before this baby's birth, from attacks of urticaria, local oedema, and hay fever after eating acid fruits or bananas, and had also once had severe asthmatic symptoms. One sister was subject to urticaria after eating bananas, and had had an attack of acute rheumatism. The other two sisters had shown no sign of idiosyncrasy to food of any kind.

During the first few months of life the baby, who was on the breast, had on several occasions had a meal of cow's milk without any bad effect. When seven months old she was given a little rice pudding with cow's milk, which was followed at once by urticaria and asthma. From that time on she had violent symptoms whenever she was given cow's or goat's milk in any form or raw white of egg. Half a teaspoonful of milk was sufficient to bring on an attack within a few minutes. The breathing became difficult and there was sneezing and coughing, with running of the eyes, rhonchi in the chest, and an urticarial rash. The baby seemed seriously ill and collapsed, and she remained so for two or three days. Once while she was on the breast she developed similar symptoms after her mother had eaten a banana, although the mother herself on that occasion was not affected. Cow's milk diluted in various ways, Glaxo, Cow and Gate milk, Nestlé's, Allenbury's, Benger's and other "infant foods," and also goat's milk, were all found to affect the child in a similar way. Peptonized milk did so also, but to a rather less severe degree. Raw white of egg caused the same symptoms, but the child could take boiled and fried eggs with impunity. With raw egg albumin she had also had diarrhoea and vomiting.

The baby was small but apparently healthy, and showed no physical signs of disease. She was fed, with some difficulty, on patent groats with water, boiled eggs, potatoes and other vegetables, fish, chicken, minced beef, gravy, and so on. When about eighteen months old she began to tolerate small quantities of cow's milk, and she has since, I understand, gradually improved.

The other type of case, though not often met with, is less uncommon, and many instances of it have been described.⁵ The symptoms may set in within fifteen minutes, but are frequently delayed for one to five hours after the milk has been taken. They may be caused by even a few drops of it. The infant turns pale, drowsy, and uncomfortable, refuses food, vomits and has diarrhoea, with some degree of fever, or sometimes a subnormal temperature and collapse; and occasionally there is glycosuria or albuminuria. In these cases there is generally no sign of urticaria or of respiratory distress. The child rapidly recovers when put on breast milk only. Often the idiosyncrasy does not show itself for some weeks after birth; and in many cases it diminishes or ceases in the course of a few months, although in others it may last into later childhood. If the children are carefully looked after the condition is rarely fatal.

It is to be noted that none of my cases of "idiopathic convulsions," so far as I have been able to ascertain, has shown any subsequent tendency to convulsions, to food idiosyncrasy, to asthma, or to skin eruptions. Should it be shown that the above hypothetical explanation of these cases is possibly correct, the further question naturally arises as to whether, when symptoms of poisoning from cow's milk, white of egg, oatmeal, or other common foods occur in young children, they might not be stopped permanently if the patient were subjected to a thorough chloralozation for some days, while continuing the offending diet; and even whether a tendency to asthma and eczema beginning in infancy might not be permanently arrested in a similar way.

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AN international congress for protection of mothers and infants will be held in Paris from July 6th to July 8th, 1922, when the following subjects will be discussed: (1) Protection of mothers before and during confinement; (2) protection of mothers during lactation; (3) protection of the infant separated from its mother; (4) assistance and instruction. Applications for membership should be made to La Ligue contre la Mortalité Infantile, 49, Rue de Miromesnil, Paris.

BILATERAL RIGIDITY IN MIDDLE MENINGEAL HAEMORRHAGE.

BY

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THE clinical picture presented by middle meningeal haemorrhage has received during the past forty years the attention of many skilled observers. Their labours have been so fruitful that in its most typical form the middle meningeal syndrome has the distinction of being one of the most clearly defined in the whole range of medicine. As is usually the case when the picture of a settled type of case becomes clear and easily recognized, the surgeon and the physician have an opportunity of noting with greater profit the variations from type which individual cases present. The frequency of the "free" or "lucid interval," the reasons for its absence, the occurrence and significance of ocular palsies and deviations, of paralyzes of the limbs, of aphasia, and the incidence of Jacksonian fits, have all received their measure of investigation and have been allotted definite values.

Two cases recently under my observation presented certain features to which attention has not hitherto been specifically drawn in this connexion; in place of monoplegia or hemiplegia there was a very pronounced degree of general muscular rigidity. Here and there in the literature of middle meningeal haemorrhage a case is recorded in which it was noted that the limbs were rigid.

A bare half-dozen are to be found amongst Wiesmann's collection of 257 cases.³ Further examples occur in Duret's comprehensive monograph,⁴ but these include severe fractures of the skull, with cerebral laceration and intradural haemorrhage. These references are so incomplete that one gets only a hazy impression of the precise nature of the condition; the authors have simply recorded the fact in a few words without further comment.

In the histories which follow I have attempted to give a fuller account of the appearance and posture of the patient. The muscular fixation was seen to involve the trunk and all four limbs, and was such that passive alteration of position was difficult, and at times impossible, without the employment of considerable force. The clinical picture is so striking that, once seen, it is not likely to be forgotten. It seems to me to be important that attention should be directed to these generalized rigidities, and that the processes attending them should be accurately observed. Kinnier Wilson¹ has recently described a similar rigidity appearing in pathological circumstances other than those here described, so that one cannot say that the essential rigidity seen in my own two cases is in any way pathognomonic of middle meningeal haemorrhage. It is, nevertheless, important to note that hypertonicity and a peculiar attitude of body have been observed in these cases of meningeal haemorrhage no less than in the cases described by Wilson, since the one throws a ray of light, if an oblique one, upon the other.

CASE I.—Fall on Head: Linear Fracture in Left Temporal Fossa: Middle Meningeal Haemorrhage: Bilateral Rigidity of Trunk and Limbs: Operation: Death: Necropsy.

On March 25th, 1921, J. D., male, aged 37, a window cleaner by trade, was at work cleaning some first story windows in a stone courtyard. He lost his balance and fell head foremost to the ground. Being temporarily stunned he lay for a minute or two and then got up and walked home, a distance of a couple of hundred yards. He sat down for a little while, telling his wife that he felt rather shaken, but soon declared that he would go and fetch his ladder and pail. This he did, but on arriving home a second time seemed decidedly worse, and went and laid himself down. He went to sleep, but when his wife tried to rouse him some little time later she found it impossible to awaken him. From this time (perhaps one and a half hours after the accident) onwards he remained unconscious. Later in the evening he was admitted to the Salford Royal Hospital under my colleague Mr. Garnett Wright, who kindly asked me to see the case. On admission he was unconscious, restless, moving arms and legs freely, and inclined to be noisy.

Symptoms and Physical Signs.

When seen by me, seven hours after the accident, the picture had changed. He lay flat upon his back, the arms rigid and extended by the sides. The hands were half-clenched and the forearms hyperpronated, so that the backs of the hands were facing the outer aspects of the thighs. The legs were stiffly extended and the toes pointed downwards, the ankles being

strongly plantar flexed and slightly inverted. The muscular rigidity was so great that considerable force was necessary to bend the limbs. Any attempt to alter the position in which the limbs lay seemed to bring on an excess of tonic rigidity, a fine tremor then appearing. The patient's head was turned sharply to the left, as were the eyes; the neck was rigid and the chin a little lifted, but no opisthotonos appeared. Cheyne-Stokes breathing was present, and at the height of the noisy, rattling respiration the tonus was exaggerated, the arms becoming even more pronated and the legs more rigid and adducted, the foot remaining in extreme plantar flexion. In the quiet periods of respiration the muscle tonus appeared slightly to diminish. There were no "tonic fits," only exaggeration of the already severe spasticity at the height of the Cheyne-Stokes breathing, and on moving a limb passively. The left pupil was widely dilated. It was practically impossible to test the tendon reflexes. Ankle clonus was present on both sides, but had a small amplitude and was exceedingly rapid. The plantar reflex gave rise to a sharp and very quick extension of the great toe on either side without flexion of the other toes. Oppenheim's, Gordon's, Craft's, and Chaddock's reflexes were absent. The abdominal muscles were rigid, but a slight response followed stroking on the right side, but not on the left. During the whole period of observation the patient made no voluntary movement of any kind. The pulse rate was 60.

Diagnosis.

In view of the characteristic history, the conjugate deviation of head and eyes to the left and the large immobile left pupil, together with the obvious signs of cerebral compression, there was no difficulty in diagnosing a left-sided haemorrhage.

Operation.

Operation was carried out forthwith. An intermuscular temporal exploration of the left meningeal field revealed a crack in the anterior part of the temporal fossa, and beneath this a huge extradural clot. The area of the clot was estimated at 12 cm. in diameter and nearly 4 cm. in depth at its thickest part. The clot lay over the left frontal lobe, and had extended forwards and upwards rather than backwards. The spurting middle meningeal artery was clearly seen, but was ligatured with some difficulty after extensive removal of bone. After the removal of the clot the brain failed to expand again although it pulsed freely, and when last seen only a slight elevation of the cortical surface had occurred. The dura was not opened. The wound was dried, drained, and sutured.

At the completion of the operation the rigidity was found to have disappeared, but the patient's general condition was desperate; the pulse had risen to 128 and the respiration was rapid and shallow. Death occurred three hours later.

Necropsy.

At autopsy no further point of any note was discovered. The brain was intact, no intracerebral haemorrhage was present, and indeed there was no trace of blood within the dura.

CASE II.—Fall on Head: Compound Depressed Fracture of Right Frontal Bone: Left Middle Meningeal Haemorrhage: Bilateral Extensor Rigidity of Trunk and Limbs: Operation: Death: Necropsy.

On May 12th, 1921, J. F., aged 26, male, fell head foremost off a roof 60 ft. high to the ground. On admission to the Salford Royal Hospital, within an hour of the accident, he was conscious and related the particulars of his fall. His temperature was then 97.5°, and his pulse 68. There was no paralysis; he could move all his limbs, and turned over on to his back at request. He was bleeding from the nose and from both ears, and presented a contused wound over the right frontal bone some 8 cm. long. An hour later he became drowsy and vomited, and shortly afterwards had a tonic seizure, his breathing at the same time becoming noisy. He was seen by me three hours later.

Symptoms and Physical Signs.

He was then deeply unconscious; the pupils, of medium size, did not react to light. He presented bilateral extensor rigidity of both legs, which were strongly adducted. The feet were in full plantar flexion, inversion, and supination. Every three or four minutes the tonic contraction of the limb became more pronounced, so that the employment of moderate force was insufficient to separate the thighs. In the intervals between the attacks the limbs were considerably less rigid, so that the legs could be pulled apart and the knees bent, but at no time was there flaccidity, at no time was there an absence of resistance. The left arm during the spasmodic attack was extended stiffly by the side in slight abduction, the wrist was strongly flexed and adducted. The fist was nearly, but not quite, closed and the thumb adducted across the palm within the fingers. The right arm presented a slightly different picture. It was held rigidly bent at the elbow at an angle of 130 degrees the wrist fully flexed and the hand as on the other side. During the tonic seizures both arms rotated inwards into a position of hyper-pronation so that the backs of the hands were directed towards the thighs. On the right side this was largely accomplished by an internal rotation at the shoulder-joint, the elbow remaining in partial flexion. On the left side, where the whole arm was stiffly held by the side, there was extension and internal rotation of the shoulder aided by pronation in the fore-arm. An attempt to interfere with the limbs during a tonic attack had little effect, and the muscular contraction was so strong

that great force was necessary to restrain the movement or correct the position assumed by its agency. As in the other case, the tonic attacks were closely bound up with a change in the rate and amplitude of respiration. At the height of the attack respiration was rapid, noisy, and deep. As far as one could see, the sequence of events was, first, a change in respiration; next, twitching of the fingers on both sides, which ceased in a few moments to give place to an immobile rigidity; thirdly, there occurred flexion of the wrist and then inversion of the arms, the legs and trunk at the same time stiffening into extension. During a few of the seizures the patient arched his back in opisthotonos, affecting particularly the lower part of the spine. The head was not seen to retract nor to rotate. There was no incontinence. Ankle clonus was rapid and sustained; plantar stimulation led to a bilateral great toe extension, which was very rapid on the right and slower on the left.

Operation.

I operated upon the patient under local anaesthesia. The necessary shaving of the head had been accompanied by an almost constant state of tonic rigidity, and the introduction of the needle for novocain-adrenaline infiltration caused another attack. It seemed as if this condition of extreme muscle tonus was the only method that he had of responding to a stimulus, and took the place of defensive reflex movements. The frontal wound was excised and a large horizontal depressed fracture exposed, the bone being driven in and jammed. The depressed portion was rapidly elevated and removed, over an area some 8 cm. by 4 cm. up to the mid-line. At the inner end of the wound a small piece of dark clot was seen overlying the anterior end of the superior longitudinal sinus. A small piece of ribbon gauze was placed over this, the wound washed well with flavine and the dura widely incised as clinical signs of compression had been so severe. Exploration of the right meningeal field revealed no haemorrhage, but a search with a silver brain spatula over the right orbital plate disclosed comminution with loose bone fragments within the dura. The wound was loosely sutured up, with the intention of performing a secondary suture in three or four days when the acute stage was passing.

At the completion of the operation the patient was flaccid, phonating with expiration, and seemed better. His pulse, which had risen to 120 at the commencement of operation, fell to 70 after opening the dura. It remained afterwards steady at 76. The noisy and stertorous breathing had ceased and the tonic seizures had passed away.

He did well for an hour or two, but his pulse rose again later in the evening, and he died seven hours after operation without again exhibiting rigidity.

Necropsy.

At necropsy an extension of the fracture was found over the left frontal bone, which was very badly damaged. To my great chagrin a large middle meningeal haemorrhage was present on this side. It was anterior in position, and indeed extended so far forward that its anterior end had actually been seen in the wound. In size it approximated to that of a man's fist, and lay over the frontal pole of the left hemisphere. The right orbital plate was broken to pieces, and there was a superficial laceration of the under surface of the corresponding frontal lobe. There was no abnormality elsewhere in the brain and but little blood free within the dura.

It has been seen that the lesions responsible for the occurrence of the rigidity in these cases were in both left-sided extradural haemorrhages of considerable size occupying Kroenlein's position 1.² In one case there were serious associated injuries of the right frontal pole. Both patients were profoundly unconscious, were in a state of severe cerebral compression, and exhibited Cheyne-Stokes breathing at the height of which the muscular rigidities were brought out at highest pitch.

I have described the tonic conditions in these cases in detail because the question of generalized muscular rigidities has gained considerably in importance from Kinnier Wilson's valuable contribution to the subject. In my own cases, so closely corresponding in their clinical pictures to his, the rigidities must have been dependent on changes in the intracranial circulation produced by the extravasations or injury. It is inconceivable that the rigidity could have been the result of a local irritation of a neural mechanism by the action of the clot on the immediately underlying cortex. I am led to the belief that the clinical syndrome depends on a relative deficiency or the reverse of the blood supply to certain regions of the brain, and that this releases lower neuronic levels. The relationship between the crises of rigidity and those of Cheyne-Stokes breathing bears out the circulatory theory of their origin. And it is only by the mediation of some great common factor such as the quantity and quality of blood supply and the variations in this supply, local and general, occasioned by lesions of varied types, that we can correlate the several dissimilar intracranial maladies capable of producing bilateral rigidity.

The term "decerebrate rigidity," though convenient, is apt to suggest that the lesion producing it must be situated about the mid-brain—that the rigidity in man is the outcome of a pathological process which is the counterpart of an actual section at the colliculi in animals. These cases of mine demonstrate clearly that the level is a physiological rather than an anatomical one, and I have refrained from applying the term "decerebrate" to the rigidities in order that this significance should not be obscured.

Why generalized rigidities should make their appearance in one case and not in another of very similar gross pathology is a delicate question. It is probable that the syndrome in man depends on a very fine circulatory balance, on a relative anoxaemia sufficient to suppress cortical control but not great enough to render inactive the causal mechanism in mid- and hind-brains. Generalized rigidity may thus never have an opportunity of arising in one case; it may be but a phase in another (as Kinnier Wilson points out); whilst in a third, where the balance is maintained for a period, the rigidities may border on chronicity.

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METATARSUS VARUS.

BY

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METATARSUS VARUS is a deformity which has long been familiar clinically to orthopaedic surgeons, but it is not described even in textbooks of orthopaedic surgery, and its essential pathology has not yet, I think, been recognized.

Whitman,¹ it is true, makes passing reference to "metatarsus varus" as an incomplete form of clubfoot (congenital talipes equino-varus) or simple talipes varus, and the treatment recommended is the same as that for the varus part of congenital clubfoot.

But metatarsus varus belongs to quite a different category from clubfoot or talipes varus. It is not a contracture deformity at all. It is an anatomical deficiency deformity, and is due to congenital absence of the internal cuneiform bone. It should therefore be classed not with talipes or other congenital contractures, but with those conditions in which deformity is incidental to gross structural defect—for example, congenital absence or deficiency of the tibia, fibula, radius, ulnar, vertebrae, etc.

Clinically, the posterior part of the foot presents a normal appearance. There is no limitation of movement at the ankle or subastragaloid joints. There may even be some eversion (valgus) of the foot at the subastragaloid joint. The anterior part of the foot is sharply adducted at the tarso-metatarsal joint, but there is no inversion of the sole. The patient, therefore, stands with the sole of the foot flat on the ground, but while the posterior part of the foot is straight, the anterior part is directed inwards or forwards and inwards. The great toe is usually adducted more than the other toes (hallux varus). There is a sharp kink, which can be both seen and felt, in the inner border of the foot, just behind the base of the first metatarsal bone. The patient can walk, but his gait is awkward, and the deformity is unsightly. So far as I know, the condition is always bilateral.

It should be noted that the displacement of the anterior part of the foot is entirely abnormal—it is not even, as in talipes, an exaggeration of a normal displacement. Adduction without inversion is not a physiological movement of the foot.

Radiography shows the adduction of the anterior part of the foot, but it does not reveal the essential defect in young children, because the cuneiform bones are not ossified at the age at which these cases usually come under observation. I have not had the opportunity of examining a patient who has grown up with this deformity.

In six feet (of three patients) that I have examined at operation the internal cuneiform bone was absent and represented by a flat disc of fibro-cartilage between the base of the first metatarsal bone and the scaphoid. The tendon of the tibialis anticus divided into two distinct slips, one of which was inserted into the fibro-cartilage, and the other into the base of the first metatarsal. It was not possible at operation to make out the condition of the middle cuneiform bone. The first metatarsal was shorter than normal and strongly adducted. The scaphoid was normal. In one case the neck of the astragalus appeared to be unduly long, and the head was bent downwards.

Treatment.

From the nature of the deformity it is obvious that the treatment suitable for talipes varus or the varus part of talipes equino-varus is quite inappropriate for metatarsus varus, and the general experience of orthopaedic surgeons is that the results of such treatment are unsatisfactory. It is true that a considerable amount of apparent correction can be brought about by manipulation and fixing the foot in plaster. But the correction thus obtained is not permanent, and the deformity always relapses more or less completely.

Anatomically the inner border of the foot is too short, owing to absence of the internal cuneiform bone, while the outer border is relatively too long. Permanent correction, therefore, can only be obtained either by lengthening the inner border or by shortening the outer. Two procedures, therefore, suggest themselves: (1) Separation of the first metatarsal and scaphoid bones, and the insertion of a graft to keep them apart; and (2) excision of bone—for example, the cuboid—from the outer border of the foot.

I have tried both these operations with, in each case, immediate correction of the deformity, but they are too recent to speak yet of the ultimate results. Theoretically, to replace the missing bone by a graft, and thus to restore the inner border of the foot to its normal length, would appear to be the ideal method of correcting the deformity. But I doubt very much whether this can be attained in practice. In the first place, the results of bone grafting, excellent as they are in very many conditions, are notoriously uncertain in cases of congenital bone deficiency. In the second place, if a fixed graft is successfully inserted into the first metatarsal and the scaphoid, and perhaps also the astragalus, it is likely that the rigid foot produced will eventually prove to be a greater disability than the original deformity.

For these reasons I think that the better practice is to remove bone from the outer side of the foot—a practice, be it noted, that is absolutely contraindicated in cases of clubfoot in young children. By removal of the cuboid the outer border of the foot is shortened and the metatarsus is abducted and brought into line with the posterior part of the foot. This, so far as my observations go, gives the best correction of the deformity with the least disturbance of function.

REFERENCE.

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TWO CASES OF PERNICIOUS ANAEMIA
WITH INFECTIVE FOCI IN THE
ALIMENTARY TRACT.

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MUCH has been written on the etiology of pernicious anaemia, and the clinical evidence points to there being an invasion of some part of the alimentary tract by pathogenic organisms in a large percentage of cases. In the two following cases there appears to be very definite evidence of either past or present pathological changes in the alimentary tract.

CASE I.

The first case was that of A. B., aged 38, a trained nurse, who came under our care on April 8th, 1921. The history of the illness was that in July, 1920, she felt very "run down," and had to give up nursing. About a month later she noticed that