of locus as well as a differential function for doing what is needed at the new point. In these post-encephalitic patients, when they are not fixed at a point, there seems to be no derangement of their ability to think or feel or look about. The disorder does not seem to be within those simpler representations of function which we find damaged by local lesions of the cerebral cortex. It seems to be a disorder with the patterns of action intact and the patient at times being unable to use them. This seems to be a disorder of re-integration of specific representations of function at a level where they are re-represented within the neural action that attends conscious adaptation. This would be (according to Jackson, 1931) at the highest neurological level.

The disorder reminds one of a flow of traffic with a signal system for stop and go. When the regulatory mechanism goes awry, the red light stays on with the green light off, and no change of flow to new positions occurs. Now the loss of the capacity to change is a disorder of timing for change. There seems to be a disorder of those neural mechanisms essential to inhibit one pattern of neural action and excite a new pattern of neural action. This loss of timing mechanisms may involve voluntary or involuntary function. The loss may be very discrete and limited to ocular or mental or part of the mental life only, or it can be multiple in both congruous and incongruous combinations. This is a loss of very specific and organized function. It suggests an effect of the encephalitic process upon some timing mechanisms so arranged as to allow highly selective damage. Such a delicate selection requires an arrangement of timing mechanisms within the central nervous system that provides for discrete and specific physiological and, possibly, anatomical organization.

With the oculogyric crises and the mental paroxysms reduced to a common type of disordered timing mechanisms at a neurophysiological level, it is difficult to proceed further since our knowledge of the physiology for the timing of action is so scanty.

Beevor (1904) followed Jackson in his contention that cortical motor representations intrinsically contained the process of timing for those changes which permit a sequence of muscular action to change the position of a limb around a joint. But in the post-encephalitic patients it seems that the defect is of a higher order where the need is to change from action at one point to action at a new point. Is this simply a more complex ordering of intrinsic timing mechanisms within cortical re-representations? Or could it be that timing mechanisms are arranged in some way apart from representations of specific function so that specific neural action can be aroused or inhibited for integrated or differentiated function? This might be possible if there were anatomical arrangements for the special function of timing with both diffuse and specific connexions to other parts of the brain. The new knowledge of the reticular substance illustrates such an anatomical arrangement with diffuse and specific projection pathways for general or particular arousal and inhibition. One is left wondering if timing mechanisms might not be represented likewise in some central structure with a regulatory control over the flow of particular and specific action. Such an arrangement seems almost necessary to account for the delicate and selective disorder that is produced by the encephalitic process.

REFERENCES

BEEVOR, C. E. (1904) Muscular Movements and Their Representations in the Central Nervous System, London.
ECONOMO, C. VON (1931) Encephalitis Lethargica. Oxford.
HOLMES, G. (1928) *Proc. R. Soc. Med.*, **21**, 994.
JACKSON, J. HUGHLINGS (1931) Selected Writings, Vol. II. London.
ROSNER, A. A. (1942) *Amer. J. Psychiat.*, **99**, 224.
SCHWAB, R. S., FABING, H. D., and PRICHARD, J. S. (1950) *Amer. J. Psychiat.*, **107**, 901.
WILLIAMSON-NOBLE, F. A. (1928) *Proc. R. Soc. Med.*, **21**, 987.
WILSON, S. A. K. (1928) Modern Problems in Neurology. London.
WIMMER, A. (1926) *Acta Psychiat. Kbh.*, **1**, 173.

W. Ritchie Russell, C.B.E., M.D.—A Study of Muscle Recovery After Poliomyelitis. This paper has been published in *Lancet*, 1954, i, 330.

President—E. A. CARMICHAEL, C.B.E., F.R.C.P.

[*November* 5, 1953]

MEETING HELD AT THE NATIONAL HOSPITAL, QUEEN SQUARE, LONDON, W.C.1

Cortical Blindness and Spastic Quadriparesis Following Apnœa in an Asthmatic Attack.—J. FOLEY, M.D., M.R.C.P. (for J. HAMILTON PATERSON, M.D., M.R.C.P.).

M. N., female aged 25, asthmatic since childhood.

22.6.53.—Admitted to hospital cyanosed after a day in status asthmaticus. A period of apnœa lasting two to four minutes was accompanied by a convulsion, but gasping respiration had started again by the time she was given intravenous aminophylline. Blood pressure 155/105. Bronchospasm diminished with ACTH. For twelve days she remained stuporous and restless with involuntary movements of the jaw and shoulders and a spastic quadriplegia more marked on the right side. Sixteen

days after the onset she could not speak but could respond to commands and raise her arms above her head, but in the next two weeks she became noisy and was transferred to an observation ward.

29.7.53.—Admitted to Atkinson Morley's Hospital. Conscious; no impairment of comprehension of speech; amnesia for whole illness; Gerstmann's syndrome. Speech grossly slurred and almost unintelligible. Vision amounted to perception of light only. Fundi and pupils normal, external ocular movements full. Constant grimacing, rolling of head and torsion movements of shoulders. In addition to severe spastic quadriparesis there was bilateral wrist drop and considerable wasting of the forearm and hand muscles, with loss of joint sense in the hands and impairment of light touch on the fingers only. The EEG consisted of virtually straight lines; lumbar encephalogram normal.

In the next two weeks there was considerable improvement. She had almost constant formed, coloured visual hallucinations of patterns, jewels and toys; though on one occasion she had an hallucination of the Royal Family she was unable to visualize her relatives or any object at will, and she no longer dreamed. Visual fields tested with a beam of light were full, with no definite central defect; she had fragmentary vision for moving objects and bright colours. Audiometry was normal. Apart from dysarthria speech was unaffected. She had a retrograde amnesia of eight months. The athetosis, wrist-drop and wasting of the hands disappeared, but spastic quadriparesis with cerebellar ataxia persisted; sensory loss was confined to joint sense and two-point discrimination in the hands. This stage was reached four months after the onset. On 20.10.53 she had three fits, after which vision deteriorated and hallucinations vanished.

Comment

Improvement in cases of cerebral damage due to anoxia is rare, though it is seen in cases of carbon-monoxide poisoning. The damage here affected the parieto-occipital and motor areas, basal ganglia, cerebellum and peripheral nerves. Destruction of anterior horn cells in the cord has been found in fatal anoxic cases, but peripheral neuritis has so far been reported only in cases of carbon-monoxide poisoning. The improvement in this patient's cerebral signs coincided with the disappearance of wasting in the hands, and much of the earlier cerebral deficit may have been due to demyelination rather than to neuronal damage.

BIBLIOGRAPHY

DOUGLAS, A. J. (1949) *Canad. med. Ass. J.*, **61**, 123.
HAGLUND, C. (1951–2) *Acta chir. scand.*, **102**, 364.
HOWKINS, J., MCCLAUGHLIN, C. R., and DANIEL, P. (1946) *Lancet*, i, 488.
WILSON, G., and WINKELMAN, N. W. (1925) *Arch. Neurol. Psychiat., Chicago*, **13**, 191.

Cerebral Anoxia Following Burns.—J. HAMILTON PATERSON, M.D., M.R.C.P.

A. J., male, aged 4. Admitted to hospital on 11.9.51 with extensive burns acquired when his night attire caught fire. Although his general condition was fair he had widespread burns over the front of the chest, abdomen and thighs. Six hours after injury he collapsed with a rapid feeble pulse and an hour later he had several convulsions which were eventually controlled with Pentothal. He had a further seizure on the third day and thereafter he was nursed in an oxygen tent. He began to take fluids by mouth, but that evening his temperature rose to 106·2° F. and his respiration rate to 96. On the next day further fits were controlled with paraldehyde, but two days later he was found to be unconscious with widely dilated pupils and spastic extremities. Subsequently his general state remained unchanged although he had no further convulsions. He had to be fed by a nasal tube.

On his first admission to the National Hospital six weeks after injury he was found to be entirely inaccessible. Occasional twitching of the face and limbs was seen but otherwise he lay immobile. He appeared to be blind and deaf but he responded slightly to painful stimuli and he showed sucking and chewing reflexes, while the corneal responses were likewise present. Both pupils were widely dilated and did not react to light. The optic discs were pathologically pale with clear-cut margins. All four extremities were extremely spastic, the right arm being held in a flexed and adducted position, the left being extended. Both legs were fully extended. Two years later, on his second admission, his general state is much the same and he does not appear to have grown much in the interval. Usually he lies quite still sucking his fingers, though he becomes restless when disturbed and cries and grunts for a while. He is now taking food by mouth but only with difficulty. He is still quite blind but he can now hear to some extent as he can be startled by a loud noise. His limbs are much less spastic, the arms being held flexed and the legs extended. The tendon reflexes are very brisk and he has bilateral extensor plantar responses.

Investigations

On first admission.—Blood count: moderate microcytic anæmia. Urine and cerebrospinal fluid normal.

Caloric responses roughly normal. EEG "that of an unconscious patient, quite unspecific in its nature."

Air encephalograms: gross generalized dilatation of the whole of the ventricular system.

On second admission.—Bilateral carotid arteriograms; normal.

COMMENT

It must be very rare for a child of this age to survive such severe cerebral damage for he has virtually become decerebrate. His peripheral blindness is of interest as most cases with loss of vision following anoxia apparently have normal fundi and pupillary reactions. His type of blindness is that which is occasionally seen following severe hæmorrhage.

Norman's Type of Diffuse Sclerosis.—DENIS WILLIAMS, M.D.

A girl of 11, with normal parents, has been studied in the National Hospital, Queen Square, since the age of 5 when she began to show disorders of movement. The condition has steadily advanced and there is now generalized and severe dystonia with intellectual defect.

Brain biopsy performed by Mr. Wylie McKissock led to histological study by Dr. William Blackwood, who showed slides typical of diffuse sclerosis of Norman's type. The case will be reported in detail when more facts are known about it.

Glomus Jugulare Tumour Presenting with Papillœdema and Obscurations of Vision.—RAYMOND HIERONS, M.B., M.R.C.P. (for S. P. MEADOWS, M.D., F.R.C.P.).

Mrs. A. N., aged 38, admitted to the National Hospital with a history of frontal headaches of three months' duration and attacks of blurred vision. She denied any other symptoms, especially deafness, vertigo or bulbar symptoms.

The only relevant feature in the past history was a discharging left ear in childhood. The family history was negative. (Examples of familial cases of this condition are, however, described in the otological literature.)

Examination revealed that intelligence was slightly below average but no evidence was noted of deterioration and speech was normal. Other abnormal signs included:

Moderately severe bilateral papillœdema with hæmorrhages and exudates. Slight left-sided deafness.

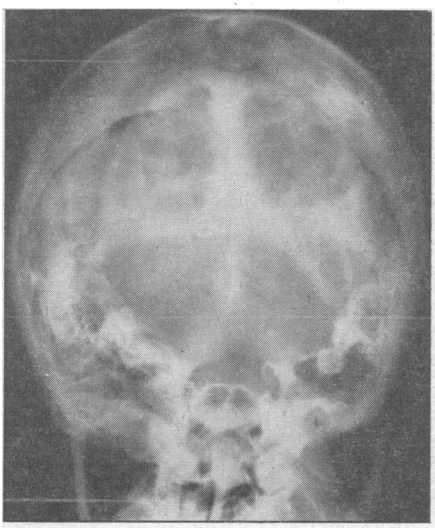

FIG. 1.

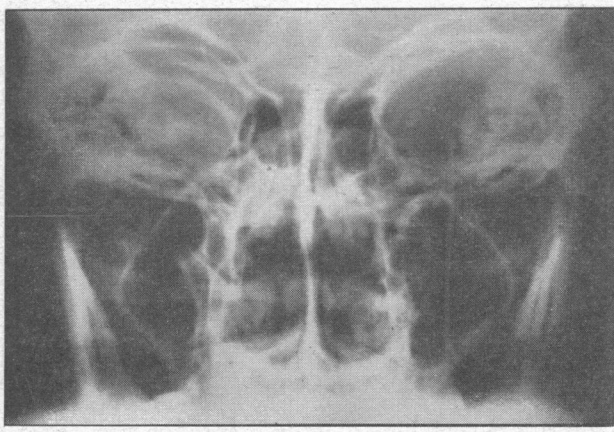

FIG. 2.

FIGS. 1 and 2.—X-rays showing massive destruction of the apex and inferior portion of the left petrous bone.

First-degree vestibular nystagmus on deviation to the right. Left corneal reflex diminished but otherwise no sensory loss detected on the face. Masseter and temporal muscles of normal power and bulk on the two sides. Slight over-reaction of the left lower face on voluntary movement and probably impaired taste over the left anterior two-thirds of the tongue. Paresis of the left side of the palate and left vocal cord. Normal power of the sternomastoids and trapezii. Very marked atrophy of the left side of the tongue with fasciculation on this side. In the motor system no abnormality of development, tone, power or reflexes detected. Minimal ataxia of the lower limbs, slightly more marked on the left and unable to walk along a straight line. No bruit detected over the cranium. The left auditory meatus seen to be filled by a dark blue vascular swelling which was considered by Mr. Terence Cawthorne to have the appearance of a glomus tumour. X-ray skull (Dr. Hugh Davies) revealed signs of increased intracranial pressure in the pituitary fossa and destruction of the left petrous apex (Figs. 1 and 2) extending into the middle and posterior fossa.

Biopsy of the tumour from the external auditory canal reported by Dr. W. Blackwood as "formed of cells with medium-sized rounded or elongated nuclei surrounded by a considerable amount of cytoplasm, the margins of the cells being generally ill-defined. Most of the nuclei are lightly stained but collections of cells with deeply staining small, rounded nuclei occur. One portion of the tumour consisted of solid alveolar masses of cells surrounded by reticulum. The material has been shown to several pathologists, the opinion being that this is neoplastic tissue, part of which has an appearance compatible with, but not undoubtedly diagnostic of, a glomus jugulare tumour."

In view of the dangers of visual failure a full suboccipital decompression was carried out by Mr. Wylie McKissock, a ventriculogram having previously shown the appearances of a large space-occupying lesion in the neighbourhood of the cerebellopontine angle on the left side.

The chief point of interest of this case is its mode of presentation. Interest in recent years in this type of tumour has been chiefly centred in the aural manifestations and with the exception of a recent paper by Dr. R. A. Henson and his colleagues (1953) few reports have been from the neurological standpoint. These authors did not, however, find papillœdema in any of their cases nor in reviewing the literature. Probably, however, with greater awareness of the condition papillœdema will be not so very rare and since this patient was demonstrated I have been informed of two similar cases.

REFERENCE

HENSON, R. A., CRAWFORD, J. V., and CAVANAGH, J. B. (1953) *J. Neurol. Psychiat.*, **16**, 127.

First and Second Division Trigeminal Neuralgia Treated by Intramedullary Trigeminal Tractotomy.—M. A. FALCONER, M.Ch.

Case I.—Miss J. D., aged 52, shop assistant.

History.—Since 1931 subject to bouts of typical tic douloureux centred in right forehead and spreading to right side of nose, upper lip, and roof of mouth. Averaged one severe bout yearly. A recent attempt at injecting the gasserian ganglion with alcohol resulted in numbness over lower jaw, but not relief of pain.

Operation (March 1951).—Right intramedullary trigeminal tractotomy performed under local analgesia. Region of descending tract of V incised to a depth of 3 mm. at a plane 4 mm. below obex. Smooth post-operative course.

Follow-up two years eight months later. Completely free of her old pain, and has no appreciable disability. Occasional tingling sensation over right lower lip related to area numbed by pre-operative gasserian ganglion injection. Not conscious of any alteration in the feeling of her right face, except in a cold wind when temperature is appreciated by left face only.

Shows complete loss of pain and thermal sensibility over right side of face and concha of ear, as well as over right side of tongue, palate, and tonsillar region. Touch spared in these regions. Right corneal reflex present but diminished. No ataxia of limbs.

Case II.—Mr. G. K., aged 48, borough engineer.

History.—Onset in 1941 of bouts of typical tic douloureux involving first and second trigeminal divisions of left face. Longest spontaneous remission nine months.

Operation (May 1951).—Left intramedullary tractotomy performed as in Case I.

Follow-up two years six months later. Is likewise free of old pain, and has no appreciable disability. Has had three transient bouts of mild paræsthesias involving left malar region and occasional mild aches in left side of nose.

Shows almost complete pain and thermal loss over left side of face, but with preservation of touch. Similar distribution as in Case I, except that pain sensibility persists in inner side of upper lip, left upper gum in region of incisor teeth, adjacent hard palate, and left side of nasal septum. This latter sparing is unusual, and is presumably accounted for by the premaxilla in its development having been supplied from the right trigeminal nerve alone.

Left corneal reflex present but diminished. No ataxia of limbs. For a short time after operation this patient exhibited also loss of pain and thermal sensibility in right lower limb, but sensibility in the leg is now normal.

DISCUSSION

This operative procedure was introduced in 1938 by Sjöqvist who advocated incising the descending tract of the trigeminal nerve within the medulla under general anæsthesia and at a level 8 to 10 mm. above the lower end of the 4th ventricle (obex). Since then several surgeons have tried the procedure, and most of those who have written about it have been disappointed in the results. Several have recorded complications such as ataxia of the ipsilateral limbs, paralysis of the vocal cords, and impairment of postural sensibility indicating damage to adjacent structures in the brain-stem, and have also reported that the resulting pain loss has been patchy with consequent partial or imperfect relief of pain. Olivecrona (1942) and Guidetti (1951), however, have written more laudatory accounts.

Four years ago I published a description of the operative technique which was employed in these two cases (Falconer, 1949), and also recorded my experience with 20 New Zealand patients operated on at intervals up to four and a half years. The procedure was of no value for post-herpetic neuralgia, but in the 11 cases of unilateral tic douloureux and 2 of bilateral tic douloureux in which the procedure had been performed, it had conferred uniform and apparently permanent benefit and without untoward complications. Two points seemed necessary for success, namely, that the incision should be at a lower level than that originally recommended, and secondly that it should be performed under local analgesia. By sectioning the descending tract at a level 4 mm. or even lower below the obex as advocated by Weinberger and Grant (1943) the complicafions listed above could be avoided; and by performing the operation under local analgesia as recommended by Olivecrona (1942) the resulting sensory loss could be checked and the extent of the incision regulated. Further, by pricking the medulla with a needle, the position of the tract can be established prior to the sectioning, because both the pricking and the sectioning cause momentary pain in the face.

These two patients presented are the first to undergo this operation in the Guy's-Maudsley Neurosurgical Unit.

Treatment by alcohol injection of the gasserian ganglion would have completely numbed one side of the face and incurred the risk of corneal ulceration. The ordinary run of patients in whom pain affects the lower half of the face are more readily treated by the usual subtotal sensory-root section, for it is technically difficult to produce pain loss in the lower face by intramedullary tractotomy without incurring a risk of some ataxia of the ipsilateral limbs. But for tic douloureux involving the forehead, and also for tic douloureux in which pain appears on both sides of the face, intramedullary tractotomy is the operation of choice, because both touch over the face and postural sensibility in the masticatory muscles are preserved. Indeed, for the latter indication it is far the most preferable surgical procedure, for the lot of a patient who is bereft of all sensibility in the lower jaw (as after bilateral ganglion injections with alcohol) is a pitiful one.

REFERENCES

FALCONER, M. A. (1949) *J. Neurol. Psychiat.*, **12**, 297.
GUIDETTI, C. (1951) *J. Neurosurg.*, **7**, 499.
OLIVECRONA, H. (1942) *Arch. Neurol. Psychiat., Chicago*, **47**, 544.
SJÖQVIST, O. (1938) *Acta psychiat. neurol.*, Suppl. 17.
WEINBERGER, L. M., and GRANT, F. C. (1943) *Arch. Neurol. Psychiat., Chicago*, **49**, 665.

Chronic Respiratory Paralysis due to Poliomyelitis Treated by Positive Pressure Inflation of the Chest.—R. A. BEAVER, B.M., F.F.A.R.C.S., and R. W. GILLIATT, M.B., M.R.C.P. (for M. KREMER, M.D., F.R.C.P.).

Miss E. H., aged 28. Admitted to the National Hospital on 7.7.53 with a history of fever for four days and headache with neck stiffness for one day. Examination on admission revealed no muscular weakness but lumbar puncture showed 468 cells per c.mm. 93% polymorphs. Weakness was first noted on 8.7.53 in left triceps and was followed by rapidly increasing paralysis of all trunk and limb muscles with failing respiration. No bulbar involvement occurred at any stage but spontaneous respiratory movement was virtually absent by 10.7.53, the patient being unable to stay out of a box respirator for more than 90 seconds.

In twelve weeks only slight recovery of the respiratory muscles has occurred and the patient still becomes distressed after two to three minutes of unassisted breathing. The vital capacity has now reached 250 c.c. and slow improvement continues. The patient has been nursed in a conventional box respirator but frequent periods of positive pressure inflation have been employed from the onset in order to allow the patient to be withdrawn from the respirator for nursing procedures and physiotherapy.

The pneumoflator designed by Beaver (1953, *Lancet*, i, 977) was used in the acute stage with a face mask of the self-sealing type (Goorney, 1953, *Lancet*, i, 422), periods of positive pressure ventilation being supervised by an anæsthetist or resident. For the chronic stage a simple non-adjustable mouthpiece has been introduced (Beaver and Gilliatt, 1953, *Lancet*, ii, 1243), which has made it possible for positive pressure inflation by pneumoflator to be carried out safely by the nursing staff without special supervision.