in infants only a fortnight old; and the same observation has been made by R. C. Clarke, who remarks also that the nasal catarrh is always associated in infants with bronchial catarrh. Now it is well known that lymphoid tissue can be demonstrated in the nasopharynx at birth, but it is rarely bulky enough to require removal until months, and more often years, later. This seems to indicate that the catarrhal infection of the nose precedes the pathological hypertrophy of the lymphoid masses.

It will be remembered also how Dr. P. Watson-Williams has suggested that adenoids are due to chronic bacterial infection of the nose. In criticism of this view we sometimes hear it said that lymphoid hypertrophy as a reaction to sepsis is a novel, hitherto unknown phenomenon. But it is by no means novel or unknown. What is the nature of the enlarged cervical glands that accompany chronic hypertrophy of the pharyngeal tonsils? The members of the whole group of enlarged lymphoid aggregations—adenoids, tonsils, and lymphatic glands—are quite obviously related to each other and to some low form of chronic sepsis, located in themselves no doubt. But from what primary source did it emanate if not from the nose? Thus, according to this view, adenoids is merely an incident in the course of chronic rhinitis.

But, attractive though this explanation may be, it does not account for all the phenomena. If chronic nasal sepsis can in this way induce hypertrophy of the nasopharyngeal tonsil, why are not adenoids also produced in adults with chronic nasal catarrh? We cannot answer this question except by assuming that there is no lymphoid tissue to undergo hypertrophy in the nasopharynx of adults—and that is not the case.

In children adenoids sometimes recur after they have been quite efficiently removed. Their cause must therefore be persisting. Again, is it chronic rhinitis? But once more, what is the explanation of their non-recurrence in adults after removal?

* Read in opening a discussion in the Section of Oto-Rhino-Laryngology at the Annual Meeting of the British Medical Association, Manchester, 1929.

As we all know, however, recurrence of adenoids in children after removal is not common, and their removal is usually followed by cure of the nasal catarrh. Probably, however, this does not mean that the adenoids had been responsible for the nasal disease, but merely that both of these disturbances had been initiated by a common infection, which the bodily resistance, heightened by the improved health that follows the operation, is able to throw off completely and permanently. What interests us to-day, however, is the exceptional happening—the persistence of nasal catarrh after removal of adenoids. This disappointment we have all experienced, when, in spite of a complete clearance of the nasopharynx, the little patient still keeps on "catching cold" at frequent intervals.

Nay! Quite often nowadays we see acute suppuration of the middle ear with mastoiditis in children who have had their adenoids removed, and whose nasopharynx is smooth. I have sometimes wondered whether, in such cases, the nasopharynx is not rather too smooth and hard, lined as it is with scar tissue; whether, that is to say, when operating for adenoids, we are not sometimes a little too thorough in our curetting. To substitute scar tissue for mucous membrane is scarcely an improvement upon Nature. This consideration has always seemed to me to be one of the points in favour of the sliding adenotome.

To return to our main theme. When nasal catarrh persists after adenoids, and perhaps also tonsils, have been removed, there are, in children, two further possible causes, which may, of course, be combined—namely, enlarged posterior ends of the inferior turbinals, and nasal sinus suppuration.

With regard to the former, the late Mark Hovell used to insist upon removing "posterior ends" at all adenoid operations. Without going quite as far as that, perhaps, we should nevertheless always examine for them at the operation, and then if they are much enlarged they should certainly be got rid of. If they are only slightly swollen they may be left, as they will probably subside with the general improvement in local conditions. If they do not shrink, however, they maintain the symptoms of nasal catarrh, giving rise to obstruction of a peculiarly variable character.

Sinusitis we have long known to be one of the common causes of nasal catarrh in adults, but it is only within the last few years that its importance in children has come to be realized. It is, of course, much less frequent in childhood—at least it is less frequently discovered. It also runs a more acute course in early life. The sinuses in childhood are relatively small; some of them—the sphenoidals, for example—amounting to little more than tiny cells, while the frontals do not begin to develop until the seventh year. So it is the ethmoids and maxillary antra that are of most importance in children, and they are frequently, if transiently, affected during colds in the head. If the surgeon makes a practice of washing out the maxillary antra in children with mastoid suppuration, as Watson-Williams advises, he will be surprised at the frequency with which he will find pus in the washings. In these conditions a single washing-out is usually sufficient to cure the nasal infection, and the ear infection also benefits.

In the ordinary way, and apart from ear complications, chronic nasal catarrh in children, adenoids and posterior ends having been excluded, should lead to an examination of the ethmoidal region and of the maxillary antra. In so doing we must remember that neither transillumination nor *x*-ray examination are of much value in childhood; negative findings, at all events, should always be doubted. For the antra, proof-puncture under general anaesthesia is alone reliable, the needle being inserted close above and not below the inferior turbinal, as the floor of the antrum is relatively high in early life. If simple lavage fails to cure the antrum suppuration, a nasal antrostomy opening may be made with a small burr or rasp. Operation through the canine fossa, however, is not recommended prior to the eruption of the permanent teeth.

One question in connexion with nasal catarrh and sinusitis in children I should like to hear your opinion upon, and that is: Is it possible that obstinate sinusitis and polypus formation in young adults is a development of nasal trouble, apart from adenoids, that begins in childhood?

The last local abnormality causing nasal catarrh I shall allude to is deviation of the nasal septum, a source of trouble much less common in children than in adults. We do meet with it, however, from time to time, as a result of a traumatism, and the surgeon may have to decide whether to operate at the risk of stunting the growth of the nose. In such a case the general condition of the paient must be the deciding factor. If the nasal obstruction and catarrh are interfering with the child's normal development, the risk should be accepted and the operation performed. I am sure I do not need to remind you that the septum operation in a child entails a peculiarly difficult and delicate manipulation. But if the septal deformity is not obviously interfering with nasal breathing the operation should be postponed until after puberty.

In adults nasal catarrh, with its consequent hypertrophies, is frequently enough the result of septal deviation, and will be materially benefited by the submucous resection and by removal of obstructing redundancies. But the operation should be strictly limited to cases which obviously require it, for I fear that there is a tendency among some rhinologists to perform submucous resection rather too readily.

Such are the local causes of nasal catarrh particularly in children, together with some of the means at our disposal for dealing with them. It may happen, however—indeed it frequently does happen—that, even after all the measures for eradicating the trouble have been adopted, the case fails to respond; the "colds" keep on recurring; and the nasal catarrh, though perhaps milder, does not entirely disappear. Or, to take the other possibility, the patient may manifest no sign of any local abnormality whatever to account for his catarrhal symptoms—apart, perhaps, from the turgescence of his turbinals, which is, of course, more likely to be the effect than the cause of his trouble. And what are we to do then?

The point is that, while a septic focus such as adenoids or an infected sinus—themselves by the way the product frequently of acute catarrh—will favour the persistence or the frequent recurrence of the catarrhal infection, clinical experience teaches us that there are people, both young and old, who, without any discoverable abnormality whatever, are nevertheless peculiarly susceptible to this infection, and what we are interested in is how to reduce or abolish this susceptibility. And the interest is one we share with mankind in general, for what we are about to discuss now is the prevention of that universal plague known as a cold in the head; acute coryza; acute epidemic catarrh.

The first question to be answered is: Is the practically universal belief that acute nasal catarrh is due to cold, to chilling of the body surface, right or wrong?

In the first flush of bacteriological enthusiasm, most of us—I plead guilty myself to the charge—denied that this was anything but a superstition, and we pointed triumphantly to the plain evidence of infection from one person to another. But longer experience, observation, and cogitation have modified our opinion to this extent—that we have regard now to the soil as well as to the seed; and so we no longer contemptuously reject the belief that exposure to cold, particularly in the case of sedentary, indoor people, will predispose to and often actually precipitate an attack.

The process of inception, however, is not quite simple. Apparently what happens is that most of us pass through an acute attack every six months or thereabouts—that is to say, we acquire an immunity that keeps the infection at bay for about six months. If we are able to lead a healthy life and are not exposed to extremes of temperature or fatigue we go the full period. But when we are nearing the limit of our period of resistance, exposure to cold—a wetting, falling asleep in a draught, a bitter east wind—is sufficient to push us over the edge, so to speak.

Moreover, there are among us people who "catch cold" more frequently than their neighbours. Most of them, but not all, will be found to have one or other of the septic foci mentioned above. Whether or no, the suggestion may be advanced that these people are "carriers" of the virus, and very prone to auto-infection and to the heightening in virulence of the infective agent to such a degree that during the acute stage it can pass from one person to another. Thus the general population, the majority of whom are presumably not themselves carriers, is periodically exposed to infection from a large number of centres simultaneously. Indeed, the well-known appearance of a pandemic in England every spring and autumn can best be explained by the concurrence of attacks in carriers susceptible of meteorological influences, and transmitted by them to a populace whose resistance is low.

To put the matter in a nutshell, a susceptible person or child—often a sufferer from chronic nasal catarrh—develops acute catarrh spontaneously, and proceeds to infect his healthy neighbours; and exposure, fatigue, malnutrition, like any debilitating circumstance, favour the onset of the disease, both in the susceptible and in the more resistant, but they are not indispensable factors in the causation.

The treatment of nasal catarrh, of the chronic as well as of the acute variety, is dictated by our views upon its etiology. You and I, as rhinologists, deal with local causes, as outlined above, sometimes with success, sometimes not. Secondly, bacteriologists, convinced of the evil wrought by bacteria, seek to maintain the patient's resistance at a high level by the administration of vaccines, with, I am afraid, not invariable success. Sometimes a course of vaccines will be followed by a year of freedom from acute attacks. At other times an acute attack will set in a week or two after the course of vaccines is concluded. Nevertheless, a course of vaccines twice a year in susceptible people is a simple, harmless procedure, which every now and then is followed by a prolonged intermission. Let us, however, again utter the warning that not even the most potent vaccine will remove nasal polypi. In other words, always look out for and remove local causes as a preliminary.

But at the same time as the specialists are trying their luck, the general physician, the general practitioner, and the general public as well, are all equally busy with their methods of treatment, with the result that the patient is subjected to a bewildering shower of remedies, all of them successful, for a cold in the head gets well of its own accord until the next time. There is, indeed, no practical method of preventing this contagious disease, and there is no specific cure. All we can do is to remove, when possible, all local sources of sepsis and irritation, and to build up and maintain a high resistance against the coryza-producing virus, whatever it may be, by sensible hygienic measures, both personal and communal.

In this connexion we ought perhaps, in our capacity as rhinological experts, to express some sort of opinion upon certain therapeutic counsels that have, of recent times, become very prevalent, if not very popular.

In the first place, we are often advised, if we wish to keep ourselves free from infection, to sniff every day various chemical solutions up the nose. By the way, I am not acquainted with any rhinologist who follows this ritual himself. Perhaps for the very good reason that it is probably quite ineffective and certainly not free from danger. I have known an acute otitis media result from this insult to the upper air passages.

Again, we have to encounter the cold-water, open-air crank, who puts down all the ills that flesh is heir to, and specifically nasal catarrh, to living and working indoors. But this is the twentieth century. Few of us can afford to revert to savagery, and, if we did, how long could we keep it up here in England without developing an acute antrum? Yet these exuberant Bohemians, not content with their own virulently ascetic practices, inflict them also upon their children; and you will see their long and skinny daughters, with red-rimmed eyes and weeping noses, sent out to face a Kingsley's nor'-easter, nude and purple from the ankles to the nates. No one will ever persuade me that that regimen is either safe or sensible—or even decent.

As a matter of fact, this hardening process is not modern. It is very ancient, having been in vogue for something like 2,000 years to our knowledge. Listen to the orations of Aristides—not Aristides the Just, I may say.

"His health," he tells us, "was bad about the time of the winter solstice. It was a stormy day. The god ordered him to rub himself over with mud, and run three times round the Temple. It was a north wind and keen frost. He did as Asklepios ordered, then bathed at the well, and felt fine. . . . But—a companion who imitated him had a paralytic spasm and could scarcely be restored to heat."

Is it asking too much of them to beseech our open-air faddists as they are strong to be merciful, remembering that most of us are but a feeble folk, who, after exposure to rigorous cold, do not experience a warm vasomotor reaction, but, like the companion of Aristides, fall therefrom into a "paralytic spasm," and can "scarcely be restored to heat."

Finally, there are those who go to the opposite extreme, and, wrapping garment after garment around themselves and their children until they have as many coats as an onion, live in a continual bath of perspiration. Rendering themselves thus sensitive to every breath of air they fear a draught as they should the devil, and no wonder, for a current of fresh air is to such people, with their soppy skins and flabby circulation, a real source of discomfort and even at times of danger.

After all, we all know quite well the golden rule of health in these matters. Let us seek the *via media*, and expose our system to extremes of no kind, neither over-coddling nor overtaxing our bodies. By that means we shall obtain the best results—save, to be sure, in the daily press, for this kind of advice is much too quiet and sensible to furnish our journalists and our journalistic medical geniuses with the excitable headings their souls hanker after.

THE INCIDENCE OF TUBERCULOUS INFECTION IN THE MILK SUPPLIES OF SCOTTISH CITIES.

BY

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FOR a number of years attention has been directed to the danger of the milk supply as a potential source of infection of the population with bovine tuberculosis. The report of the Royal Commission on Tuberculosis drew attention to the need for adequate control of the milk supply by local health authorities, and since the issue of that report considerable improvements have been effected, both by legislation and by voluntary efforts on the part of individual milk producers. The increased control of city milk supplies by more frequent periodical tests, the Tuberculosis Order (by which local authorities have the power to inspect all cows which are giving milk for public consumption, and to order the slaughter of those which are either excreting tubercle bacilli in their milk or are in an advanced stage of the disease), and the voluntary establishment of tuberculin-tested herds by individual owners, all tend towards ensuring a decreased incidence of infection in the milk supplied to the large centres of population.

During the last few months considerable attention has been devoted in the public press and elsewhere to the potential danger of the milk supply, and somewhat indiscriminate statements have been made as to the actual extent of the tuberculous infection of milk. Unfortunately no collected data of the present position with regard to infection appears to be available, and, in order to fill this gap in our collected knowledge, an attempt has been made to put together such relevant information as is available with regard to the milk supply of Scotland.