and a relapse again occurred on leaving off the medicine. I lost sight of him till the end of 1876, and found him in his original condition. In January, 1877 I began to treat him with the constant current (ten cells gradually increasing) directed transversely through the head in the zygomatic fosse, varied occasionally by the application of one pole here, and the other on the bridge of the nose. After one or two applications, while he felt somewhat giddy, various subjective smells were experienced during the passage of the current, which he described as "gassy", "rank", etc. At the end of a week of daily treatment with the current, he began to smell strong odours; subjective sensations also occurred at intervals, and taste became more acute. He gradually and steadily improved, and, after a few weeks' treatment, the power of smell returned, so that he could recognise such things as assafoetida, musk, coffee, tobacco. He could readily distinguish between one smell and another, but continued to have some difficulty as to identification of the substance. There has been no relapse, and now (Feb. 28th, 1878), at the end of a year, he continues to enjoy perfect taste, and his powers of smell, which were never very acute, he thinks are as good as ever.

I will not attempt to decide what was the exact *modus operandi* of the galvanic current, whether it acted by stimulation of the olfactory nerve direct, or by stimulation of the cerebral centres of taste and smell; but, as a therapeutic experiment, it may be regarded as worthy of repetition in similar cases. But, as regards the pathology of this case, I think it is evident that both smell and taste were abolished independently of each other, and that we cannot account for the loss of taste by the loss of smell; nor can we say that the olfactory nerves were ruptured. It is also in the highest degree improbable that the loss of taste and smell could have resulted from simultaneous affection of the various nerves concerned in these functions; situated as they are so widely apart from each other, and bound up more or less with others not conjointly affected. But it might well happen that such a blow on the vertex as this man received would cause such injury to the subcortical regions, by what is usually termed counterstroke; or by what Duret terms the *cône de soulèvement*, as to cause impairment or temporary abolition of the functional activity of the cerebral centres of taste and smell, which, as experiments on monkeys indicate, are here localised. Hence I would take this, and similar instances in which smell and taste proper are abolished by cranial injuries, as clinical corroboration of physiological experiment.

In respect to tactile sensation, though this form of sensibility is more frequently affected than any of the others by cerebral disease, it is extremely difficult, from a clinical standpoint only, to establish a distinct relation between this and certain cortical lesions, or localise the centres of tactile sensation. Motor paralysis and tactile anæsthesia are frequently associated with each other. But that the cerebral centres of motion and tactile sensation are distinct from each other, is evident from the fact that we may have the most complete motor paralysis without impairment of tactile sensation, as is the case with cortical lesions. And though motion is more or less impaired by the abolition of tactile sensation (by which motion is mainly guided), yet we have many instances in which the power of voluntary motion is retained notwithstanding the complete annihilation of tactile sensation, cutaneous or deep. There is, therefore, no organic fusion of the motor and tactile centres with each other, seeing that each may be affected independently of the other, and the two do not vary quantitatively with each other when they are conjointly affected.

The facts of cerebral disease in general, and of cerebral hemianæsthesia in particular, would seem to show that in respect to tactile sensation there is less bilateral representation in each hemisphere than as regards the other forms of sensibility. For, in central hemianæsthesia, tactile sensation is always most deeply affected, and may still remain greatly impaired after all the other forms of sensory impairment have disappeared. Hence, in the slighter forms of affection of the posterior third of the internal capsule, tactile sensation only may be impaired. Hence, also, with motor paralysis due to lesion of the anterior division of the internal capsule, we frequently get partial or temporary impairment of tactile sensation, owing to pressure on, or slight organic or functional derangement of the posterior or sensory fibres.

If, therefore, tactile sensibility be more unilaterally represented in each hemisphere—and this we might conclude from the remarkable power we possess of localising the seat of tactile impressions on any part of the body—we should naturally expect to find that lesions of the cortical centres of tactile sensation should be accompanied by symptoms of impairment or abolition of this sense. These centres, as I have already indicated, are situated in the hippocampal region. Lesions of this region are not, however, common; and I have not been able to find any cases of localised lesion of the hippocampus, except

those to which I have already alluded in connection with chronic epilepsy and insanity. But, as we have no record of the facts relating to the condition of tactile sensation in these cases, I must leave this question to be settled by future clinical investigation.

There are, however, some facts which would seem to indicate that lesions in the neighbourhood of the hippocampus do cause affection of tactile sensation, though doubt may be entertained as to whether the phenomena are dependent on direct affection of the hippocampus, or affection of the posterior front of the internal capsule, directly or indirectly.

Mr. Jonathan Hutchinson* concludes, from his observations on cranial injuries, that contusion of the sphenoidal lobe more particularly, causes, along with partial motor paralysis, paralysis of tactile sensation on the opposite side of the body. As I have said, these effects may be attributed to injury of the sensory fibres of the internal capsule; but contusion of the sphenoidal lobe might also be interpreted as injury of the hippocampal region; and if the impairment of sensation in the cases described by Hutchinson were to be proved absolutely restricted to tactile sensation, we should have good grounds for considering the phenomena dependent on lesion of the cortical centres here situated. The definitive settlement, however, of these various points must be left to future research.

I have now brought under your notice a considerable number of facts, both positive and negative, in reference to the localisation of special sensory regions in the human brain; and though the positive clinical evidence is as yet comparatively scanty, and leaves much to be desired, I entertain the hope and belief that it will not long remain so. And I trust that those who rely more on the evidence of human pathology and the phenomena of disease than on the facts of experiment, even on the most human of the lower animals, and do not, therefore, share my own very decided convictions as to the localisation of special sensory regions, will take the facts I have adduced into careful consideration, and, when opportunities occur, investigate the conditions as to sensation in cerebral disease, with the accuracy and thoroughness which are much needed in order to clear up the doubts and obscurities which still surround this important question.

CROUP: DIPHTHERIA: TRACHEOTOMY.†

By ROBERT L. BOWLES, M.D., Folkestone.

I HOPE I shall be excused by this society for so persistently bringing before it the well worn subjects of the heading of my paper. But, although these subjects have now for some years past anxiously engaged the attention of our profession, it does not appear, from the long and interesting correspondence in the *Lancet* during the year 1875, and the discussion at the Royal Medical and Chirurgical Society in the latter part of that year, that any decided opinion has been arrived at; still, the tendency of opinion (led chiefly by the authority of Bretonneau, Trousseau, Sir William Jenner, Dr. George Johnson, Dr. Semple, and others) is clearly in the direction of admitting that membranous croup is always diphtheria, *i.e.*, dependent only upon the specific contagium of diphtheria. Dr. West, whose excellent article on croup has always appeared to me to express the truth, in holding the opposite opinion, confesses that he is wavering; and there are many others in the same position as himself.

To no members of our profession is this matter of so much importance as to us who live in the provinces; especially to those of us who are in lone country districts. Croup is of more frequent occurrence in the country; and as many of us are compelled to act upon our own responsibility and act promptly, it is most desirable that we should be well supported by the authority of the leaders of our profession in any views we may feel ourselves compelled to adopt. Suppose, for example, one of us has lost a case of croup in a large country house—in the house of our best patient—that we had looked upon the case as catarrhal and inflammatory, and had treated it with tartar emetic or calomel, the family goes to town and is there told that "such theories are exploded, that the case was certainly one of diphtheria and ought to have been treated by wine, tincture of iron, or such like remedies, and that it was no wonder the child died". The happiness of the family is gone, and the reputation of the doctor with it. It well behoves us, therefore, to give anxious consideration to this question, and to think for ourselves; we have, I think, more opportunities of making accurate and complete investigations into the origin of our cases, than

* *Medical Times and Gazette*, 1875, p. 165.

† Read before the East Kent District of the South-Eastern Branch of the British Medical Association.

our London hospital physicians, and we ought, moreover, to press our experiences and our difficulties on those gentlemen, and prevent them, if there are doubts, from laying down dogmas, that will certainly influence the public and all those members of our profession who do not take the trouble to think for themselves. To this end, then, I now again contribute a paper on croup and tracheotomy.

CASE I.—In September 1874, I was requested by Mr. Hackney of Hythe to see a case of diphtheria, with laryngeal complication; the child was in imminent danger of suffocation; tracheotomy was at once decided upon. There was a membranous patch on each tonsil, but no glandular complication, no swelling of the cellular tissue, and no history of contagion. Was this inflammatory croup or diphtheria? The operation was successful, and on the seventh day, the child appeared quite well, full of fun and mischief; on that day, the mother left it for a time, and for fun, during the mother's absence, the child threw off the flannel covering the tube; the next morning, signs of bronchitis appeared, and the child died in two days.

CASE II.—In January 1875, I was again called in by Mr. Hackney to see a case of what he, this time, called croup in a child which had taken cold, and which had not, so far as could be found, been subjected to the infection of diphtheria. It had commenced with catarrh, and bore the character of the old-fashioned croup; there was no glandular swelling or swelling of the cellular tissue. The case had been treated with tartar emetic and ipecacuanha, and was getting worse, but it was not thought justifiable to operate then; the child was to have a chance, as we had often seen cases of this nature yield to emetics. On looking into the throat, there was a small patch on one tonsil of closely adherent membrane; and then at once arose the terrible question, Is this croup, after all, or is it not diphtheria? Shall we treat the case with tincture of iron and wine, and the larynx with saturated solution of sulphate of copper; or shall we continue the antiphlogistic treatment of inflammatory croup? Yielding to authority and the tone of thought of the present day, we resolved upon the former course, contrary to the opinion we had formed before seeing the membranous patch. The next night, the child was worse, and but for tracheotomy would have died from suffocation. The child lived two days after the operation, and died from exhaustion with symptoms of bronchitis. In neither of these cases was Mr. Hackney able to obtain a *post mortem* examination.

CASE III.—On the evening of Saturday, January 8th, of the present year, I was requested, in Dr. Jervis's absence, to see a case of croup which he had been attending for four days. The history was that the child had a cold on the preceding Tuesday, running at the eyes and nose and slightly croupy cough. On Wednesday evening, the cough became more croupy. On Thursday, the cough was still more so, and the breathing slightly stridulous at times; the child was fourteen months old and teething, so the gums were lanced, and an ipecacuanha and squill mixture given, with instructions that, if the child became very croupy in the night, an emetic of ipecacuanha wine was to be given; the child did become croupy, and the emetic was administered. On Friday morning, the child was better and looking not much amiss; in the evening, croup as usual was worse; and on Saturday morning better, but the breathing perhaps more stridulous than on any preceding morning. A warm room, a steaming kettle, poultices, and other adjuvants, had been employed by Dr. Jervis from the first. On Saturday evening late, as I said before, I first saw the child, *i.e.*, four days from the commencement of the case. The croupy symptoms had become much worse and the breathing persistently stridulous, or rather, I would say, sibilant, notwithstanding the emetics. The child was prostrated by the vomiting. I carefully examined the throat, but could find no false membrane.

January 9th.—On Sunday morning, the sibilant breathing was evidently increased and not at all relieved by vomiting, which had occurred several times during the night; and now (8 A.M.) the child looked very faint and ill; the bowels had been purged three times; the motions were thin and very offensive, evidently from the medicines. Again I carefully examined for signs of diphtheria, but found no false membranes in the pharynx, and no swelling of the cervical glands or cellular tissue, or any history of contagion or bad drains connected with the illness. On the other hand, the child's nurse, the mother, and two servants, had had colds, all partaking of the laryngeal character, before the child was taken ill; and coincidentally with and subsequently to it, the grandfather and grandmother and lady's maid all had colds of a like nature. Colds were also prevalent in the town, but there was, to my knowledge, no diphtheria. Moreover, there were distinct dulness on percussion and fine rhonchus at the lower half of the right lung. The pulse was quick, but the child was so alarmed at my approach that I found it difficult to be accurate as to its number, and for the same reason I could make no satisfactory use of the ther-

mometer. I made up my mind, therefore, that I had to deal with inflammatory croup excited by catarrh; that doubtless false membrane already existed; and that, to save the child's life, tracheotomy would probably become necessary. Emetics were badly borne; but I ventured, in consequence of the presence of bronchopneumonia, to try five-minim doses of antimonial and ipecacuanha wines.

On Monday morning, January 10th, I found that even those small doses caused so much sickness and faintness that they were discontinued, and small doses of calomel were prescribed, in addition to mercurial inunction. The breathing was now very difficult and markedly diaphragmatic, whilst the intercostal spaces were depressed at each inspiration; the cough was dry, harsh, and husky; the voice thin and weak; the respiratory murmur feeble on the right side and accompanied with fine rhonchus and dulness on percussion. Late at night, Mr. Pollock arrived, and the operation of tracheotomy was anxiously discussed by him, Dr. Jervis (who had also returned), and myself. Just lately, there were some large moist *râles* to be heard in the trachea and larger bronchi, so it was thought we ought to give the child a chance of being saved the operation until the morning. Early the following morning, we found things just as they were, except that the trachea and bronchi had been cleared of their loose mucus. The child had not slept, but had suffered much from dyspnoea, and was constantly throwing itself about. It was put under the influence of a mixture of chloroform and ether; and at 9 A.M. the operation was leisurely performed by Mr. Pollock. Veins were pulled aside, and certainly less than thirty minims of blood were lost.

On the 12th, at 10 A.M., the child was comfortable and doing well, and took its food without difficulty. The skin was cool; respiration 40, pulse 160. 7 P.M.—He was sleeping quietly. Respiration 50; pulse 130, very weak. He coughed less and took but little food. He was ordered to take a teaspoonful of brandy every four hours.

January 13th. At 10 A.M., the child had taken its brandy and food well, and was much better in every respect. Pulse 140; skin cool.

January 14th. It was found that the child had taken two drachms of brandy every two hours, and was evidently slightly intoxicated, and had now been sick three times. It had had a dark greenish motion. The inunction was stopped, and the brandy was diminished to thirty minims. As dried mucus collected in the tube, the flannel was kept more moist.

January 15th and 16th. All went well, with the exception of a vomiting attack. Skin cool; pulse 124; respirations 40. He coughed more, and brought up a quantity of sticky brownish muco-purulent secretion. The right lung was much clearer; the *râles* were to be heard, but no tubular breathing.

On the 17th, 18th, and 19th, he was doing well. He looked pale and weak; coughed less. He took egg and bread and butter.

On the 20th, the tube was removed; but almost no air passed by the larynx. Violent coughing and struggling commenced. The child could get no air by the wound or larynx; for during inspiration the sides of the wound were sucked together. On expiration, false membrane was seen flapping through the wound. A fenestrated tube was immediately inserted, but with some difficulty, in consequence of its sides (it was a bivalve) catching on the sides of the trachea, pressing it backwards, and thus nearly suffocating the child. During the violent coughing and struggling, much muco-pus was expectorated. In the evening, the child was quietly asleep, breathing almost entirely by the nose. Respirations 32.

On the 22nd, the child seemed very uneasy. Although there was no dyspnoea, the tube was found to be stopped up with pus; it was removed, and its removal was followed by about half a drachm of pus and a little blood.* No air passed by the wound.

January 25th. The wound was healed; the child convalescent. The urine was examined for albumen, but there was none.

Neither in these cases, nor in two other successful cases of tracheotomy for membranous croup which I formerly brought before this society, was there any secondary paralysis.

If I might venture, gentlemen, to offer a suggestion, I would say that it would be desirable as much as possible that the discussion, which I hope will result from the reading of the foregoing cases, should be divided into two portions: 1. Whether they were cases excited by the specific contagium of diphtheria, or not; and 2. The merits of the operation of tracheotomy. I would more especially draw your attention to the first portion, which is almost daily the subject of our anxious consideration, and upon which we have to take prompt and decided action, resulting in good or evil of the utmost gravity, according as that action is right or wrong. In reference to this, I

* It is probable that this tube had not been in the trachea, but in the cellular tissue in front, and that what air did come by the tube was by the fenestra.

would just remark upon the great danger of stamping everything as diphtheria where there is found fibrinous exudation. We know that it is found as a result of scald from hot water, and I have seen a throat after the removal of tonsils that I could not have distinguished from diphtheria.

Three years ago, my coachman lost his voice, and I found a thick tough exudation over the whole of the posterior wall of the larynx. There had been bad measles in the house in which he lodged, and bad drainage. There was no glandular enlargement or puffing of the cellular tissue. I believed it to be diphtheria, and treated it as such; but it was bad for many weeks, and only yielded to iodide of potassium or warm weather. There was *no history whatever of syphilis*. The next winter, it was just in the same condition; and this year again it was like the two preceding winters, although each time he was living in different houses and under different circumstances, and no other members of his family had suffered in a similar manner. My coachman attributed it, and I now think with reason, to the exposure to cold, as he is never in any way ill excepting in the bad weather of winter.

In bad scarlatina, in bad measles, and at the end of many exhausting diseases, membranous exudations are to be found; and I think it very probable that of late years many cases have been attributed to the specific contagion of diphtheria, which really owned no such origin.

MALARIAL FEVER AT ROME.

By LAUHLAN AITKEN, M.D., Rome.

THE existence of a malarial element in the climate of Rome is so generally admitted, that it is quite unnecessary to enter into the history of the subject, to point out the foci of infection in the Campagna, to discuss the different effects of the poison, or even to mention the many varieties of malarial fever which authors describe. In this paper, I shall only deal with the effects of the malaria on unacclimatised strangers, English or American, who have been under my care. So dormant is the poison during the winter and spring months, that acclimatised members of the nationalities mentioned, who can afford to live well and to reside in a healthy quarter, are, so far as my six years' experience goes, never affected; but visitors, who are careless of consequences and heedless of the ordinary warnings, often overwork themselves in sight-seeing, become heated in the sun, and stand for hours in museums, galleries, and churches, exhausting their nervous systems, or expose themselves more directly on the Campagna, or even within the walls of the city, to a chill which results in an attack of so-called Roman fever. What this fever is the residents are often very puzzled to know, as strangers include under the generic term all kinds of ailments from a simple cold to a severe enteric fever; and it is sufficient to fall ill in Rome to be credited everywhere with being a victim of that vague disease. The result is naturally a wide divergence of views on the subject, and an alarm proportionate to the amount of confusion created. *Omne ignotum pro terribili* is the idea of all who speak of Roman fever.

That the malarial poison does produce, more particularly in the unacclimatised, a type of fever which can scarcely be classed under any such heading as intermittent or remittent, is certain. To this form the name "subcontinuous typhoid" has been given by Professor Baccelli of Rome. The term, fortunately, has not as yet become widely known; for, though the type of fever may be regarded as subcontinuous, yet, in whatever sense we take the word typhoid, the epithet is misapplied. If we regard it as used to express the group of symptoms called typhoid, or the typhoid state, my experience contradicts such an use, as none of the forty-five cases I have treated throughout have presented those symptoms which British physicians at least class under this heading. On the other hand, it is evident that Baccelli does not look on this fever as the product of the union of the malarial poison with that of decomposing excrementitious matter, as he carefully distinguishes his subcontinued typhoid from true ileo-typhus (enteric fever). Indeed, Baccelli seems to contradict himself in his lectures on the subject, as in one part he says that "the two morbid entities—the typhoid and malarial poisons—each losing its autonomy, become so fused together, but with the malarial predominating, that they give rise to the special type—subcontinuous typhoid"; while in other pages of the same pamphlet he asserts that "the subcontinuous typhoid is a fever resulting from malarial infection, pernicious as to type"; and again, "that, when a subcontinuous typhoid breaks out, every other morbid element, rheumatic, bilious, or typhoid, becomes subordinate to the malarial". It is, therefore, difficult to determine in what exact sense Baccelli uses the term typhoid; and I prefer to regard those

fevers as truly malarial, though presenting a remarkably continued course.* I am far from denying the occurrence of the combined type of fever which may appropriately be called typho-malarial. Indeed, I have treated such cases; but, as might be expected, the more severe poison very soon predominates, and the bowel-symptoms and eruption make the diagnosis of an enteric fever clear.

Etiology.—The fever to be described results, then, from malarial poisoning; the predisposing causes being, in the cases I have seen, unacclimatisation; lowered vitality from anxiety of mind, from chronic diseases, or from previous acute complaints, particularly the malarial; pregnancy and the puerperal state; or a combination of some of these; while the exciting cause in every instance I have known has been a direct chill when the body was overheated, or its power of resisting cold diminished by any such causes as have just been mentioned, or by exposure during the night, when the heat of the body is naturally lower. A few lines from my case-books will illustrate this.

E. P., a young lady aged 22, goes to an evening party after a fatiguing day's sight-seeing; dances until well on in the morning; stands, when perspiring, at the door awaiting her carriage; gets a chill, and the next day is laid up.

J. G., a gentleman aged 28, has a hard day's work after the hours on the Campagna, arrives much overheated at one of the gates after sunset, dismounts there, and drives to his hotel in an open cab without putting on a greatcoat. Two days afterwards, he shivers, and he has a somewhat prolonged fever.

S. S., a young lady aged 17, just convalescing from bronchitis, sits at an open window one cold afternoon in December, gets chilled, and has the most severe attack yet treated by me.

The Rev. Mr. D., aged 30, reading with some Oxford students, has one of the headaches to which he is liable; and, after a long heating walk to get rid of it, he sits down about sunset on the grass outside one of the gates, and gets what proves to be a mild attack.

D. W., aged 19, a young gentleman, rising one morning in December with a stitch in his side—dry pleurisy it turns out to be—takes his cold bath as usual, does not react at all, and has also a mild attack, which masks the pleuritic symptoms entirely for more than ten days.

An exactly similar account of the origin of the complaint might be given in nearly all the forty to fifty cases mentioned.

It is certain that the young are much oftener the victims than those advanced in life, which partly arises from their greater imprudence, and partly from their greater predisposition to contract fevers. So much is this the case that, though quite as many, if not more, elderly people come to Rome, yet among my cases four only have been over forty years of age. So far as sex is concerned, the numbers have been pretty equal; and the severity of the attack has apparently depended more on the previous state of the patient than on the fact of the chill having occurred within or without the walls of the city.

Incubation.—This could not be determined in every case; but, in nearly all in which the time of getting the chill was definitely fixed, it did not extend over two or three days. In several, the first symptom—more or less decided shivering—began within a few hours. In the young lady mentioned, in whom the attack followed a bronchitis, the temperature was 102.2 deg. within seven hours from the time of the chill. In another young lady, the shivering and headache began within a few hours from the time at which she had felt cold when sitting in a draught at an afternoon tea. In one gentleman (over forty), the incubation-stage seemed much longer. He was ill for about eight days with indefinite symptoms; loss of appetite; furred tongue; urine laden with lithates; evening headache and disturbed sleep, but no rise of temperature—pointing, as I supposed, to functional liver-disorder, but quite unrelieved by the treatment adopted; when he was sent to Albano, where he became worse instead of better, and from which he returned after a few days with a well-developed attack. At what exact period of his stay at Albano, the fever first showed itself, is uncertain. It may be objected, that the symptoms previous to the appearance of the fever did not necessarily constitute the incubation-stage; but I incline to think they did, from the non-specific treatment adopted doing no good at all. In the other three cases of elderly people being affected, I was unable to fix the date of the chill. Indeed, two of the patients had been under the care of other physicians before I was sent for; and in the fourth I could not definitely determine the length of the incubation, which probably varies with the age and power of resistance of the patient, as well as with the dose of the poison. General languor, an inclination to sigh and yawn, insomnia, aching pains in the muscles, and loss of appetite, are symptoms common to the incubation-period of all forms of fever.

* Although venturing to differ from Professor Baccelli as to the name of the fever, I would here express my appreciation of the great value of his communications on the subject.