and citric acid, after some time, "saltish." The trapezius on the right side is much wasted, so that the right shoulder is considerably lower than the left, and the right supraspinous fossa of the scapula is flatter than the left. The right sterno-mastoid is also smaller than on the opposite side. There is no deviation of the head.

This patient was subsequently admitted here under my care on March 10th, 1874, whilst I was in charge of the wards of Dr. Russell Reynolds. His condition was then found to be substantially the same, and it underwent very little variation during several subsequent years, during which I saw him from time to time. I have been reminded by Dr. Dawson Williams that he came to my wards to see me as late as the first half of 1880, but whether I have ever seen him since I am unable to say.

Here again no definite cause could be assigned for the patient's malady. It is true there was a doubtful venereal history, but only separated by a period of three or four months from the time when the paralytic of the cranial nerves commenced, rather than by an interval of some years as is usually the case with such lesions. And although the patient was submitted to a long course of treatment with large doses of iodide of potassium together with liquor hydrargyri perchloridi no appreciable benefit was produced. The absence of pain is a point in which this case differs notably from the two others, and another difference is to be found in the long stationary condition and duration of the disease. It agreed with the others, however, in the absence of limb paralysis, of optic neuritis, and of vomiting. I believed this patient's malady to be due, therefore, not to an ordinary new growth, but rather to a chronic thickening of the dura mater over the basilar portion of the occipital and the contiguous borders of the temporal bones. You will recollect how close together the apertures are situated through which the cranial nerves from the seventh to twelfth inclusive pass, on each side of the base of the skull, and consequently how easily they might all be more or less affected by some limited pathological process involving the bones or their covering in this region. I think, therefore, if we had had an opportunity of making an examination that we should have found either a simple pachymeningitis, or else perhaps some localised hyperostosis, which compressed these nerve roots at their points of exit from the skull, in the region indicated.

Taste was very distinctly impaired in this case, and as the roots of the fifth nerves were not affected, we may imagine that the gustatory fibres of the chorda tympani were contained in the nerves of Wrisberg, which go to the brain with the roots of the facial nerves, and subsequently join the glossopharyngeal nuclei.

It only remains for me to add a few words in conclusion concerning the diagnosis of such cases as we have just been considering, in which multiple paralytic of cranial nerves is caused by some disease of the base of the skull itself. We may expect to find in them evidence of a unilateral or other limitation of the lesion (such as occurred in the first and third cases); an absence of paralysis of limbs, of fits, of optic neuritis, and possibly of vomiting; although pain in the head may be severe, continuous, and widespread in seat. In addition to this combination of characters, already sufficiently distinct from those of tumour of the brain, we may have the absence of syphilis or any other assignable cause, and the possible presence, after a time, of a growth in the posterior part of the pharynx, or some other of the structures in connection with the mouth.

There is little likelihood of cases of multiple paralysis of cranial nerves due to peripheral neuritis being confounded with those occasioned by disease of the base of the skull, because in all the former class of cases there would be the coexistence of more or less paralysis of limbs (probably on both sides of the body), and there would, in the majority of cases, be the history also of some acute specific disease, of some alcoholic or other poisoning, or of some exposure to cold, as more or less immediate precursors.

Unfortunately the prognosis, and the chance of doing any lasting good by remedial or other treatment in these cases, is about as bad as it could well be. We must, for the most part, be content to endeavour to alleviate the patient's sufferings as far as possible, to procure sleep, and to stave off the tendency to death which may present itself in various ways. The actual duration of the malady may vary considerably in different cases.

FURTHER OBSERVATIONS ON "NECROSING ETHMOIDITIS."

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RECENT discussion on this subject has disclosed so much confusion of ideas as to seem to demand a restatement of the case, in the course of which I shall refer to some of the criticisms which have found utterance regarding the condition referred to in the title of this paper.

Quite at the commencement of the past decade I had adopted the custom of examining the nose in almost every patient coming under my observation at the Throat Hospital. Many of them were very chronic cases, among which post-nasal catarrhs formed a large contingent. In some of these I found enlargement of the middle turbinated bodies, the behaviour of which, as watched week after week, attracted my earnest attention. Thus some, after attaining the size of a date stone, appeared to remain stationary, while others became much larger, so as to come to occupy all the space between the septum and the outer wall of the nose. In several of the latter a fissure was observed to form, in the course of time, in some part of the presenting aspect of the spongy process; and through this fissure what seemed like granulations were subsequently seen to project. Upon exploring the cleft with a probe bare and rough bone was felt in certain cases. Recognising here a morbid condition quite new to myself, I removed with a snare several of the larger of these masses, and placed them in spirit, submitting them later for microscopical examination to Mr. Thurston, then Pathologist to King's College Hospital.

It is to be noted that no treatment had been carried out in these cases beyond that of the use of an alkaline lotion. Chromic acid had not then come into vogue in nasal surgery—at any rate, it was quite unknown to me at that time for this purpose; so that whatever was discovered by Mr. Thurston in the specimens he examined could not have been brought about by this reagent, nor indeed by any manipulation on my part. Mr. Thurston's report on these specimens, together with his drawings, was laid before the Medical Society in 1885, and was afterwards published in the *Lancet* of that year.¹

A careful study of the structural changes brought to light by this investigation, taken in conjunction with the clinical appearances and the course of these cases, had impressed me with the belief that the essential character of the pathological changes in progress during the early stages of the disease was that of a hyperplasia and fibrosis of the soft parts; that this hyperplastic and fibrosing process led in the natural course, on the one hand to the development of myxomatous overgrowth, and on the other hand to the invasion with irregular absorption and consequent disintegration of the delicate subjacent bony layer, which was observed to break up into small detached fragments; and that this necrobiotic change was reinforced and accelerated by concomitant thickening of the coats of the arteries, causing narrowing or ultimate obliteration of their lumen, thereby starving the tissues by cutting off their blood supply.

Actuated by this belief, I conceived it my duty to remove these masses as soon as the existence of the above-mentioned changes could be ascertained, with the view of obviating the morbid developments which had been observed eventually to result from them.

I desire here to draw attention to the fact, in the cases referred to as amongst the first observed by me, that when exploring with a probe the cleft above described in the middle turbinated body the instrument was allowed to take its own course through the fissure, and that it thus found its way upwards and outwards towards the outer wall of the nose, to a point corresponding with the continuity of the spongy process with this wall. It was exactly at or near this spot where communication with the ethmoidal cells is most free, that necrosis was found. This fact has been insisted upon by me from the first; and in the directions given for the detection of necrosis,² it is stated that the probe should be passed—

¹ *Lancet*, July 18th and 25th, 1885.

² *Nasal Polypus, etc., in Relation to Ethmoiditis*, page 13.

above the surface of the lower, and under the projecting shoulder of the middle spongy bone, in such a way as to pass between the latter and the outer wall of the nose. After passing a short distance in this direction it will enter the ethmoid bone through the hiatus semilunaris, which is usually abnormally large in this affection. The search for exposed bone should be extended by withdrawing the probe, and directing it towards the roof of the nose above the tumour; it will then enter the orifice of communication with the posterior ethmoidal cells, which communicate in turn with the sphenoidal sinus. This opening is also usually enlarged, and here rough and exposed bone, offering a very firm resistance, may be encountered.

Although the points in the diagnosis now recapitulated were then duly set forth, it was only later, since the publication of the work above quoted, that their full significance came to be realised. My earliest observations being concerned with the changes occurring in the middle spongy body, I was naturally disposed to believe that the morbid processes were manifested mainly in connection with that body. With increasing experience, however, it became apparent that necrotic changes were more often present in the ethmoidal wall of the nose and the adjacent ethmoid cells than in the turbinated bone itself. Having convinced myself of this fact, I placed considerable emphasis upon it in a paper read at the meeting of the British Medical Association at Bournemouth in 1891.³

It was there asserted that necrosis declares itself in a certain number of instances "clinically in situations where the disease was not removable by operation: for example, the nasal wall of the ethmoid cells. Where this wall had in part disappeared the ethmoid cells themselves exhibited evidence of necrosis, presumably arising by extension from the turbinated process, though in some instances the disease probably originated in the cells, and affected the spongy process later on." In the same paper attention is called to the large class of cases in which "the middle spongy bone shows no departure from the normal, except perhaps in looking redder and of less even surface than usual. The necrosis will in such cases be found in the walls of the ethmoid cells, which are frequently partly broken away, so that the probe grates against the detritus as it enters one of these cells."

The obvious inference from these remarks is that implication of the middle turbinal process is frequently secondary to disease, seated further from the surface, in the cells of the ethmoid bone. That this is so I am daily more convinced; but since neither these cells nor any sufficient portion of this part of the nasal wall can be directly observed by the eye, but only explored by a suitable probe; and as moreover it is not clear how these diseased areas are to be obtained for microscopical examination, how, it may be asked, are we to recognise in this region the presence of the processes which precede necrosis, and which, in all probability, lead up to it? So far as I can see at present this seems likely to be done only by observing and comparing the changes attending the development of necrosis in the spongy process itself, when this happens to be also involved in the disease.

What these changes are has been repeatedly described. The same fibroid and mucoid proliferations, producing arterial narrowing and obliteration, progressive absorption and disintegration of the osseous lamellæ, and development of myxoma, which are sometimes seen at work in the turbinated body, may not unreasonably be presumed to occur likewise in connection with the ethmoid cells, which are not thus visible. It would seem also justifiable to imagine that in this region, where the thickness of the bony plates scarcely exceeds that of tissue paper, the morbid processes tending to bring about necrosis induce such a result more rapidly than they do in the spongy body. Inasmuch, too, as the earliest information we can obtain of their existence here is through the detection of rough and bare bone by means of the probe, it is evident that, from a practical point of view, the condition presents itself to the surgeon as one of necrosis from the first.

With reference to the detection of dead bone in this situation, I may here mention the fact that among the students attending my hospital practice, who are always invited to examine these cases, very different degrees of tactile sensibility are noticeable. Thus, out of say four students present, there would be almost always one who could not distinguish between dead bone and other structures, although the rest could readily do so. This would appear to suggest that delicacy of "touch perception" may be as variable in different individuals as is the appreciation of musical tone; and if this be true, as is inherently not improbable, it may possibly furnish an explanation of a certain proportion of the dissentient criticism which has been so freely expended on this subject.

As regards the probe I recommend for the detection of necrosis, it should obviously be adapted to the particular region to be explored. When, for example, necrosis is present at the fundus of a ruptured cyst—the cleft of which faces the observer—an ordinary fine surgical probe is as good as any other. In examining, however, the structures between the outer wall of the cavity and a greatly enlarged middle spongy bone, or those in the space above the latter, which includes the greater part of the ethmoidal wall, such a conical-ended surgical probe could not be introduced at all in many cases. Not having at first an instrument suitable for this purpose, I employed a canaliculus probe—not by any means a very fine one, but such as could be easily insinuated through the passage. The suggestion that the necrosis found was caused by its use could not possibly be made by an experienced observer, who would be familiar with the fact that, even in severe traumatism—for example, simple fracture of the leg—necrosis is almost unknown. What probability, then, is there that it could be produced by the scratch of a probe? That which I have long adopted is a flattened band of soft silver, rather less than a millimetre broad, and of about the thickness of the larger-sized canaliculus probes, with the end carefully rounded and finished. It is mounted on a handle, and is so soft as to readily take the shape requisite for its introduction into the sinuses communicating with diseased parts situated in or beyond the outer wall of the nose.

It would appear from the tone of some of the correspondence which has seen the light in connection with this subject that I am not sufficiently alive to the importance of the proper naming of this condition of disease. I confess that, when I think of it in association with certain other points which have been made very evident by this discussion, the naming of the disease seems to me to be a matter of secondary importance. At present the object I have in view is to obtain from medical men, for what I have called "necrosing ethmoiditis," the recognition of the pathological changes which constitute it an entity in disease. Compared with this, the name by which it is called is, as I have said, a matter of quite subsidiary consequence.

Anxious as I am to avoid all polemical matter, I am nevertheless constrained to refer briefly here to an apparent misunderstanding which requires correction. In the course of the correspondence which has recently taken place in the BRITISH MEDICAL JOURNAL in connection with this subject, a letter appeared from Dr. Sidney Martin regarding my citation of his examination and formal report on the specimens of this disease, which had been submitted to him by me. In this letter he, irrelevantly enough, "disclaims responsibility" for affirming the existence of "necrosing ethmoiditis." As I have already stated, Dr. Martin was simply requested to examine some specimens of this affection three years after I had already published a full account of it, the object of this second investigation being to confirm or confute, as the case might be, the results of Dr. Thurston's previous research. In adverting to Dr. Martin's evidence I never imputed—it is scarcely conceivable that I could have intended to impute—to him any responsibility for the enunciation of an opinion upon a disease, which is something more than a categorical account of histological changes. His report, however, stands good, and constitutes complete evidence of the truth of my thesis, in so far as this is capable of proof merely by microscopical examination, for it corroborates in every particular the prior results obtained by Dr. Thurston.

The abstract of the report now alluded to was reproduced *verbatim* in my paper which appeared in the BRITISH MEDICAL JOURNAL of March 12th, 1892. The exigencies of space forbid its repetition *in extenso* here, but I may perhaps be permitted very shortly to summarise its substance. The report shows:

(1) That in every one of the twenty specimens examined there was "a profound change in the mucous membrane, the result evidently of a chronic inflammation."

(2) This change consisted of simple increase of fibrous tissue, or of this together with small-celled infiltration, or, again, of the same changes with the addition of increase of myxomatous tissue.

(3) In nearly all the cases the glands were compressed by the fibrosis, and in many were so completely atrophied that only a trace of them could be found.

(4) The venous sinuses were likewise compressed in every case but two; in some they were even obliterated, in others distorted and separated from each other.

³ BRITISH MEDICAL JOURNAL, March 12th, 1892.

(5) The arteries gave evidence of marked change in five cases; their coats were thickened, their lumen narrowed, containing thrombi in some situations.

(6) There was absorption of bone in nine cases. In some places the absorption had progressed right across the bone, the place of the latter being filled up by fibroid tissue. In one case "necrosis was complete," in another "only partial," and in both the arteries were affected as above described.

(7) Some cases showed "cysts expanding the bone, associated with fibrosis of the mucous membrane."

I give *verbatim* Dr. Martin's conclusion as appended to his report:

"Conclusion.—From the result of the examination of the specimens I should conclude that the disease is apparently a primary chronic inflammation of the mucous membrane, which may take several courses: 1. There may simply be the fibroid condition of mucous membrane, *with or without* absorption of bone. 2. There may be a tendency to great development of myxomatous tissue. 3. There may be necrosis of the bone in connection with diseased arteries (2 cases out of 20), Cases 1 and 7. 4. There may be cystic formation, Cases 2, 9, 11."

In forwarding his report,⁴ of which the above is a faithful epitome, Dr. Martin favoured me with the following statement: "I enclose final report of specimens of middle turbinate disease. I have stated the matter as fairly as I can from a pathological standpoint. If you intend to publish drawings, it will be well to have a skilled draughtsman to do the specimens of necrosed bone and of absorption of bone." I have only to add that Dr. Martin selected his own draughtsman, Mr. Lewin, who is well known for the fidelity and skillfulness of his work, and that each drawing was submitted to and approved by Dr. Martin.

I think, therefore, that, in reviewing all the ascertained facts, no impartial and competent critic can reasonably resist the convincing nature of the evidence which has been accumulated for the existence of definite and constant pathological changes observed in association with the clinical manifestations of this form of nasal disease.

FURTHER EXPERIMENTAL NOTE ON THE CORRELATION OF ACTION OF ANTAGONISTIC MUSCLES.¹

By C. S. SHERRINGTON, M.A., M.D. F.R.S.

In a previous communication² it was shown that physiological contraction, and even mere mechanical tension of the flexor muscles of the knee, exerts considerable physiological influence upon the activity of the antagonistic muscles, the extensors. For instance, the elicitation of the "jerk" from the extensors can be rendered impossible or difficult for a time by appropriate excitation of the flexors, and can, on the other hand, be much facilitated by flaccidity or paralysis of the latter.

In order to judge whether under these circumstances the briskness of the knee-jerk varies directly with the degree of tonus of the extensor muscles, the rapidity of onset of rigor mortis has been selected as a guide to the degree of tonus obtaining in them before death. The experiments of Brown-Séquard, Kölliker, and Hermann and his pupils, have discovered that section of the nerve of a muscle, even when performed very shortly before the death of the animal, considerably delays the time of onset of rigor mortis in the muscle. After control experiments which simply verified the statements by these observers, experiments were instituted on the influence of section of the motor spinal roots upon the time of onset of rigor mortis. The examination showed that a marked delay of onset of rigor mortis was thus produced. The delay seemed to be as considerable as after section of the entire muscular nerve. The effect of section of the posterior roots was next examined, and found to be a marked retardation of the onset of the rigor; the retardation was greater if the spinal cord remained intact than if the cord were previously severed across in the region of the first lumbar segment. The effect of placing and keeping one of the hind limbs in the pose most favourable for elicitation of the

jerk (knee flexed) and the other limb in the position in which the jerk is restrained (knee extended) was investigated, in every case after previous severance of the spinal cord at the first lumbar segment. It was found that upon the side on which the knee had been kept flexed the onset of rigor was delayed in the extensor muscles, whereas upon the opposite side, in which the knee had been kept extended, rigor was delayed in the flexors. This infers that the tonus of the extensors is heightened by excitation of the antagonistic set, and conversely.

The research was also extended to the examination of the mutual association of action of antagonistic muscles about other joints than the knee. It had been noticed in an earlier series of observations³ that during excitation of the cortical areas of the hemisphere, when isolated movements of the pollex and hallux are being initiated, the movement of response obtained is often reversed by section of the peripheral nerves supplying the muscles predominating in the movement obtained. Thus, for instance, flexion can, by section of the flexor nerve, be at once converted into extension or *vice versa*. Sometimes, however, movement in the same sense, although diminished in force and extent, persists even after section of the motor nerve to the predominant group of an antagonistic pair, thus indicating that in some cases accompanying contraction of the one group of muscles there is concomitant inhibition of the antagonistic. This evidence of inhibition of one set of the synergetic muscular couple during the co-ordinate movement induced by cortical excitation is in the cases of the digits, hallux, and pollex of comparatively infrequent occurrence, but it is quite usual in the case of ocular movements. By stretching or by exciting with tetanising currents the inferior oblique muscle of the eye, reflex movements in both eyes can be readily evoked, but these are of variable direction. When the external rectus muscle of one eye (for example, of the left eye) having been put out of action, the frontal cortex of the right hemisphere is excited, the eyeballs, if previously directed to the right, revert both of them to the left—that is, the excitation which evokes contraction of the right internal rectus evokes also relaxation of the left internal rectus. Again, when the internal rectus has been put out of action—for example, in the left eye—excitation of the left frontal cortex produces, if the eyes have been previously directed to the left, an immediate movement of both eyeballs to the right, the left eye frequently rotating beyond the median primary position. Here the same excitation of the cortex which induces contraction of the right external rectus muscles induces synchronously a relaxation of the left internal rectus muscle.

These inhibitions of the contraction of one antagonist concurrent with augmentation of the contraction of its opponent are obtainable as well from appropriate areas of the occipital cortex as from areas in the so-called motor region. Also there can be inhibited from the cortex not merely the "tonus" but the active contraction of ocular muscles. During volitional movements similar phenomena occur, but appear to be less obvious than during experimental excitation of the hemisphere with moderate currents. Although inhibition of contraction is apparently so extremely common a factor in the mechanism of the mutual co-ordination of the antagonistic lateral straight muscles of the eyes, they yield occasionally good indication of the existence of synergetic contraction as well as synergetic relaxation. The mutual association of the two oblique muscles of the eye seems to be usually of the nature of concomitant contraction, not of contraction coupled with relaxation. On the other hand, the muscles which close and open the palpebral fissure appear to be altogether independent one of the other in the action. In their case section of the particular peripheral nerve concerned in by the movement is at once followed by total disappearance of the movement, and that without reversal. Although the cerebral cortex exercises inhibition apparently so usually in the field of innervation of the third nerve, the dilatation of the pupil so readily produced by excitation of that portion of the cortex appeared whenever tested to be due to impulses discharged *via* the cervical sympathetic, and not to inhibition of the constriction exercised *via* the third nerve.

⁴ December 26th, 1889.

¹ Abstract of a paper communicated to the Royal Society.

² Sherrington, *Proceedings of the Royal Society*, February 1st, 1893.

³ *Journal of Physiology*, vol. xiii, October, 1892.