Discussion

The great difference in results as regards the saving or improvement of vision as well as mortality is striking, but is easily explainable on anatomical grounds. When the tumour is of large size the anterior cerebral arteries, the anterior communicating vessel, and the distal anterior cerebral arteries are stretched tightly over its dome and posterior surface: the floor of the third ventricle is displaced far upwards and backwards, and the foramina of Munro may be occluded, resulting in dilatation of the lateral ventricles and greatly raised intracranial pressure. Damage to any of these structures is all too easily inflicted, with a possibly fatal outcome.

The optic nerves and chiasm are very extensively involved in these vast tumours, the nerves being greatly elongated and displaced backwards if not laterally as well, while the chiasm is broadened and thinned, and is pushed backwards and downwards. It is infinitely more difficult to free these structures from the tumours, for they are often adherent to the capsule, softer in consistency than normal, and very readily harmed during dissection. For this reason improvement in visual acuity or field is more difficult of achievement, while aggravation of pre-existing defects is all too common.

Operative intervention in early cases is a much simpler matter, especially with the aid of hypothermia and intravenous urea, which, by producing great shrinkage of the brain, enable the frontal lobe to be raised with ease and without trauma, providing an admirable view of all essential structures.

Unfortunately, early examination will not always reveal a small tumour, for the duration of symptoms was found to be actually less in the large tumours than in the small ones. This discrepancy can perhaps be explained by postulating the differing rates of growth of the tumours, but it is also possible that the exact point of origin of the growth is responsible in part. Once a tumour has reached a large size its area of dural attachment is extensive, and it can be imagined that some of them originated at a point well away from the actual optic foramina and sellar lip, thus allowing them considerable room for expansion before pressure on the nerves or chiasm becomes serious. The smaller, more strategically placed tumours in the immediate vicinity of the optic foramina would at once involve the appropriate optic nerve. Nevertheless, a slow rate of growth must be the answer in those patients with small tumours and a history of many months or years of visual disturbance. Although the suprasellar tumours are only a small group representing some 6% of meningiomas in this series (but nearly 9% in Cushing and Eisenhardt's) the establishment of the diagnosis at an early stage may well make the difference between life and death to the patient, to say nothing of the preservation, as opposed to the loss, of eyesight.

The average duration of two and a half and three and a half years of symptoms in these two series certainly leaves much to be desired in the direction of early diagnosis.

During this 25-year period when these 50 suprasellar meningiomas were encountered, 14 additional patients were subjected to exploration of the optic chiasm on the same clinical basis. In 5 of the 14 unmistakable chiasmal arachnoiditis was revealed, in one a glioma of the optic chiasm was found, and in the remaining eight cases no abnormality of optic nerves or chiasm

could be seen. No benefit accrued in any of these 14 cases as a result of exploration of the chiasm and nerves, although these structures were dissected free in the cases of arachnoiditis. The one patient with a chiasmal glioma could not be treated by excision of the tumour as this would have produced bilateral blindness. In none of these 14 patients was harm done by exploration.

It cannot be emphasized too strongly that progressive failure of vision, even in a single eye, justifies the earliest possible admission for specialized investigation. Only in this way can many tragedies be averted.

Summary

Fifty cases of suprasellar meningioma are reported, of which 17 were "small" and 33 "large."

The symptomatology was almost entirely confined to diminution in visual acuity and/or visual fields.

Surgical excision of the small tumours is attended by no mortality and there was a high proportion of improvement in visual signs and symptoms. Surgical excision of the large tumours is extremely difficult and has a high mortality rate.

Emphasis is laid upon the need for early diagnosis in the hope of being able to operate when these tumours are of small size.

Unfortunately length of history is no guide to the size of the tumour, and it is evident that there must be wide variations in their rates of growth.

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VALUE OF NERVE-EXCITABILITY MEASUREMENTS IN PROGNOSIS OF FACIAL PALSY

BY

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Facial paralysis of lower-motor-neurone origin may be due either to physiological (reversible) block or to degeneration of the facial nerve. The prognosis depends on which has occurred, the chances of full recovery being good in physiological block but poor in degeneration. The present study demonstrates how accurately this knowledge may be acquired early in the course of the paralysis by a simple electrodiagnostic test.

Historical Note

Electrodiagnosis of facial palsies was first described by Duchenne (1872). He distinguished between motor-nerve-excitability tests and those based on local stimulation of individual muscles. For stimulation of the facial nerve as it leaves the stylomastoid foramen he recom-

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mended that the active electrode be placed inside the external auditory meatus. In discussing "rheumatismal" facial palsy he claimed that electromuscular investigations permit distinction between those palsies which recover easily and rapidly and those which persist despite all means of treatment. In the first group muscular contractility on nerve stimulation was diminished and in the second absent; his tests were reliable one week from the onset of the paralysis. He also observed that nerve excitability once lost did not return even after recovery of voluntary movement.

More recently (Campbell, 1954; Richardson and Wynn Parry, 1957; Collier, 1959) nerve-excitability tests have again been advocated, as they distinguish clearly between physiological block and nerve degeneration. This information is obviously essential for any assessment or comparison of methods of treatment, for patients with physiological block, because they recover rapidly, completely, and without treatment, must be accounted for in any therapeutic trial. Surprisingly, in no published report so far has this been done.

Principles and Method

The early changes in muscle and motor nerve distal to nerve section are well summarized by Adams, Denny-Brown, and Pearson (1953). The distal portion of the nerve maintains its electrical excitability for 48 to 72 hours, after which the axis cylinders fragment with abolition of excitability and conduction. The motor end-plate, however, retains its full excitability for a further 5 to 10 days, during which time the last fragments of axis cylinder in the motor end-plate disappear. The excitability of the end-plate then diminishes so that only the muscle fibres remain excitable. Thus the characteristics of reaction of degeneration and the changes of denervation in the intensity-duration curve occur. In practice, therefore, if electrical stimulation is applied direct to recently denervated muscle it may respond with the characteristics of normal muscle for up to 10 days before showing the characteristics of denervated muscle. If, however, the motor-nerve trunk is stimulated, excitability and conduction will generally disappear three days after a denervating lesion has occurred (Landau, 1953). By this method, therefore, the state of the nerve may be determined earlier than by the plotting of intensity-duration curves derived from affected muscles.

Motor-nerve excitability is conveniently tested by applying an electrical stimulus to the nerve trunk peripheral to the suspected site of the lesion. The intensity of current necessary to produce minimal visible contraction of the muscles supplied by the nerve is compared with the intensity needed on the unaffected side.

In this investigation the facial nerve was stimulated by applying the active electrode (cathode) to a point below the mastoid process and behind the angle of the jaw. The indifferent electrode (anode) was placed at the back of the neck. To localize the stimulus, to avoid unwanted contraction of other muscle groups, and to avoid direct stimulation of the facial muscles, the active electrode used was small (area approximately 1 sq. cm.) and the indifferent electrode large (150–200 sq. cm.). A square pulse of one millisecond duration was delivered from a high-output impedance (constant current) electronic stimulator. The normal side was tested first and the minimal effective current intensity noted (usually between 3 and 8 mA). The active electrode was moved around until the site of greatest response was found,

particular note being made that muscles supplied by all branches of the facial nerve responded. The equivalent position on the affected side was then stimulated. The results obtained were one of the following: the affected side responded fully at the same current intensity as the normal (excitability unimpaired), it responded fully but required more current than the normal (excitability diminished), or it failed to respond regardless of how much current was applied (excitability absent). These three findings indicate physiological block, partial nerve degeneration, and complete degeneration respectively.

Where increased currents were used, care was taken that contraction of the masseter was not mistaken for a response of the facial muscles. By virtue of the position of the stimulating electrode the platysma may respond by direct stimulation when increased currents are used, and it is important to realize that this does not imply the presence of nerve conduction. Minor variations of current intensity occurred between the two sides in normal individuals, and we have regarded an increase of at least 2 mA necessary before considering excitability to be diminished.

All patients with facial paralysis referred to the department of physical medicine and rheumatology of the Royal Free Hospital for electrodiagnosis have been tested by this method since 1954. Patients who had incomplete lesions (one branch affected) or facial weakness rather than paralysis have been excluded from the series because it is well known that such patients have an excellent prognosis. Cases in which traumatic section of the nerve had occurred have also been excluded. Thus the present series consists of 197 patients (107 male and 90 female), with an age range of 4 to 76, all of whom have been followed up until their condition appeared static. All patients have had an aural examination (the majority in the E.N.T. department of the Royal Free Hospital) and none had any evidence of an upper-motor-neurone lesion or additional lower-motor-neurone lesions. They were classified on the following aetiological basis: suppurative conditions of the middle ear or mastoid (local sepsis); herpes of the geniculate ganglion (otic herpes); post-operative or direct trauma (local trauma); no evidence of any known predisposing cause (Bell's palsy).

Two cases occurred after temporary inferior-dental-nerve block, and because the aetiology of this seems obscure they have been included separately.

Patients were tested as soon after the third day after onset as possible, 56 (28%) being tested before the seventh day, 99 (50%) before the fourteenth day, 123 (62%) before the twenty-first day, and the remainder thereafter. The result of the initial motor-nerve-excitability test was used in this series though patients were retested at frequent intervals and additional methods of electrodiagnosis (the plotting of intensity-duration curves and electromyography) were also generally used.

Time of recovery was easy to assess when recovery was complete, but in incomplete recovery the time taken to achieve a static condition is of necessity much less definite, and we are aware of the inaccuracy in assessing this latter.

Recovery was classified in three grades. (1) Complete, meaning full volitional activity and absence of distortion at rest or on movement. (2) Good volitional activity with some distortion of facial movements but normal in repose. (3) Poor volitional activity with distortion on

movement and at rest; or, no recovery at all. The majority of the patients were assessed as out-patients, but in some cases where long distance or inconvenience was involved we have accepted the patient's own assessment recorded on a questionnaire.

Results

The results are presented in the Table and the Chart, and need only brief elaboration. A small proportion of patients in whom nerve excitability was unimpaired initially, and therefore in whom an excellent recovery was anticipated, later lost excitability. This occurred in 13 patients, 10 of whom had Bell's palsy, one local sepsis, one trauma, and one herpes. However, four of those with Bell's palsy made a full recovery eventually, the remaining patients having some residual distortion. Therefore an erroneous good prognosis was made in 9 out of 77 patients (11%). In none of these patients was excitability lost before the seventh day, and in the majority this occurred between the tenth and twenty-first days. In another three patients excitability was retained in some branches of the nerve and was absent or diminished in the others; in these cases recovery in each branch followed the expected course.

The Chart illustrates the recovery rate of each group, based on the time taken to achieve maximal recovery. This Chart represents the time-course of 177 patients;

Division of Cases of Facial Palsy in the Three Electrophysiological Groups Into Their Aetiology and Degree of Recovery

Aetiology	Nerve Excitability	Cases	Full Recovery	Incomplete Recovery	
				Distortion on Movement	Distortion at Rest and on Movement
Bell's palsy (117)	Unimpaired	61	55 (90%)	4 (7%)	2 (3%)
	Diminished	21	10 (48%)	9 (42%)	2 (9%)
	Absent	35	7 (20%)	11 (30%)	17 (50%)
Local sepsis (19)	Unimpaired	6	5	1	—
	Diminished	5	2	3	—
	Absent	8	1	2	5
Otic herpes (13)	Unimpaired	4	3	1	—
	Diminished	4	—	2	2
	Absent	5	—	2	3
Local trauma (46)	Unimpaired	4	3	1	—
	Diminished	5	2	2	1
	Absent	37	3	11	23
Dental block (2)	Unimpaired	2	2	—	—
	Diminished	—	—	—	—
	Absent	—	—	—	—
Total ..	Unimpaired	77	68 (89%)	7 (9%)	2 (2%)
	Diminished	35	14 (40%)	16 (46%)	5 (14%)
	Absent	85	11 (13%)	26 (31%)	48 (56%)
Total		197	93	49	55

Time of return to maximum recovery (not necessarily full recovery) after the onset of paralysis. Full recovery occurred in 89% of patients in group 1 in six months, in 40% of patients in group 2 in 10 months, and in 13% of patients in group 3 in 18 months. A further 16% in group 3 had no recovery at all in 18 months. x—x Group 1: nerve excitability unimpaired. ●—● Group 2: nerve excitability diminished. ○—○ Group 3: nerve excitability absent.

those not included are the 13 originally unimpaired which degenerated and 7 who could not attend for follow-up and who could not remember the time of recovery with sufficient accuracy.

Discussion

These results show that, as may be expected, patients suffering from physiological block as judged by retention of nerve excitability after 72 hours have an excellent chance of complete recovery; what is significant, however, is that this information can be obtained early with good accuracy. The time of recovery in the uncomplicated cases of this group ranged from 1 to 20 weeks, but two-thirds had fully recovered by six weeks. Clearly, this group requires no treatment except perhaps instruction in attempting to perform facial movements daily in front of a mirror.

Where nerve degeneration has taken place, as judged by loss of nerve excitability, the onset of recovery cannot be expected before three months—the time compatible with 1 in. (2.5 cm.) per month as the functional rate of regrowth of nerve. The time to maximal recovery ranges from 3 to 18 months. A depressing fact about this group is the poor chance of full recovery after nerve degeneration has occurred. Treatment of this group of patients was an attempt to preserve the viability of the denervated muscles. The conventional methods of an internal dental splint and daily stimulation of the affected muscles by interrupted galvanism were carried out in most patients in this group. Cases occurring post-operatively were often re-explored, but formal decompression in Bell's palsy was performed on only two patients (at other hospitals). It is interesting that, in keeping with Duchenne's findings, even when full volitional recovery had occurred after degeneration, nerve excitability returned in only a minority of patients. This may be due to greatly increased currents being necessary to stimulate the nerve, and this is often precluded by pain. Furthermore, a high-intensity stimulus causes strong contraction of the masseter and other jaw muscles, which makes observation of the facial muscles difficult, so possibly this finding is more apparent than real.

It seems from our results that when degeneration of the facial nerve occurs it usually happens early, probably within the first two or three days of the paralysis. The number of patients whose nerve excitability disappeared after being present initially was small, though when this did occur it usually happened after the tenth day.

Taverner (1955) investigated a series of patients with Bell's palsy using electromyography alone, and made a correct prognosis in 93 out of 96 cases. However, this method has the disadvantage that it is based on the detection of fibrillation potentials (indicating denervated muscle fibres). These can be late in occurring (up to 18 days after denervation) and are seldom detected before 10 days. Furthermore, nerve-excitability tests are more comfortable for the patient and require less-complicated apparatus. Electromyography can also be misleading in prognosis because fibrillation potentials may be detected when only a small minority of the nerve fibres have degenerated, suggesting a worse prognosis than is justified. Thus we have occasionally detected fibrillation potentials in the presence of unimpaired nerve excitability and complete recovery has resulted. Conversely we have found that the retention of isolated normal motor unit action potentials in the absence of nerve excitability gives no more favourable a prognosis than when there is complete lack of volitional activity.

However, should there be doubt about whether or not the facial nerve has been cut then the presence of these action potentials gives immediate assurance that the nerve is in continuity.

Gilliatt and Taylor (1959) measured conduction time in the facial nerve between the stylomastoid foramen and orbicularis oculi in three patients before and after complete nerve section. They found no change in the latency of the muscle response but noticed a sudden diminution of amplitude after the third post-operative day, together with disappearance of the visible twitch. In a further four patients whose facial nerves were completely sectioned during removal of acoustic neuromata modified intensity-duration curves were plotted from orbicularis oculi at daily intervals. The results of this test showed an increasing threshold to 0.1 and 1 millisecond stimuli from the fourth post-operative day. Our experience has been that, whereas nerve-excitability tests usually give a clear indication and require little interpretation immediately after the third day, intensity-duration curves may alter insidiously at first. Thus we feel simplicity of apparatus, performance, and interpretation commend the use of nerve-excitability tests in facial palsy.

The practical application of nerve excitability tests is in the assessment of treatment. Widely different measures have been advised in the treatment of Bell's palsy, such as decompression, the use of cortisone

derivatives, and cervical sympathetic block. The efficacy of these or any other methods cannot be assessed unless the number of patients with physiological block are known, for 90% of these will achieve full recovery regardless of treatment.

Summary

A simple test of motor-nerve excitability is described. This test distinguishes between physiological block and degeneration of the facial nerve as early as 72 hours after the onset of facial palsy.

The results of this test and their relation to the time and degree of recovery in facial-nerve palsies of various types are described.

It is suggested that such a test should be used in the assessment of treatments of facial palsy.

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URINARY INFECTION AND ANAEMIA IN PREGNANCY

BY

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Two of the most common causes of anaemia in pregnancy are iron and folic-acid deficiency. A significant proportion of patients, however, who are receiving these haematinics remain anaemic or become anaemic as pregnancy progresses. This has been observed in the antenatal clinic of the City General Hospital, Stoke-on-Trent, where both iron and folic acid have been given for a number of years (Giles and Burton, 1960). Anaemia in these cases might be due to excessive haemodilution (Dieckmann and Wegner, 1934), failure on the part of the patient to take her drugs, or possibly to some other factor such as intercurrent infection. Since the urinary tract is by far the commonest site of infection in pregnancy, an attempt has been made over the past two years to determine whether urinary infection is related to some of these cases of refractory anaemia.

Any disorder in late pregnancy, whether it be anaemia, urinary infection, or both, must be treated without delay. Elaborate investigations or controlled therapeutic trials are therefore not usually practicable, and it was apparent that any study of this problem would have to be based on relatively simple tests. The accepted criteria of urinary infection have been based by some authors (Stansfeld, 1954; Hutt *et al.*, 1961) on finding an excess of leucocytes in measured specimens of urine and by others (Kass, 1957; Turner, 1961) on cultural methods or on the more reliable but time-consuming "pyrexal" test (Pears and Houghton, 1959; Hutt *et al.*, 1961). In the present investigation, carried out on large numbers of out-patients, a simple combination of some of these methods was adopted, using mid-stream rather than

catheter specimens, as the latter were thought to be too dangerous (Kass, 1957).

Methods

All patients attending the hospital antenatal clinic received prophylactic iron by mouth after their first visit and routine folic acid from the twenty-eighth week of pregnancy until after the confinement. Women in whom a haemoglobin below 70% (10.4 g./100 ml.) had been observed on more than one occasion were included in the present investigation. A brief case-history was taken, the haemoglobin and mean corpuscular haemoglobin concentration (M.C.H.C.) were determined, and a stained blood film was examined. A mid-stream specimen of urine was obtained with suitable precautions, and where it was thought advisable a specimen of blood was taken for serum iron, serum vitamin B₁₂, and, latterly, for serum folic-acid estimation. The bone-marrow was examined by sternal puncture in a number of cases.

Urine specimens were cultured, using standard loopfuls, on blood and MacConkey agar; a portion of the specimen was then set aside for leucocyte count and the remainder centrifuged. The supernatant was examined for protein and the deposit for the presence of casts, red cells, and leucocytes. In all cases where the leucocytes exceeded 5 per high-power field a leucocyte count was performed on the portion of the specimen previously set aside for this purpose. Counts of 25,000/ml. or more were considered significant; in severe infections the count amounted to several million leucocytes per ml. Cultures were regarded as