and under lip had just become involved in the tremor. Other symptoms, marking the case out clearly as one of paralysis agitans, were present. These were, briefly, profuse perspirations, characteristic position, restlessness necessitating getting up and sitting down again every few minutes, fixed and expressionless aspect of the face, power of control of the movements for a very short time by a strong effort of the will, and stiffness of the muscles at the back of the neck with marked hindrance to both passive and active bending back of the head. It is needless to say that the character of the tremor was typical. The patellar reflex was present on both sides, that on the left being more marked than that on the right. Grasset[5] also briefly records a case of what he calls "hémiparalysis agitans post-hémiplégique," in which there was tremor at rest in the affected side, sensation of heat, and "soldering" of the head and neck. This is, however, not a typical case, and no indication is given as to the period which had elapsed between the onset of the hemiplegia and the time of the patient's coming under Grasset's observation. Excluding this case as doubtful, we have here three cases, including my own, in which true paralysis agitans supervened immediately upon (presumably) cerebral hæmorrhage, and this renders the acceptation of a theory of coincidence infinitely more difficult. We seem to be forced to the conclusion that the cerebral lesion stood in a causal relation to the subsequent phenomena. Granting, then, that these are true cases of paralysis agitans, and that the hemiplegia acted as the immediate cause of the development of the malady, are we justified in concluding that the tremor and other symptoms were due to the anatomical site of the lesion? Auerbach answers this question in the affirmative, arguing analogically from those cases already referred to in which post-hemiplegic tremor resembling that of paralysis agitans was found post mortem to be associated with hæmorrhages in the optic thalamus, nucleus caudatus, and posterior part of the internal capsule. But he omits to mention that in none of these cases was there any spread to the opposite side of the body, and in none of them were there symptoms of paralysis agitans properly so called. Neither in his case nor in mine, at the time when the patients came under observation, was there any hemianæsthesia, which had been present in the subjects of the necropsies. He admits this to be a difficulty, and suggests as the possible explanation that the lesion in his case may have been in the pons, and considers that the close proximity of the motor tracts of both sides in this situation may have accounted for the subsequent spread of the tremor to the opposite side. In the absence of any post-mortem anatomical evidence we can only speculate as to what is most probable, and I must confess that this explanation of Auerbach's is unsatisfactory to my mind. I think it much more likely that the *rôle* played by the cerebral lesion in these cases was simply that of a determining cause in subjects already predisposed. There are many cases on record in which the shaking followed immediately upon a shock of some kind, physical or emotional. Even a slight injury often seems to play the part of the immediately determining cause of the tremor, or, at all events, it determines the part in which the earliest symptoms shall manifest themselves. Thus Handfield Jones[6] had a case in which the right arm began to shake after the patient had been knocked down by a blow on the right shoulder. Charcot[7] records several similar cases. In one the tremor began in the left leg after a bruise to the thigh. At first occasional, this became permanent, and finally extended to the other limbs, whilst other symptoms of paralysis agitans developed. In another case the tremor followed a sprain of the left ankle before the pain and swelling had subsided; it began in the left foot and spread to the other limbs, other symptoms being well marked. I have myself had a case in which the patient had somewhat severely strained the shoulder of the arm first affected shortly before the tremor began. But it is useless to multiply instances of a well-known fact, and I merely mention here cases in which mental shock, such as the sudden violent death of a friend,[8] or even the loss of a five-pound note,[9] have been sufficient to start the tremor which has subsequently proved to be that of paralysis agitans. Such comparatively slight shocks being capable of acting in this way, it is not surprising that the profound shock of a cerebral hæmorrhage, wherever its situation, should, given the necessary predisposition, bring about the same series of phenomena. And just as in an ordinary person a blow on a limb or a mental shock is not followed by these consequences, so in the ordinary cases of hemiplegia no such condition results, even in those where, from the site of the lesion, a tremor resembling that of paralysis agitans is set up. The commencement of the manifestation of the disease in the paralysed limbs also corresponds to the history of cases following external trauma, and it is not surprising that the parts controlled by the cortex of the damaged hemisphere should be first affected. I do not propose to enter here into the problem of the pathology of paralysis agitans, but merely suggest that such cases as these which follow immediately upon an internal or external trauma support the view that the essential change is a nutritional one of the higher (cortical) motor centres; for it is hard to understand why, if any of the various forms of gross organic lesion which have been described by various observers be the essential element in the production of the symptoms, they should remain inert as causes until called into play by such trifling causes as those mentioned above; whilst, on the other hand, it is quite conceivable that, given an already lowered condition of nutrition of the cortical centres, a comparatively slight cause might still further lessen their control over the lower motor centres, and thus allow of the first manifestation of the tremor. A cerebral hæmorrhage would be particularly likely to do this on the side of the brain on which it occurred, and this, I suggest, is the explanation of these cases of post-hemiplegic paralysis agitans.

Manchester-square, W.

[5] Progrès Médical, 1880, No. 46.
[6] Brit. Med. Jour., 1873, vol. i., p. 221.
[7] Leçons sur les Maladies du Système Nerveux, 4th edition, tome i., p. 185.
[8] Charcot: Loc. cit., p. 183.
[9] Atkins: Journal of Mental Science, 1881–82, p. 535.

CASE OF ORDINARY OBLIQUE FRACTURE OF TIBIA AND FIBULA,

COMPLICATED WITH COMMINUTED FRACTURE OF THE NECK OF THE FIBULA; EXCELLENT RECOVERY FOR FIVE WEEKS; THEN EVIDENCE OF HÆMORRHAGE AND PROBABLE INJURY TO ANTERIOR TIBIAL ARTERY; AMPUTATION OF THIGH; RECOVERY.

BY JOHN B. FRY, L.R.C.P. LOND., M.R.C.S. ENG.

A MAN aged sixty-seven years, while unloading hay from a cart, on March 2nd, 1894, stepped backward on to the loose tailboard, which tilted, bringing him to the ground with considerable violence. I found an oblique fracture of the tibia and fibula at the junction of the middle and lower thirds, with much displacement, the upper fragment of the tibia projecting nearly through the skin. There were considerable ecchymosis and bruising of the skin, but only over the seat of fracture. The man was at once sent to the Cottage Hospital, Thames Ditton, where I put the leg in Cline's splints. With the exception of a good deal of trouble caused by extravasation of blood and formation of bullæ, which left superficial sores, he apparently made an excellent recovery. The leg was placed in a water-glass bandage, and he left the hospital at the end of five weeks free from all pain and swelling and able to move about by the aid of crutches. Ten days later he complained of great pain in the upper part of the front of the leg, which, however, lasted only half an hour. On the following day, he having had another attack of pain of the same character before I saw him, I cut off the bandage and sent him to the hospital. A swelling was now noticed on the external aspect of the leg between the tibia and fibula, and slightly below the level of the tubercle of the tibia. It had a circumscribed glazed appearance, and was of the size of half an orange. The patient complained of no pain, and no pulsation could be detected, but as there was well-marked fluctuation it was thought to be a superficial abscess. On opening it pus escaped, followed by small clots, and copious bleeding occurred which could only be controlled by pressure of the femoral artery. With the assistance of Dr. Lyons and Dr. Senior I amputated the leg above the seat of the swelling. The whole of the parts were found ploughed up and macerated by effused blood. The destruction of tissue being so evident, it was thought prudent to amputate higher up; accordingly I adopted the

circular method at the junction of the middle and lower thirds of the thigh. The greater part of the wound healed by first intention, and in a short time the whole had granulated up, and the man left the hospital on May 30th. Examination of the amputated leg revealed a severe comminuted fracture of the neck of the fibula in which there was not the slightest attempt at repair, the fragments lying loose in the macerated fibres of the surrounding muscular tissue. The posterior tibial vessels were uninjured and free from atheroma, but on following the anterior vessels it was at once seen that they were the source of the bleeding, although owing to the disorganised condition of the uscular tissue they were traced with difficulty. In close proximity to them were several small splinters from the fibula, but it was quite impossible to determine which fragment had injured the vessels. There was a large extravasation of blood in front of the interosseous membrane lying amongst the degenerated and destroyed fibres of the tibialus anticus and the extensor muscles of the toes. The hæmorrhage had also extended backwards in the muscles under the gastrocnemius and soleus, but had not destroyed the tissues to the same extent as in front. It did not extend as far as the fracture of the tibia and fibula, which was found firmly united, but had almost reached the limits of the popliteal space above.

Remarks.—The case at first appeared to be a simple one of fracture of tibia and fibula, and it was not until nearly seven weeks after the accident that any further symptoms developed. The injury at the neck of the fibula took place at the same time as the fracture below, but there may be some doubt as to whether the anterior tibial vessels were injured at the same time, or the artery perforated or ulcerated by the movements of the patient after leaving the hospital.

Esher.

THE TRANSMISSION OF DIPHTHERIA BY NON-SUFFERERS.

BY HERBERT PECK, L.R.C.P. EDIN., D.P.H. CAMB.,
MEDICAL OFFICER OF HEALTH TO THE ORMSKIRK RURAL DISTRICT COUNCIL.

THE following series of cases is of interest as showing the possibility of the conveyance of the diphtheria poison by persons who do not themselves suffer from the disease.

HOUSE A.

CASE 1.—A man aged forty-five years began to be ill on Oct 4th, 1894. The case was typical and was followed by double "drop-wrist." No other person in this house suffered from sore throat.

HOUSE B.

This is half-a-mile distant from House A. There was no communication between the houses till Dec. 26th, when the occupier began to work for the preceding patient.

CASE 2.—A child aged seven years and a half had a sore-throat on or about Jan. 2nd, 1895. The case was a typical one, followed by paralysis of both legs. The patient died from cardiac syncope.

CASE 3.—A child aged four years began to be ill on Feb. 6th. This was a typical case and resulted in death from asphyxia.

CASE 4.—A child aged twelve years had a sore-throat on Feb. 16th. The case was typical and was followed by dimness of sight and strabismus. Bacteriological examination of cultures from the throat showed the presence of large numbers of diphtheria bacilli.

HOUSE C.

This farmhouse stands on a peat-moss and is one mile and a quarter distant from Houses A and B.

CASE 5.—A youth aged eighteen years, brother of the last three patients, worked here from Jan. 15th. His mother stated that he was at home for a few minutes on the days his sisters were buried, but at no other time. He himself admitted having been home on each Sunday to change his shirt, which he did in the room occupied by the patients. He stated that he did not suffer from sore-throat at any time, but bacteriological examination showed typical diphtheria bacilli in large numbers to be present in his throat. There is no history of his ever having had diphtheria.

CASE 6.—A youth aged eighteen years began to be ill on Feb. 26th. He worked on the same farm as the last youth and slept with him. He went home to House D on the 27th, being then "full of cooth" and suffering from sore-throat, and unable to work. Typical diphtheria bacilli were present in great abundance on March 15th.

HOUSE D.

This is a mile and a half distant from House C, two miles from House B, and two miles and a half from House A.

CASE 7.—The patient was a boy aged thirteen years, who slept with the last patient, his brother. He came home from chapel on March 3rd, complaining of sore-throat. On the 8th he was seen for the first time by his medical attendant, and was then dying. No bacteriological examination was made.

CASE 8.—A child aged three years, sister of the last two patients, began to be ill on March 12th. The case was a typical one, and an abundance of typical diphtheria bacilli was found on bacteriological examination.

Remarks.—House B was probably infected by the father of the family, who himself did not suffer from the disease. His son (Case 5) did not suffer from the disease, though he was proved to have its germs in his throat, but he was the means by which it was carried to House C, whence it was taken to House D.

Ormskirk, Lancashire.

ON SO-CALLED GOUTY PSORIASIS.

BY T. FREDERICK PEARSE, M.D. BRUX., F.R.C.S. ENG.

PSORIASIS occurring for the first time in or after middle life presents distinctive characters and requires different treatment to psoriasis occurring in the young. Most writers divide psoriasis into syphilitic and non-syphilitic. Leaving aside syphilitic eruptions of a scaly nature (to which the term "psoriasis" is incorrectly applied for a scaly syphilide), I wish to draw attention to some differences in the cases of what are termed true psoriasis. There are roughly two chief varieties: (1) the psoriasis of the young, and (2) the psoriasis of the gouty. This distinction was first pointed out by Liveing. The former variety is characterised by occurring generally before puberty, by attacking the extensor surface of the limbs, by the scales being numerous and thick, by the almost entire absence of itching, by the absence of any eczematous eruption or history of it, by being symmetrical, and by the effect of treatment. The latter variety never commences in childhood and rarely before middle age; there is generally a history of gout in the patient or in his family. It does not affect the extensor more than the flexor surface; the itching is considerable, the patches are thin, not very scaly, and the scales are small and easily rubbed off. The eruption is scattered irregularly over the body and may be said to show a disregard for symmetry. Moreover, it has a great tendency to be complicated with undoubted eczema, and sometimes assumes an inflammatory aspect almost passing into eczema. It is often made worse by the ordinary applications for psoriasis. These patients bear arsenic badly, and stimulating fluids and highly seasoned articles of food, as well as sugar, affect the eruption unfavourably. This form in some cases alternates with acute attacks of gout, and in others seems to be suspended by them. At times the eruption develops very rapidly, even in a night. Under "gouty" remedies it often rapidly disappears—much more rapidly than psoriasis ever does in the young. The treatment most successful is the free use of liquor potassæ internally, with perhaps the addition of quinine. The addition of colchicum, however, is sometimes of great service. These patients require the ordinary routine and dietary for gout. They generally require to drink more water, and alkaline waters taken freely are especially to be advised. As the skin acts feebly in many gouty persons, bathing frequently is desirable, and warm baths may be recommended. The diet should be plain, and fish or white meat should be preferred to beef or mutton. Plenty of fruit and vegetables are desirable, but the more complete the avoidance of wine, beer, or spirits the better. Clinically, this variety is closely allied to dry eczema. Its appearance is similar and the itching in both cases is marked. Contrary to true psoriasis, it is not symmetrical and does not attack the same parts. What is its pathology? Is it a true psoriasis occurring in the gouty and modified in its clinical characters by the gouty state, or does gout have a psoriasis-like eruption