the pre-anaesthesia detailed for each patient and a timely shot of pentothal or whiff of gas and oxygen the local anaesthetic is robbed of its terrors. Some patients have declared that they would not wish for any other method.

The After-progress

After the average major operation patients are well mentally and physically. The day after operation they are obviously subdued, with a raised pulse rate, but thereafter progress is uneventful. There is a complete absence of the anxious, suffused, and chesty facies and of headaches, and seldom is there any vomiting. The old troubles of the post-operative chest—cough and hiccup, distension of the abdomen, and retention of urine—have practically disappeared. A former house-surgeon said: "Every case of partial gastrectomy done under a spinal or general anaesthetic had a cough of some sort about the third day, but not these patients done under local." Another said concerning partial gastrectomy subjects under local anaesthesia: "After the first day they recover 'like an appendix.'"

An early disadvantage experienced was that a few wounds broke down superficially and healed slowly, which was considered to be due to adrenaline in the anaesthetic: 2 minims per 1 oz. was then used. This was reduced to 1 minim per 1 oz., and healing has since been perfect, even in carcinomatous subjects.

Several operations were on children; these passed off smoothly, and the recoveries were uneventful. A whiff of gas was given during the intra-abdominal manipulation, lasting in each case less than a minute. The proceedings were explained to the children with the same courtesy and detail as to an adult, and they co-operated admirably.

TABLE I.—367 Cases Operated on Mainly under Local Anaesthesia (September, 1937, to April, 1940, inclusive)

Local infiltration only	194
" " plus gas and oxygen	90
" " " pentothal or evipan	62
" " " light open ether	12
" " " auto-administration of gas (by Minnitt gas apparatus)	9

TABLE II.—The Cases Detailed

Acute appendicitis	49 (no deaths)
Ligature and injection of varicose veins (groin and knee—many bilateral)	39
Hernia, radical cure	37
Partial gastrectomy	40
Cholecystectomy with drainage of common bile duct	27
Laparotomy and colostomy	13
Laparotomy and chronic appendix	33
Steinach II ligature	12
Thyroidectomy (toxic goitre)	11
Suprapubic cystostomy	11
Meatotomy, circumcision, or amputation of penis	10
Gastro-enterostomy	9
Carcinoma of recto-sigmoid junction (Hartmann's operation)	8
Operations on the kidney	7
Perforated peptic ulcer	4
Lobectomy for simple tumour of breast	5
Prostatectomy	4
Tuberculous glands of neck	4
Cholecystgastrostomy	3
Radical excision of the breast for carcinoma	3
Fractured patella	4
Intestinal obstruction	4
Chronic appendicitis	3
Operations on uterus and ovaries	2
Carcinoma of bladder: partial cystectomy	1
Subacute osteomyelitis of skull: resection of area of skull	1
Various minor operations	23

All emergencies have been done with local anaesthesia whenever possible: the mortality and complications have been reduced and the after-progress has largely been uneventful.

Conclusion

In order to operate effectively under local anaesthesia the following points must be observed:

The anaesthetic must be injected into the entire operation area. This is so obvious as to seem redundant, but the need is often forgotten, and the surgeon is reluctant to stop operating, with the consequence that pain, straining, groans,

and dissatisfaction result. "Taking a chance" that a part is insensitive is mostly unsuccessful.

The surgeon must wait for the anaesthetic to act; therefore operations under local anaesthesia, especially in the early stages, last longer than those under general or spinal; but later, taking the induction period into account, the difference in the time is not so wide.

A precision of technique and a daintiness are exacted which are unnecessary with the conventional methods. The tissues are divided by cutting instead of tearing and stripping, and swabbing must be minimal. A measure of discipline and control is required from the surgical team that is not necessary with the usual anaesthetics.

Local anaesthesia cannot be popular in teaching hospitals, for students must have anaesthetic practice; but where there is not this additional responsibility, continued use has crystallized in the opinion that for most operations of general surgery local anaesthesia carries the maximum of safety to the patient.

Summary

Personal experiences of the use of local anaesthesia practised since May, 1937, are given, together with the salient points that have arisen.

Pre-anaesthesia and supplementary anaesthesia are dealt with.

General, spinal, and local anaesthesia are compared in their effects on the blood pressure and pulse rate.

Points to be observed in operating under local anaesthesia are stressed.

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CEREBRAL THROMBOPHLEBITIS

BY

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Thrombophlebitis of the superficial cerebral veins is a well-recognized complication of otitis media. Courville and Nielsen (1934, 1935, 1937) believe that the pathway of infection is as a rule by way of the lateral sinus. Thence the thrombosis may spread upwards into the superior longitudinal sinus and so downwards into tributary veins, or it may take the path offered by the inferior anastomotic vein (vein of Labbé), which opens at one end into the genu of the lateral sinus and at the other end into the superficial middle cerebral vein (see Figure). In either

case cerebral symptoms may be absent unless or until one of the main tributary veins from the region of the Rolandic fissure is involved. There is then a rapid onset of weakness or paralysis, often preceded by focal epileptic attacks.

If the clot is aseptic there may be no evidence of constitutional disturbance. Thus, in a patient with otitis media the sudden onset of focal symptoms—fits or paralysis—may be the first evidence of an aseptic thrombophlebitis beginning in the lateral sinus and spreading by one of the pathways described to the cortical veins (Symonds, 1937). Either pathway must traverse the subdural and subarachnoid spaces. If, therefore, there are organisms in the clot, subdural abscess or leptomeningitis may result. As a more frequent complication an abscess of the brain may develop in the zone of cortical thrombophlebitis. In a great many cases, however, these hazards are escaped, the spread of the thrombophlebitis ceases, collateral veins soon provide an adequate circulation, the affected area of cortex recovers its function, and the symptoms rapidly disappear. If the pathway of spread has been along the superior longitudinal sinus, increased intracranial pressure may result either from venous engorgement or from accumulation of the cerebrospinal fluid, which normally drains into the great venous sinuses through the arachnoid villi. So long, however, as the clot remains aseptic, organization and canalization will restore the normal circulation, and these symptoms also will disappear.

It is well known that thrombosis of the lateral sinus may also result from direct spread of infection from the tonsil or nasopharynx, and again may be symptomless so long as the clot is aseptic. I have recorded a case of right peritonsillar abscess in which the patient developed a left facio-brachial monoplegia, followed by the signs of increased intracranial pressure, with spontaneous recovery; the assumption being that an aseptic thrombophlebitis of the right lateral sinus had extended along the superior longitudinal sinus to involve the right superficial middle cerebral vein. Thrombophlebitis may also develop in the anterior end of the superior longitudinal sinus from direct spread of infection in the nose or accessory air sinuses and extend *backwards* to involve the tributary veins. Again, if the process is aseptic, fits or paralysis may be the first clinical evidence of this happening. The direct spread of infection, however, is not the only cause of aseptic thrombophlebitis of the dural sinuses and their tributary veins. This may occur as the result of a generalized liability to thrombophlebitis in certain states of infection or malnutrition. Whenever, therefore, there is a sudden development of cerebral symptoms in a patient with thrombophlebitis elsewhere in the body, or in the course of an illness in which thrombophlebitis is known to be common, the possibility of this occurrence in the dural sinuses or cerebral veins should be considered.

The object of the present communication is to record five cases in which the diagnosis of cerebral thrombophlebitis appeared reasonably founded. In two instances the cause was direct spread of infection from frontal sinusitis, and in another it was direct spread (presumably) from the nasopharynx. In the other two cases the symptoms developed after parturition at a stage in which thrombophlebitis elsewhere in the body is common. In one of these cases the diagnosis was confirmed at an exploratory operation.

Frontal Sinusitis: Case I

A youth aged 17 years 8 months developed influenza complicated by acute right frontal sinusitis on March 16, 1938. The sinus was drained by external operation on March 21. Pain was at once relieved, and within forty-eight hours the temperature, which had been high, returned to normal. The wound drained satisfactorily; the patient felt well in himself, and ate well. On March 25 his mother noticed that he was apathetic. No other abnormality was noticed until 4 p.m. on March 26, when he was about to use the bed pan; he then found that he had lost power in the left leg and was

weak in the left arm. There was no headache, vomiting, or rise of temperature.

When I saw him at 9 p.m. he was quiet, alert, and sensible. The fundi showed marked engorgement of the retinal veins. There was no weakness of face, palate, or tongue. There was slight falling away of the extended left arm, the left hand-grip was weak, and movements of the left fingers were slower than those of the right. The left lower limb was completely paralysed at all joints and flaccid. No sensory impairment of any part to light touch, pin-prick, or tests of postural sense could be discovered. The arm-jerks, knee-jerks, and right ankle-jerk were absent, the left ankle-jerk feebly present; the right abdominal reflexes were normal, the left feebly present; the right plantar response was flexor, the left extensor. Lumbar puncture showed clear fluid with a pressure of 300 mm. The fluid contained 13 cells per c.mm., of which 96% were lymphocytes and 4% polymorphs. The protein was 50 mg. per 100 c.cm.; the Nonne-Apelt and Pandy tests were weakly positive.

The diagnosis made was thrombosis of the superior longitudinal sinus together with a tributary vein. It was considered that the good general health and absence of rigor were excellent prognostic points. No further treatment was considered necessary.

On March 28 Dr. R. E. Smith, who had care of the patient, reported complete recovery from the paralysis. I saw the patient again on April 3 and found no abnormal signs in the nervous system except a little slowness of independent movement in the left toes. The temperature had remained normal and the general condition good. On April 5 Dr. Smith reported that in the course of an hour or two the patient had again developed weakness of the left limbs, amounting to paralysis of the foot, weakness at knee and hip, and slight weakness of the arm. There was no headache, vomiting, or drowsiness, and the temperature was normal. It was considered that there had been an extension of the thrombophlebitis, but with no evidence to suggest abscess formation. The weakness this time persisted without change until April 12. The cerebrospinal fluid was examined on that day and showed 5 cells per c.mm., of which 8% were mononuclears, some of them with granular inclusions (? blood pigment). No polymorphs were seen. The protein was 40 per 100 c.cm. Dr. Greenfield examined the fluid and gave the opinion that the slight excess of cells might be explained by the after-effects of a sinus thrombosis.

On April 15 the patient found himself able to move his left toes, and in the course of the next few days he made a complete recovery. He has since remained perfectly well.

Comment.—This case illustrates well enough how quiet may be the period during which thrombosis is extending. The onset of paralysis on the first occasion was sudden; the recovery so rapid that it could hardly be explained except in terms of anastomotic circulation. On the second occasion, as might be expected, the re-establishment of circulation, and therefore of cortical function, was slower, but none the less complete. The abnormalities in the cerebrospinal fluid were characteristic of an infective cortical thrombophlebitis. The distribution of the paralysis—leg affected severely, arm slightly, and face not at all—is what might perhaps be expected when thrombosis spreads from the superior longitudinal sinus into a tributary vein. This distribution is, however, by no means constant.

Case II

A man aged 20 was transferred from an Air Force hospital to a military hospital for head injuries on February 22, 1940, on account of fits and right-sided weakness. The family and past history contained nothing of importance. The recent story was that three weeks previously he had had a cold and sore throat, with a purulent discharge from the right nostril. On February 12 he complained of headache, and on the 14th he was admitted to a R.A.F. hospital with evidence of an acute right frontal sinusitis. The temperature was 103°, the pulse 120. He was treated with sulphapyridine (M & B 693).

X-ray examination revealed a pansinusitis, with fluid levels in both frontal sinuses and both antra. The antra were washed out, and his condition improved. On February 19 his temperature fell to normal.

At 7 a.m. on the 22nd, shortly after he had been observed to be in normal health and about to eat his breakfast, he was found unexpectedly to be unrousable. The orderly officer was called, and when he arrived a few minutes later he found the patient cyanosed and generally convulsed. The epileptic attack lasted some minutes. At 7.50 a.m. while lying quietly unconscious he suddenly had another fit. There was generalized tonic convulsion, followed by clonic convulsion. The attack lasted two minutes. He was examined by Squadron Leader Brinton at 9.15 a.m. He was then conscious, but confused and slow. Examination revealed a slight weakness of the right lower face and the right upper and lower limbs, a grasp reflex in the right hand, and continuous twitching movements in the right forearm, hand, and anterior tibial muscles. The arm-jerks were feebly present and equal, the right abdominal reflexes diminished, the right knee- and ankle-jerks increased, the right plantar response feebly flexor, with fanning of the toes, the left flexor. At 3.45 p.m. Squadron Leader Brinton found him mentally alert. The physical signs were unchanged. The cerebrospinal fluid was under a pressure of 110 mm. and was reported to contain 62 cells per c.mm.—"probably granulocytes."

The patient was transferred to a military hospital for head injuries that night. The physical signs were unchanged. The white cell count in the blood was 7,725 per c.mm.—75% polymorphs. On the following day he was examined by Major H. S. Sharp, specialist in diseases of ear, nose, and throat, who reported that both frontal sinuses were draining well into the nose. As the patient remained well in himself and apyrexial it was decided not to explore the frontal sinuses.

On February 25 he awoke at 2 a.m. with a stinging sensation in the right leg from knee to foot and in the right arm from elbow to hand. He felt the right arm and leg slightly weaker. Twelve hours later the stinging sensation was almost gone from the right arm; it was present, though less severe, in the right leg. There was no fresh development of physical signs in association with this episode. In the course of the next week the abnormal physical signs rapidly disappeared and the nasal discharge ceased. He remained apyrexial and was free from headache. Improvement was maintained during the subsequent weeks, and he was ultimately discharged from hospital recovered.

In the earlier stages of this case abscess was suspected, and although a full review of the facts led to the diagnosis of thrombophlebitis the possibility of an abscess secondary to this process was kept in mind. Lumbar puncture was therefore performed at intervals, and the pressure and contents of the cerebrospinal fluid were as follows:

Date	Pressure	Cells per c.mm.	Type of Cells	Protein mg. per 100 c.mm.
28/2/40 ..	140	18	Lymphocytes	35
9/3/40 ..	130	20	"	25
16/3/40 ..	115	12	"	10
29/3/40 ..	115	5	"	10
12/4/40 ..	60	8	"	20
27/4/40 ..	125	0	"	50

Jugular compression was performed on each occasion and showed a normal rise of pressure from either side. There was therefore no manometric evidence of obstruction of the large dural sinuses.

Comment.—The following points in this case are worth mentioning: (1) The occurrence of bilateral convulsion in the epileptic attacks. This we know may occur with a lesion (for example, a tumour) confined to one frontal lobe. Another explanation, however, may be found in the assumption that tributary veins from both frontal lobes were occluded. (2) The decision, which proved a wise one, not to operate. The argument that so long as the sinuses were draining freely into the nose there was no need for operation proved its soundness. (3) That for

several weeks after all symptoms and signs of the cerebral lesion had cleared up the pleocytosis in the cerebrospinal fluid continued.

Nasopharyngitis

That cerebral thrombophlebitis may follow an illness so trivial as nasopharyngitis at first sight appears unlikely. McAlpine (1937), however, has recorded, under the title of toxic hydrocephalus, cases in which the symptoms of increased intracranial pressure have followed such an infection, and it seems not improbable that in these cases thrombosis of the dural sinuses may have been the cause. It has already been remarked that the venous pathway from nasopharynx to lateral sinus is short and that many instances have been recorded of lateral sinus thrombosis following peritonsillar abscess. Once thrombophlebitis is established in a dural sinus there is no doubt that its spread may be extensive and symptomless. In the following case the diagnosis of cortical thrombophlebitis on clinical grounds seemed highly probable.

Case III

The patient, a boy aged 15, was admitted to a R.A.F. hospital on February 2, 1940. I saw him with Flight Lieutenant Denis Williams, who had care of the case and who has kindly given me his notes. The family and past history were uneventful. The recent story was that a fortnight before admission he was taken ill with sore throat, fever, sweating, and headache. He was ill for six days with an extremely sore throat and intermittent headache. A week before admission he picked up a mug with his left hand, but found he could not hold it. This weakness persisted. Two days later he could not control the muscles of the left side of the face, and on getting out of bed the left knee was weak and the leg dragged. These symptoms had persisted unchanged.

On admission he was mentally clear and alert. The fundi and fields were normal. He showed weakness of the left face, but none of palate or tongue. There was weakness of left upper and lower limbs, but no defect of sensation. The arm-jerks were present and equal, the left abdominal reflexes relatively diminished, the left knee-jerk brisker than the right, the ankle-jerks present and equal, the right plantar flexor and the left occasionally extensor. The cerebrospinal fluid showed a pressure of 120 mm. and contained 130 cells per c.mm., of which 70% were lymphocytes and the remainder red blood cells. The Wassermann reaction was negative in the blood and cerebrospinal fluid.

Following his admission the temperature remained normal. He was well in himself, and at the end of ten days the weakness began to improve, so that by February 20 he had practically recovered. On this day, however, he felt shivery, his temperature rose to 100.4°, and he complained of severe headache and nausea. At the same time there was a recurrence of weakness in the left face and arm, especially in the latter. The left arm-jerks were markedly increased; the left plantar response was doubtful. There were no other abnormal physical signs. By February 23 he had begun to improve again, though still complaining of headache. His temperature had remained normal since February 20. Subsequent recovery was rapid, and he was discharged well on March 8.

Comment.—This case is an example of the paralytic as opposed to the epileptic form of onset, and in some respects resembles Case I. The first onset of weakness was sudden. After an interval of two days there was a sudden extension of the weakness, and after a period of improvement there was a relapse associated with a brief rise of temperature. The pleocytosis in the cerebrospinal fluid was of the kind often seen in proved cases of cerebral thrombophlebitis, and it is difficult to envisage an alternative pathology.

Following Parturition: Case IV

For the notes of this case I am indebted to my colleagues at the National Hospital, Queen Square: Dr. Purdon Martin and Mr. Geoffrey Jefferson.

A married woman aged 32 was admitted to hospital on March 18, 1934, having had frequent left-sided epileptic attacks. On March 4 she had been delivered of her first child. Labour was uneventful, forceps being used in the third stage. The puerperium was normal except for a rise of temperature to 99° on the third day. On March 17 she complained of headache, and noticed a transient feeling of uselessness in the left upper limb. On the 18th, half an hour after waking, she had an attack in which the left arm and then the left side of the face began to twitch. She then lost consciousness for about two minutes, and was drowsy for some time afterwards. She had four more attacks of the same kind in the next five hours.

On admission the patient was drowsy and she complained of headache. There was no neck stiffness or Kernig's sign. The fundi were normal, as were all functions of the cranial nerves except those participating in a left sensorimotor hemiparesis, which was complete in the left upper limb, less marked in the face, and least in the lower limb.

In the first twenty-four hours after admission she had seventeen fits, all of the same nature. For five or six minutes there would be slight clonic movements of the left face and upper limb, with a tendency for the head and eyes to be pulled to the left. The movements generally gained in intensity, spread to the left side of the trunk and lower limb, and then became generalized, with loss of consciousness for two or three minutes. Her blood pressure on admission was 122/84. The general examination was negative. Lumbar puncture on admission showed the cerebrospinal fluid under a pressure of 130 mm. The fluid was slightly blood-stained and yellow, and contained 1,700 red cells per c.mm. with no excess of white cells. The protein was 45 mg. per 100 c.cm.; the Wassermann reaction was negative. During April, although there was some improvement in the hemiparesis, she complained a good deal of headache. By April 23 (five weeks after admission) she had 2D papilloedema in the right eye and 3D in the left, with a few small haemorrhages.

On May 6 lumbar puncture showed a pressure of 240 mm. The fluid was clear, with no cells, and the protein 50 mg. per 100 c.cm. There was continued improvement in the hemiparesis, but on account of the headache and papilloedema an exploration was decided upon. This was performed by Mr. Jefferson on May 24. The dura mater when opened was found to be adherent over the vessels in the Rolandic fissure. In this fissure there was a firm opaque whitish streak. Arterial pulsation was normal, and the impression was gained that what had been seen was a thrombosed vein. The pre-motor cortex looked gummy and softened, but electrical stimulation produced movements in the corresponding limbs.

The patient recovered from the operation, and was discharged on June 26 with only a slight weakness of the left upper limb and left side of the face. She subsequently made a complete recovery and has remained well.

Comment.—In this case it is to be presumed that the extent of the initial thrombosis in the cortical veins and the consequent degree of venous congestion were enough to cause subarachnoid haemorrhage. This has been recorded in other instances, and in fatal cases multiple haemorrhages have been observed on the surface of the brain (Symonds, 1937). The absence of increased intracranial pressure at the onset indicates that at that time there cannot have been any extensive involvement of the superior longitudinal or lateral sinuses. The later development of papilloedema and a high cerebrospinal fluid pressure were evidence of extension of clot in the dural sinuses, while the simultaneous improvement in the weakness indicated an improvement by anastomosis in the local cortical circulation.

Case V

A married woman aged 25 was admitted to Guy's Hospital under my care on June 25, 1939, on account of right-sided weakness and fits. There was no family history of epilepsy, and no personal history of illness except a miscarriage in the third month of pregnancy three years previously. About

three months before admission she had become pregnant again, and on June 14 she miscarried. She remained in bed for a week under medical care. Lochia continued for about ten days in rapidly diminishing amounts. There was no record of any rise in temperature. She complained of a nasty headache towards the end of that time, but was otherwise well. On the morning of June 23 when answering the door she noticed her right knee suddenly give way. The weakness lasted for about a minute and passed off completely. She went for a walk and noticed no abnormality. In the afternoon, again while standing, her right leg momentarily gave way "as if somebody hit me in the back of the knee." This sudden momentary weakness of the right leg recurred about five or six times that day, but this did not disturb the daily routine.

During the night she woke with a numbed sensation of the whole right side of the body except the face as if it had gone to sleep. She slept again, but in the morning found the numb sensation the same, and on getting up discovered her right leg to be so weak that she could hardly stand on it. Later, while preparing the breakfast, she found she could not hold a knife, and had to get her husband to help her with the cooking. By the time she had had breakfast her right side was almost completely paralysed, and she had a bad headache. On the following day at 11 a.m. she suddenly had twitching and jumping movements in the right shoulder which lasted for a few minutes. This recurred a few minutes later. At 4 p.m. the twitching began again and spread to the right thumb and lower limb, continuing for several minutes without loss of consciousness. In the subsequent five hours there were several further attacks of this kind lasting up to fifteen minutes. In one of these she lost consciousness, and the convulsive movements spread to the left side of her body.

On admission she was mentally clear with no dysphasia. The visual fields were full. The fundi and cranial nerves revealed no abnormality. The right upper and lower limbs showed flaccid weakness. There was no defect of sensation. The arm-jerks were absent, the knee- and ankle-jerks were equal but feeble, and both plantar responses were extensor. The cerebrospinal fluid showed a pressure of 200 mm.; there were no cells, and the protein was 0.05%. The Wassermann reaction was negative in the cerebrospinal fluid and blood. The blood pressure was 100/75.

During the first few days after admission the temperature fluctuated between 99° and 100° and the pulse rate between 80 and 100. The hemiparesis remained unchanged. She complained only of headache. On June 28 on waking she complained of weakness of the *left* arm. This passed off in a few days.

Examination of the blood on June 30 showed: Hb, 70%; R.B.C., 4,400,000 per c.mm.; W.B.C., 7,000 per c.mm.; platelets normal; blood-clotting time, 2 minutes 40 seconds; bleeding time, 3 minutes. The blood sedimentation rate was observed as follows: June 6, 24; June 10, 16; July 12, 12; July 14, 14; July 17, 9; July 20, 7; July 27, 5; August 2, 4.

The patient left hospital on August 3 completely recovered and has remained well.

Comment.—Here as in Case I the lower limb was first affected, and at its worst the right hemiparesis spared face, palate and tongue, and speech. The cerebrospinal fluid, as quite often happens, contained no abnormal constituents. It was, however, under increased pressure, which suggested a primary thrombosis of the superior longitudinal sinus. The subsequent occurrence of transient weakness of the left arm supported this hypothesis.

Conclusions

It must be admitted that in all except one of the cases related in this paper visible proof of the diagnosis of thrombophlebitis was lacking. The clinical symptoms, however, exactly resemble those which have been recorded in cases (most of them following lateral sinus thrombosis from otitis media) in which the diagnosis has been proved post mortem. It appears to me probable that cerebral thrombophlebitis by direct extension from an infective

source or occurring as part of a generalized tendency to thrombophlebitis is not very uncommon and frequently runs a benign course. Such cases in the past may have been erroneously attributed to arterial thrombosis or embolism, or to encephalitis. Some, especially those in which the onset has been with focal epileptic attacks, have doubtless been diagnosed as meningitis or abscess. The latter diagnosis has occasionally led to unnecessary and probably harmful operation. I can recall two cases in which I was responsible for this error, and in Case IV above the diagnosis was so much in doubt that it needed an exploratory craniotomy to satisfy physician and surgeon that the patient would make a spontaneous recovery. For these reasons, and in the hope that they may stimulate further inquiry, it has seemed worth recording these few observations.

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THE POSSIBILITIES OF NERVE GRAFTING *

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The problem of bridging a nerve gap urgently demands an answer at the present time. The problem arose in the last war, and attempts were made, by means of multiple strands of cutaneous nerves or lengths of vein and other hollow tubes, to guide the down-growing axons from the proximal end of the nerve across the gap into the distal end. The results on the whole were disappointing, and nerve grafting was discredited until the work of Duel and Ballance (1932). These workers were interested in the problem of bridging a gap in the facial nerve in the Fallopian canal, and they were able to show, in animals and in man, that this gap could be successfully bridged by means of a nerve graft taken from the external cutaneous nerve of the thigh. Their work was fully reported, and it was obvious that they obtained brilliant success in restoring movements to a paralysed face.

In 1935 we started an inquiry, in collaboration with the late Sir Charles Ballance, to determine why there should be a divergence in the results of nerve grafting. Our experiments were carried out in cats and monkeys, and the early results were reported in detail (Bentley and Hill, 1936). They led us to believe that Duel and Ballance's success in facial nerve grafting resulted from the meticulously accurate fit they secured between the graft and the two ends of the facial nerve. They were aided in this by the size of the nerve, so that they were able to obtain a graft of approximately the same dimensions, and also by the helpful circumstance that the graft could be laid in a bony groove and accurate apposition obtained without the necessity for sutures. Our experiments taught us particularly the importance of endoneural scar at the two suture lines in preventing the down-growth of regenerating nerve axons. Any inaccuracy of approximation of graft to nerve or invasion of nerve tissue by sutures caused a great increase in the amount of scar at the anastomosis.

* Abridged from a lecture given at the British Postgraduate Medical School, University of London, July 26, 1940.

with proportionate obstruction to regenerating fibres. Particularly was this true at the distal suture line between the graft and nerve, in which the scar has some weeks to form a barrier before the fibres reach the suture line. It is probable that the disappointing clinical results of cable grafts and vein transplants reported by Platt and Bristow (1919-20, 1923-4) after the last war may be explained on the basis of scar-tissue formation. Our experiments suggest that under the conditions they describe fibrosis would be so extensive as to prevent any nerve fibres reaching the distal end of the nerve.

How a Nerve Graft Functions

We were interested in considering how a nerve graft functions in bridging a nerve gap, and histological studies of our nerve grafts at different periods after operation suggested to us that the graft formed a scaffolding along which the down-growing axons could grow. Duel and Ballance had had the conception of multiple neurilemmal tubes awaiting the passage of regenerating axons, and had believed that it was advantageous to allow the graft to undergo preliminary Wallerian degeneration before using it at operation, in order that the neurilemmal tubes should not be blocked by the products of axon degeneration. In a series of experiments designed to test this hypothesis we could find no difference in the rate of regeneration through fresh or degenerated grafts. The neurilemmal tubes certainly acted as a conducting medium for the new nerve fibres, but the presence of the products of degeneration seemed in no way to hinder the rate of axon growth. The importance of a nerve graft was that it provided the necessary scaffolding or framework along which nerve fibres could grow as easily and rapidly as along an ordinary piece of nerve after section and suture. It would be impossible, we believe, for anything other than nerve to provide this essential scaffolding.

It is possible, therefore, to bridge a nerve gap, given the essential scaffolding that a nerve graft provides and the minimum of endoneural scar at the suture lines. A certain amount of scar formation at the anastomosis is inevitable, but it must be kept to a minimum if a functional result is to be obtained, and we suggested that in order to obtain satisfactory results two things were essential: first, the greatest care and accuracy of technique in suturing, including only the nerve sheath in sutures of fine silk; and, secondly, employment of a graft of the same calibre as the nerve, so that accurate approximation of the cut ends of graft and nerve can be obtained.

Grafting Limb Nerves

How, then, can the problem of the limb nerves be tackled? Multiple strands of cutaneous nerve (cable grafts) do not provide the conditions essential for success, and no donor nerve of the same size as a main limb nerve is available from the patient himself. Nerve grafting in the case of limb nerves becomes impracticable unless it can be shown that a nerve taken from another individual can be used as a graft. This introduces a serious problem. Homeografts (that is, taken from another individual of the same species), in general, are reported not to survive in their new host. There is incompatibility between the cells of the graft and the tissues of the host, and such grafts excite connective-tissue reaction and are ultimately absorbed (Loeb, 1930). We thought it possible, however, that a homeonerve graft might, before it was absorbed, provide the essential temporary scaffolding along which the nerve axons could grow, and that a homeograft might prove successful providing the endoneural scar at the two suture lines was not too extensive. In our previous work (1936) we had shown in cats that a homeograft 3 cm. in length would successfully bridge a short nerve gap.