not live long enough for the slowly occurring skin-change to take place; but though I have searched through many monographs on the subject, I cannot find that any one has tried to discover *why* they died so early. And I venture to believe that the explanation is not only a perfectly simple one, but that it opens up a marvellously wide field for future investigation. For if patients, without bronzing of the skin, die in almost exactly one-fifth of the time lived by those in which pigmentation occurs, it is perfectly plain that bronzing of the skin, *per se*, does not conduce to death, and therefore that the cause of the greater fatality, in the first class, must be looked for in an unusual gravity of the constitutional symptoms, which, together with the skin-change, make up the whole outward expression of the internal capsular disease. That these are unusually grave, is self-evident, seeing that the heart fails so rapidly. It may, therefore, plainly be inferred that the mischief which produces these, must be more intense in non-bronzed than in bronzed cases. Now, inasmuch as the skin-change occurs sometimes long after, and sometimes before, the onset of the constitutional symptoms, and sometimes not at all, it follows that these two constituent features of the disease cannot be of absolutely identical origin. In other words, either two distinct causes must be at work, or the same cause must act upon two separate parts, attacking first one and then the other, in order to produce these two non-contemporaneous results. I think that the idea of two distinct causes may be dismissed, because, after twenty years of patient and accurate investigation of this subject, by the most able pathologists and clinicians of this and other countries, no evidence in favour of such a theory has been shown to exist. It is probable, therefore, that we have to deal with one settled cause, acting upon two separate organs, or upon two separate parts of one and the same organ. Now, let us turn to the pathology of the disease, and what do we find? First, we find that the constitutional symptoms, the anæmia, the asthenia, the breathlessness, the evidences of cardiac and of gastric irritation, have all been, and I think well, explained by the theory of nerve-implication. But this has hitherto been looked for, and supposed to occur, *outside* the suprarenal bodies. And the one weak point in the theory has hitherto been, that nearly every observer has found different or modified changes in these nerves from that recorded by nearly every other investigator. Turning, however, to the minute anatomy of the capsules themselves, we find that Leydig (*Lehrbuch der Histologie*, Frankfurt, 1857, p. 189), as the result of his laborious investigations, comes to the conclusion that the medullary portion of the organ is essentially a "ganglionic nerve-centre," and also that numerous observers have traced branches from the sympathetic, from the pneumogastric, and from the phrenic nerves, into the substance of the medulla. I would ask, then, what need have we to hunt through neighbouring structures, when it seems that we have, in the very capsules themselves, both nerves to be affected, and a degeneration which must affect them?

And now I would suggest, what our argument has led us to believe to be at least possible, that the constitutional symptoms of Addison's disease are due to degeneration of the *medullary portion* of the suprarenal capsules, and that the reason, therefore, why bronzing does not occur in some cases, may be because, in such, the intensity of the mischief is greatest in that part, and causes death before the slowly developed skin-change is perceptible.

But, secondly, as to the bronzing of the skin. We find, histologically, that it is due to the deposit of pigment in the deeper layers of the rete mucosum. Clinically, we find that it is predominant where pigmentation is normally present; and, so far as I can discover, that is all we know about it. Our argument above led us to the belief that we have to deal with one common cause, a caseous degeneration, acting on two separate parts, perchance of one and the same organ. Now, it is noteworthy that we have, apparently, in the suprarenal body, an organ which is, it seems, in a superlative degree, composed of two totally distinct parts. For, turning again to the minute anatomy of the capsule, we find that Kölliker (*Manual of Human Histology*, Sydenham Society's Translation, vol. ii, p. 219), while agreeing with Leydig, above quoted, in referring the medullary portion to the nervous system, regards the cortical part as made up of "blood-vascular glands." If this be correct, I would suggest, as an almost logical sequence, that bronzing of the skin may be due to degeneration of the *cortical layer* of the suprarenal bodies, perhaps in consequence of some physical or chemical change in the constitution of the blood, taking place through the affection of the "blood-vascular glands."

I have noted, in my description of the case above given, that parts of the cortex were healthy, and that generally, in that portion, the disease appeared to be in its initial stage; whereas the medullary portion had undergone much caseous, or farther advanced, degeneration;

and in several museum-specimens, I have found the same naked-eye appearances where bronzing was absent. Argument then, and the still more forcible logic of hard facts, seem to point to the above conclusions. The truth, or the error of the theory, must be decided by accurate and extended observation as to the distribution of the disease, macroscopically and microscopically, in future cases. If it be true, I need hardly point out what a wonderful field for investigation it will open up, not only into the functions of the ductless glands, not only into the chemical phases of degenerative changes, but also into the great practical question of cure. For, seeing that the disease is inevitably fatal, if it be proved to be confined simply to the suprarenal capsules, why should not the organs be extirpated? Physiologists have shown that their presence is not essential to life. I venture then to think that the question I would leave future observations to finally settle, is not only important theoretically, but, in its aim, intensely practical also.

I would briefly summarise, therefore, that (1) the average age at death in Addison's disease is between 30 and 35; (2) males are much more subject to the disease than females; (3) cases with bronzing of the skin usually die within three years, and cases without bronzing, within five months from the appearance of symptoms from progressive asthenia; (4) the *constitutional symptoms* are probably due to degeneration of the *medullary portion* of the suprarenal capsules; (5) *bronzing of the skin* may be due to degeneration of the *cortical layer*; (6) if death be chiefly due to the capsular changes, then the successful extirpation of these bodies might cure the patient.

EVISCERATION OF THE EYE, AND ITS RELATION TO THE BACTERIAL THEORY OF THE ORIGIN OF SYMPATHETIC DISEASE.

By P. H. MULES, M.D.,

Surgeon to the Eye Hospital, Manchester.

IN the last paragraph of my paper on "Evisceration of the Globe," *BRITISH MEDICAL JOURNAL*, Dec. 19th, 1885, I assume that "freedom from sympathetic inflammation, after evisceration, will prove conclusively the bacterial origin of sympathetic disease." I will shortly give my reasons for this statement.

For sympathetic disease to arise in a sound eye, it is necessary that the infecting eye shall have, first, a nidus, or point of infection or irritation; secondly, a track by which such shall be forwarded to the second eye.

In removing the contents of the globe, we certainly remove all soil for the growth of bacteria; but we leave the "tracks," be they blood-vessels, lymph-spaces, or nerves; and although such forcible removal may eventually obliterate the blood and lymph-channels, this can hardly be said of the nerves themselves, at least, for a very long time. Now, if the tracks are there ready to conduct whatever may be required of them, be it sympathy, blood-poison, nerve-inflammation, or bacteria, and we irritate them to the utmost by forcible tearing, medicated application, a permanent foreign body, and other hitherto supposed incentives to secondary sympathetic disease; yet under this treatment, the injured globe quiets down, the sympathetic irritation (reflex neurosis) of the sound eye passes away, and sympathetic inflammation does not occur, obviously the tracks in that portion where they are ruptured, cannot originate sympathetic disease, but must be the conductors only of what is produced, irrespective of themselves. Can the removal of the end organs account for this absence of initiative? Such a view has never been held, nor has it anything to recommend it. Experiment, then, having so far proved that from eviscerated globes sympathetic inflammation does not extend; that the nerves, blood and lymph spaces, under excessive provocation, cannot originate of themselves a disease capable of progression to the sound eye; we arrive, by a process of exclusion, at the fact that it can only be a new formation, an independent cell-growth, introduced probably from without. This point, once allowed (and it appears to me incontrovertible), we have left only the views of Berlin, Leber and Snellen, and Mr. Hutchinson.

Berlin, Leber, and Snellen agree as to the nature of the infective material being bacterial, but differ as to its track, whether vascular or lymphatic; so that, for the purpose of this paper, we may assume them to be one. There remains Mr. Hutchinson's view; "that sympathetic ophthalmitis is of a simple inflammatory character, depending on parallelism of structure for its secondary deposits." To emphasise this Mr. Hutchinson (*Ophthalmological Transactions*, vol. v, p. 74) refers to puerperia. Now this is a disease in which infecting micro-organisms must bear a large share in the production, and is strong evidence in favour of the "bacterial theory of sympathetic

disease;" and another point which I believe I am right in stating as opposed to Mr. Hutchinson's view is, that no well-authenticated case exists (at least of late years) where true sympathetic ophthalmitis originated without a traumatic interference with the exciting eye. Certainly the evidence, as at present adduced, is incomparably stronger in favour of a bacterial origin; and the demonstration by Max Kries of lymph-space infection acquires such additional force and certainty as to make the evidence overwhelming. It is interesting, before closing the subject, to compare the three methods of treatment now in vogue.

In evisceration we have the antithesis of neurectomy. The neurectomist, with a courage that might be better employed, seeks to secure the safety of his patient by a process of "bottling up;" he professes a sublime indifference to bacterial gambols, and by "cutting off their exits, hopes to confine them within the globe." The enucleationist assimilates his treatment somewhat to that of evisceration by removal of the *fons et origo mali*; but like a heavy man seeking to catch a butterfly, throws himself headlong upon insect and flower, and congratulates himself upon his skill, though the flower be broken and the insect crushed. Of the three operations, I cannot doubt that the profession will elect, in all suitable cases, "evisceration." The medical profession is notoriously conservative; but the public, when cosmetic operations are required, are apt to decide somewhat for themselves, and will naturally prefer a pleasing result to the oftentimes hideous deformity which at present exists.

As an example of the method of traumatic infection, I may quote a case from my outpatient clinique of Wednesday, November 21st. "A mechanic was struck on the left cornea by a small chip of steel; a corresponding mark on the iris showed the track of a minute foreign body, which we could not detect" (that it is in the eye is certain). Vision was good, in spite of a streak of blood in the vitreous body; but the point of special interest was a bubble of air floating in the aqueous, which had evidently been carried through the cornea, but wiped off as the chip penetrated the iris. There had been no escape of aqueous humour, the iris being in its proper plane and the anterior chamber full. We shall watch the further development of this case with great interest; at present, operative interference is contraindicated.

REMARKS ON THE THEORY OF BRONCHIAL ASTHMA.

By E. CRESSWELL BABER, M.B. Lond.,

Surgeon to the Brighton and Sussex Throat and Ear Dispensary.

DR. BULKLEY's paper, in the BRITISH MEDICAL JOURNAL for November 21st, 1885, on Asthma as related to Diseases of the Skin, and the article by Sir Andrew Clark, on the Theory of Bronchial Asthma, in the current number of the *International Journal of Medical Sciences*, lead me to make a few remarks on the congestive theory of asthma, more especially as the views which I have formed agree to a great extent with those expressed by the latter author. I do not propose to discuss the validity of the spasmodic theory of asthma. This has been ably done by Sir Andrew Clark; I merely wish to bring forward a few facts, the result of a prolonged study of the nasal mucous membrane during life, which appear to lend support to the view that the bronchial mucous membrane undergoes a temporary swelling similar in kind to that which occurs in certain morbid states of the nasal mucous membrane, though of course infinitely less in degree.

The respiratory nasal mucous membrane contains, as is well known, a large tract of erectile tissue, which is chiefly situated on the inferior turbinated bones. This consists, roughly speaking, of numerous large venous vessels, which, by their distension, cause temporary swelling or erection of the turbinated bodies. The swelling may be observed going up and down very rapidly, and within certain limits, is a normal occurrence. When excessive, and accompanied with sneezing and serous secretion, it constitutes the neurotic attacks which are very commonly seen quite independently of hay-fever. Sir Andrew Clark describes these cases as "hay-fever;" but, inasmuch as they are unconnected with pollen, and are not accompanied by fever, the term is obviously inapplicable to them, and should be confined to those instances in which, from their occurrence at certain periods of the year, pollen is the probable exciting cause. The symptoms already mentioned may be attended by any of the numerous reflex phenomena now known to be associated with the nose, the chief of which are: cough, asthma, redness and itching of the outside of the nose, nightmare, migraine, constant headache, supra-orbital neuralgia, giddiness, epilepsy, etc. Hay-fever is, therefore, according to this view, simply a neurosis of the nasal mucous membrane,

accompanied by certain reflex phenomena, which at particular seasons of the year is produced by the pollen of plants in specially predisposed persons. In other cases, these neurotic attacks are caused by some local irritation in the nasal cavities (such as nasal polypi) or by other irritants inhaled in the air, or they are produced reflexly by the irritation of some other organ (for example, by the action of strong light on the eyes). The constitutional element is, as Sir A. Clark insists, a very important factor in the causation of these symptoms. When a morbid predisposition to erection of the turbinated bodies exists, very slight and trivial causes, which it is unnecessary to detail here, are sufficient to produce swelling of these structures.

The intimate connection which exists between the bronchial mucous membrane and that of the nasal cavities is shown, not only by the occurrence of neurotic nasal symptoms (swelling of the turbinated bodies, watery secretion, and sneezing) at the commencement of an attack of bronchial asthma—a circumstance, in my experience, of very common occurrence—but also by the fact mentioned above, that asthma is frequently a reflex symptom produced by intranasal irritation, many cases of this description being now on record. If further proof were needed, Hack has reported a case in which an attack of bronchial asthma was produced by the application of a powerful irritant (galvanic cautery) to the nasal mucous membrane, in a person who had previously never suffered from the complaint. In many cases, also, bronchial asthma has been temporarily or permanently relieved by treatment applied to the nasal cavities. These few points, which might easily be enlarged upon, suffice to indicate the intimate relation existing reflexly between the nasal and bronchial mucous membranes.

It is, moreover, a remarkable fact that, in true hay-fever, all the other symptoms, with the possible exception of the sneezing, appear to be of a congestive (vaso-dilator) character, whence it may be argued that the asthmatic symptoms are probably of similar nature.

Sir Andrew Clark not only compares the supposed bronchial swelling to that occurring in the nose, but speaks of "mucous wheals" in the bronchi, and likens the swelling, which he supposes to occur there, to patches of urticaria, whose pathology, according to a recent authority (Fagge's *Principles and Practice of Medicine*, vol. ii, p. 673) is that of "acute inflammatory oedema of the cutis, which fills the lymph-spaces and expels the blood from the venules." The much greater frequency of association of asthma with neurotic nasal symptoms than with urticaria, and the closer resemblance of the two mucous membranes, I venture to think, favour the view that if the dyspnoea of asthma, as seems probable, is produced by temporary tumefaction of the mucous membrane, such swelling partakes more of the nature of that occurring in the nose than of the wheals of urticaria.

Whether the structure of the bronchial mucous membrane admits of its undergoing a pathological swelling, by simple distension of its blood-vessels, is a question which must be decided by future research.

An attentive study of the nasal mucous membrane, I have for some time thought would throw light on the pathology of the less accessible parts of the respiratory tract. It is impossible, for instance, to treat many nasal cases without arriving at the conclusion of the importance of distinguishing between the local symptoms which are neurotic, and those which are truly catarrhal in character. The former are marked by distension of the erectile tissue and watery secretion; the latter, by distension of the capillaries near the surface, and by the presence of a mucous or muco-purulent discharge. In the nose, we are able very fairly to separate these two conditions, although, of course, they are very commonly associated. The importance, if possible, of drawing a similar distinction in the bronchial mucous membrane needs no recommendation of mine; it has not only a theoretical, but a very practical, significance.

BRIGHTON THROAT AND EAR DISPENSARY.—At the annual meeting of the Brighton Throat and Ear Dispensary, it was stated that the number admitted during the past year alone was 757; of these, 163 were paying patients, from whom the amount received was £44 9s. 11d., including the "mites" placed in the surgery boxes by many others. The subscriptions, donations, etc., amounted to £217 14s. 9d., which sum had enabled the committee, for the first time for several years, to meet all expenses, and to discharge their last year's indebtedness to the treasurer, amounting, together, to £184 6s. 3d., leaving a balance in the hands of the treasurer of £33 8s. 6d. There was, however, still a debt of £700 on the building fund.

OXFORD UNIVERSITY: DEMONSTRATORS.—J. R. Green, B.A., of Trinity, has been appointed Demonstrator in Physiology, in place of Dr. Lea, resigned; and H. D. Rolleston, of St. John's, Assistant Demonstrator of Physiology, in place of J. R. Green.