

at the very commencement of that disease, and when it is not seemingly quiescent, I think the remedy should be tried, because no drug is known to have anything like the beneficial effect of tuberculin upon those tubercular diseases which can be seen and handled, and evidence, it seems to me, is accumulating tending to show that the beneficial effects which we see produced in lupus and other similar conditions by the use of tuberculin are also produced in the diseased lung which we cannot see. And another reason for its use in very early stages of lung disease is that, in cases of this kind, there is, so far as I know, no evidence to show that there is material risk in employing tuberculin. The remedy has been already used in several of these early cases, and no evil, but, on the contrary, material good, to the patients concerned has resulted in no inconsiderable number of these early instances of lung disease. In such cases as these, I can see no reason why there should be any hesitation in using tuberculin as a remedial agent. In lupus vulgaris, however, the beneficial effects following the proper use of tuberculin are so evident and so marked, that I feel sure this remedy will very soon become an essential part of the treatment of that disease. In two of my cases of lupus fresh points of infection were distinctly evident, and these manifested themselves to me, and to the patients and others, during the last four weeks or so of the five months' course of treatment to which these patients were submitted, and while they were taking daily increasing doses of tuberculin ranging between 200 and 1000 mgr. The cases now referred to are those marked on the table as L. F. and E. F. In the former case the points of fresh infection were observed upon the upper lip and right cheek, and in the latter over the right temple. These two cases both show, as is indeed already well known from other sources, that in spite of the excellent results which, up to a certain point, practically always follow the use of tuberculin in lupus vulgaris, danger of the reappearance of the disease is so great that, as Koch himself says, we must make "use of all other auxiliary methods to assist the action of the remedy."

From a paper of this kind it is inevitable that much that is of importance and of interest must be omitted. I have tried to indicate certain points in this question concerning the value of tuberculin as a remedy, but of necessity I have left very much unsaid.

I congratulate the Society upon the fact that you, Sir, preside over our debates, for there is no one else into whose hands the conduct of our proceedings could be better confided, or in whose sense of what is just and right to all concerned we could more surely place a perfect trust.

#### NOTE ON A SUCCESSFUL CASE OF OESOPHAGOTOMY FOR REMOVAL OF A TOOTH-PLATE IMPACTED IN THE OESOPHAGUS FOR FIVE YEARS AND NINE MONTHS.

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THE longest period of impaction of a foreign body in the oesophagus removed successfully by operation which I can find recorded is three years and a half: the patient was a child; the foreign body a halfpenny. The case is recorded by Mr. Bennett May.<sup>1</sup> In the woman whose case I relate now the foreign body was a silver-gilt tooth-plate with three gold hooks, and carrying three teeth, and had been swallowed five years and nine months before the date of operation.

E. B—, aged forty-four, married, mother of a family, was admitted for the first time into the Sussex County Hospital on Nov. 6th, 1889. She complained of difficulty in swallowing, was thin, but fairly nourished. She said that on the morning of May 4th, 1884, whilst suckling her infant, and turning in bed, a tooth-plate slipped from its position in the upper jaw to the back of the throat; she felt almost suffocated, and being unable to seize the plate pushed it down her throat. Afterwards she experienced difficulty in swallowing and could only take liquid food, occasionally thickened with minced meat or bread crumbs.

The dysphagia varied, but was always present and had much increased lately. She referred the seat of obstruction to a point about an inch and a half below the episternal notch. There had been occasional dyspnoea, lasting only for short periods, a minute or two. She was quite sure of the date of the accident, as she was at the time suckling her youngest child. A probang passed down the oesophagus met with obstruction ten inches and a half from the teeth; a coin-catcher got hold, but traction only seemed to tilt the plate into the surrounding tissues. One pair of long oesophageal forceps gripped the body firmly, but slipped off, and no fresh hold could be obtained. Further measures were then proposed, but the patient wished to return home.

She was readmitted on Jan. 30th, 1890. On Feb. 17th, under chloroform, oesophagotomy was performed. The usual incision was made, about three inches in length, along the anterior border of the left sterno-mastoid muscle. The omohyoid muscle was divided. The carotid sheath having been drawn outwards and the trachea and thyroid gland in the opposite direction, the oesophagus with the recurrent laryngeal nerve on its surface was then exposed; a full-sized bougie was now passed through the mouth down to the obstruction, and the oesophagus opened on its posterior surface. On passing the finger into the wound the foreign body could just be reached lying somewhat obliquely to the canal of the tube, the upper part projecting into the canal, the rest of the plate lying in a sac to the right of the gullet. The irregular surface of the plate was so closely surrounded by the walls of the sac that great difficulty was met with in its extraction. Various forceps were tried, but slipped off. It was finally grasped by a strong straight pair, and by a combined movement of pulling and rotation, requiring considerable force, it was at last removed. About an ounce of bright arterial blood followed the removal of the plate. The wound was well washed with weak carbolic lotion. One small artery to the sterno-mastoid required ligature; the oesophagus was not sutured. The upper two-thirds of the external wound were closed, the lower one-third left open, and a drainage-tube reaching up to the oesophagus was fixed in the lowest part of the external incision.

There was only slight shock, but no vomiting, after the operation. The wound was frequently syringed with boric acid solution (ten grains to an ounce of water), some of which the patient was directed to swallow occasionally; the teeth and gums were also cleansed with the same solution. No nourishment was given by the mouth during the first two days, but four ounces of pancreatised beef-tea were administered per rectum every four hours. In spite of all, the wound became offensive, the saliva flowing from it copiously. On the third day, as the wound showed no sign of repair and the patient was losing ground, an attempt was made to introduce food into the stomach through a No. 8 gum elastic catheter passed from the mouth. The mere passage of the instrument down the oesophagus produced retching, and although only about an ounce of milk had passed into the stomach, it all returned through the wound. No further attempt was then made to feed through the mouth until the morning of the fifth day from the time of operation. A small soft catheter was then passed through the mouth into the gullet beyond the wound, but not into the stomach. A little cold milk was then allowed to trickle by degrees through the catheter, but it all regurgitated through the wound. The presence of the catheter in the oesophagus so long unused to solids seemed to produce spasm. During the day the patient became much feebler and very restless, and complained of great hunger. The wound was now covered with greyish exudation, and the whole side of the neck down to the clavicle in front, and to the level of the spine of the scapula behind, was red and puffy. It was determined to make another attempt to get food into the stomach, and on the evening of the same day, the fifth from the operation, the patient was raised and supported in bed, and leaning slightly forward was given some milk to swallow in the natural way. She drank ravenously ten ounces of milk, only about a teaspoonful coming through the wound, which was then syringed out in the sitting posture. From that time she began to recover. She took her food, consisting of milk only, or with an egg and brandy, always in the sitting position. The wound soon discharged copiously, and the redness and puffiness of the neck gradually subsided. On the twenty-first day from the operation she swallowed bread-and-milk; on the twenty-fourth day she took fish; on the

twenty-eighth day the wound was healed. The patient was discharged well a few days later with no alteration of the voice. When seen some months afterwards she was stout and well, and could take food without any trouble.

*Remarks.*—Out of just 100 cases recorded, including my own, I find twenty-three deaths. In nearly all the fatal result was due to exhaustion and cellulitis of the neck. The two most important questions, therefore, in the after-treatment of these cases are feeding the patient and keeping the wound clean. There are many different opinions as to the best means of feeding the patient. Barton<sup>2</sup> and Markoe<sup>3</sup> are in favour of feeding through a tube introduced into the œsophagus through the wound. This plan involves disturbance of the wound, the more so if the œsophagus be incised on its posterior surface, which may be regarded as the seat of election if the position of the recurrent laryngeal nerve be considered. Southam,<sup>4</sup> in his two successful cases, fed by enemata for the first fourteen days, and subsequently by a soft tube introduced through the mouth. I tried this in my own case and failed. Abbé<sup>5</sup> approves of closing the œsophageal wound and advocates feeding through a tube protruding from the mouth from the first. Lediard<sup>6</sup> allowed rectal alimentation for the first three days, and then fluids by the mouth. Dr. Geo. Fischer<sup>7</sup> says every patient, whether the œsophagus has been closed or not, may be allowed to swallow fluid diet a few hours after operation without the aid of the stomach-tube. So far as the experience of a single case may justify an opinion, I would advise that the patient be fed by peptonised beef-tea enemata, with or without brandy, for the first twenty-four hours or less until the risk of vomiting from the anæsthetic has subsided, and then be allowed to swallow milk. If the incision be made in the posterior portion of the œsophagus, and the patient drinks in a sitting position, very little escapes by the wound, even if the edges of the gullet have not been sutured; no irritation seems to be produced, but vomiting and retching are most injurious. The wound may be syringed out with a weak solution of carbolic acid (1 in forty) whilst the patient is sitting up, if the œsophagus has been incised posteriorly, without any harm resulting. In my case strong boric acid solutions failed to keep the wound sweet. The carbolic lotion was much more effectual. Two teeth have been broken away; one probably when the first attempt at extraction was made with the œsophageal forceps, the other at the time of the operation.

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## SOME POINTS ON THE RELATIONSHIP OF THE EYE TO THE CARDIO-VASCULAR SYSTEM.<sup>1</sup>

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SOME points of connexion between the eye and the cardio-vascular system are already well established. Eye symptoms occur in chronic albuminuria, syphilis, diabetes, and anæmia; also in some cases of chorea and epilepsy, migraines, and exophthalmic goitre. Various drugs affect both—e.g., alcohol, tobacco, atropine, lead. Dr. Broadbent, in writing on high-tension pulse, mentions glaucoma as one of the conditions associated with high arterial tension, of which he regards it as a consequence, although the mode of causation is not clear. There being, then, some *prima-facie* evidence for a connexion between the eye and the cardio-vascular system, I propose to offer some observations tending to show that the connexion is more intimate than it is at present usually considered to be. Dr. Saundby, in his Lectures on Bright's Disease, speaking of optic neuritis, says: "Neuritis probably occurs as an idiopathic affection, especially in hypermetropic eyes." And again, speaking at Birmingham: "I would express my belief that children with hypermetropic eyes are especially liable to severe attacks of optic neuritis, which may be accompanied by headache, vomiting and giddiness, so as to simulate some coarse intra-cranial

disease, the whole phenomena being, however, directly referable to the visual disturbances."<sup>2</sup> Dr. Gowers writes: "One other fact must be mentioned in connexion with the diagnosis of the cause of optic neuritis. In many cases in which slight neuritis of chronic course is associated with symptoms which would scarcely suggest the existence of disease such as would cause neuritis, hypermetropia exists. This combination may be noted, for instance, in chlorosis, in epilepsy, apparently idiopathic, and other slight symptoms of cerebral disturbance. It is doubtful, in the present state of our knowledge, what share is to be attributed to the hypermetropia in the production of the neuritis, and from the commonness of hypermetropia the coincidence may have been accidental; but the fact deserves notice."<sup>3</sup> In cases of optic neuritis in anæmia mentioned by other observers, it is also generally noted that the refraction was hypermetropic. And personally I think hypermetropia of frequent occurrence in anæmia. Dr. Stevens<sup>4</sup> of New York considers that chorea is caused by hypermetropia, and Dr. Gowers has noted the connexion of optic neuritis and hypermetropia in chorea.

Now, while this association of hypermetropia with diseases causing optic neuritis has been recognised, I do not think sufficient importance has been attached to the significance of it, especially when taken in conjunction with the fact that optic neuritis from all causes occurs with far greater frequency in the hypermetrope than in the myope, or, I believe, in the emmetrope. Optic neuritis in the myope is, in my experience, unknown, and in the experience of others an occurrence of extreme rarity. So far, I have collected three cases only of optic neuritis in which myopia was noted. Now this fact, if fact it be, of the exemption of myopes from optic neuritis, while interesting in itself, may not at first sight appear to have any connexion with the subject before us. But the whole contention of my paper is that we are wrong in considering hypermetropia and myopia as merely local conditions; they are the outward and visible sign of an inward and vascular state. I hold that both are associated with certain general conditions, that the changes which lead to myopia are not limited to the eye, but are part of a general change, and that the arrest of development in the hypermetropic eye corresponds with similar partial development in other structures—in short, that there is a myopic and a hypermetropic cardio-vascular system. Following on this, it might not be unreasonable to expect that myopes may be free from certain diseases affecting hypermetropes, and also *vice versa*.

Two explanations have been given me of the absence of optic neuritis in myopes: 1. The comparative rarity of myopia itself. 2. That local disease must have a local cause. I admit that the frequent connexion of optic neuritis with hypermetropia may be partly accounted for by the far greater frequency of hypermetropia than myopia; but this does not, to my mind, offer anything like a sufficient explanation of the total absence of optic neuritis in the myope, save from exceptional causes. Myopia may indeed be rare, but surely not so rare as to prevent common diseases, such as frequently cause optic neuritis, having been observed in myopes, and the occurrence of optic neuritis in a certain proportion of these cases. The extreme rarity of optic neuritis in myopes would appear to point to one of two things—either a structural difference in the myopic eye, or a structural difference in the body of the myope, rendering him less liable to this form of disease. Occasional cases of optic neuritis in myopes, from exceptional causes, show that the condition is not structurally impossible. But on the point as to whether the myope enjoys any exemption from chronic albuminuria and other diseases giving rise to optic neuritis, I can make no statement.

I propose, then, to give shortly the chain of observation which led me to the conclusion that myopia and hypermetropia ought to be considered as indicating a general, instead of a local, condition, and it may be well to observe that my conclusion was arrived at as the result of my observations, and that it differed entirely from my original theory. Amongst other differences between the two classes of hypermetropes and myopes, the mental and physical differences are early forced on one's notice. But while I had for a long time made mental notes on the two classes, and had from time to

<sup>2</sup> Ibid., July, 1887.

<sup>3</sup> Ibid., Sept., 1886.

<sup>4</sup> THE LANCET, Dec. 28th, 1889.

<sup>5</sup> New York Med. Jour., 1886.

<sup>6</sup> Clin. Soc., vol. xviii.

<sup>7</sup> Deutsche Zeitschrift Chir., Bd. xxv., Heft 6.

<sup>1</sup> A paper read before the Harveian Society, Nov. 6th, 1890.

<sup>2</sup> Saundby: Optic Neuritis in Children, Birmingham Med. Review, 1885, xviii.

<sup>3</sup> Medical Ophthalmoscopy, p. 85.

<sup>4</sup> New York Medical Record, Aug. 12th, 1876.