

$10^9/l$; $69\ 000/mm^3$). Two weeks later he developed painful swelling of the left forearm. Within two days he complained of pins and needles in the ipsilateral thumb and index and middle fingers.

Examination showed a tender, fluctuant swelling below the left elbow anterolaterally, the forearm being 1 cm bigger in circumference than the other. There was also diminished sensation to light touch and pinprick over the dorsal aspects of the lateral three fingers and adjacent part of the hand but no muscle weakness. On investigation the haemoglobin concentration was $14.0\ g/dl$, platelet count $213 \times 10^9/l$ ($213\ 000/mm^3$), and erythrocyte sedimentation rate (ESR) 33 mm in first hour. The Rose-Waaler test was positive (titre 1/128). Plain radiographs of chest, hands, and elbows were normal, but those of the feet showed erosions. During arthrography of the left elbow there was prompt extravasation of injected contrast (Conray 280) into the soft-tissue swelling (see figure). Electrodiagnosis showed a normal right radial nerve sensory action potential of $20\ \mu V$ over the first dorsal interosseous muscle on stimulating 10 cm proximally; on the left, however, there was no potential visible. No denervation was found on electromyography of extensor digitorum communis.

The patient was advised to rest the arm in a sling, and 50 mg hydrocortisone acetate was injected into the elbow. A week later the swelling subsided and the forearm was only 0.5 cm bigger than the other. Penicillamine was reintroduced but not increased above 500 mg daily. Four months later, when the arthritis was well controlled and the ESR 12 mm in first hour, there was no recordable change in nerve conduction. In March 1979, 10 months after the onset of paraesthesiae, sensation was returning in the left thumb.

Comment

Rupture of synovial effusions is a recognised complication of RA and commonly occurs from the knee.² Less often it affects the elbow.³ In our patient synovial rupture was confirmed by arthrography. He experienced paraesthesiae in the hand within two days after noticing the forearm swelling. In keeping with the sensory deficit in the hand, we could not record the radial nerve sensory action potential. Absence of motor nerve disorder, both clinically and electrodiagnostically, suggested an isolated pressure neuropathy affecting the purely sensory superficial terminal branch of the radial nerve as it descends anteriorly over the lateral epicondyle of the elbow. Another explanation is vasculitis with occlusion of the vasa nervorum, which may complicate RA.⁴ Against this in our patient was the absence of digital ischaemia or other cutaneous lesions of vasculitis, usually present in such cases.⁵ Moreover, there was no evidence of neuropathy elsewhere.

¹ Hanna, B D, *et al*, *Rheumatology and Rehabilitation*, 1975, 14, 212.

² Jayson, M I V, *et al*, *Annals of Physical Medicine*, 1969, 10, 175.

³ Goode, J D, *Annals of the Rheumatic Diseases*, 1968, 27, 604.

⁴ Schmid, F R, *et al*, *American Journal of Medicine*, 1961, 30, 56.

⁵ Pallis, C A, and Scott, J T, *British Medical Journal*, 1965, 1, 1141.

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Extracranial to intracranial micro-revascularisation for the treatment of completed ischaemic stroke

Extracranial surgery for internal carotid artery occlusion has had poor results, and attention has turned recently towards the possible improvement of cerebral blood flow by intracranial procedures. Jacobson and Suarez¹ first used the operating microscope for anastomosing small vessels, and Donaghy² reported seven human middle cerebral artery embolectomies using a T tube. Yasargil and Donaghy³ then reported the first successful extracranial to intracranial bypass in man. The end of the superficial temporal artery was anastomosed to the side of a cortical branch of the middle cerebral artery. We report here the results of extracranial to intracranial revascularisation in nine patients.

Patients, method, and results

Completed strokes had occurred between one and a half and 12 months (mean six months) before surgery. The main deficits were weakness of the arm, dysphasia, intellectual deficit, and to a lesser extent, reduction in power in the leg (see table). All patients were alert and had some evidence of retained function in the affected hemisphere. All patients had low density areas comparable with cerebral infarction on computerised axial tomography. The deficits were stable at the time of surgery and were thought unlikely to show further spontaneous improvement. A power record of the affected muscles, an assessment of the speech deficit, and regional cerebral blood flow estimations were performed and repeated after the operation. Psychological assessment was carried out before and after operation; the tests used were of intelligence, verbal fluency (in the left-sided cases), visual perception (in the right-sided cases), and memory for both verbal and visual material.

The operation⁴ was performed by a vascular surgeon with experience in microvascular surgery, together with a neurosurgeon. Using the operating microscope, the end of the superficial temporal artery was anastomosed to the side of an appropriate cortical branch of the middle cerebral artery using interrupted 10/0 sutures.

All nine of the anastomoses remained patent as assessed by arteriography or directional doppler; with a patent anastomosis it is possible to feel the pulsation of the superficial temporal artery. All patients are alive and well at the time of writing, and the procedure had an insignificant morbidity; there was no skin flap sloughing and the scar proved to be neat and lie with the hair line. Cerebral blood flow and oxygen utilisation increased. The first patient of the series had striking improvement in power. Before the operation his right arm had been totally paralysed for six weeks, and within 10 days after the procedure movement appeared, first at the shoulder, then at the elbow, and finally at the wrist. At the end of six weeks his arm movements were completely normal. Three months later his inaccessibly stenosed internal carotid artery occluded and he suffered a recurrent completed stroke. His deficit on this occasion was severe but the arm was relatively spared. Most patients had a reduction in tone with improvement in power, enabling an improvement in gait and stability (see table). The results of psychological testing were summarily expressed as differences between preoperative and

Summary of results in nine patients

Case No	Age and sex	Presentation	Duration of history before surgery (months)	Clinical neurological outcome	Difference on psychological tests	
					Verbal (intelligence/fluency/memory)	Non-verbal (intelligence/perception/memory)
1	55 M	Completed stroke, R arm totally paralysed	1½	R arm returned to full power	+5/+2/+1	+2/-/+1
2	46 M	Completed stroke, weakness L arm and leg, mild dysphasia, no sensory loss, left handed	4	Improvement in power and walking ability	+1/-/+1	0/+5/0
3	59 M	Completed stroke after TIA, R arm and leg weakness, dysphasia	1½	Improvement in power and walking ability		
4	52 F	Completed stroke, weakness and in coordination L arm	9	Improvement in left arm power	-1/-1/-1	0/+8/+1
5	69 M	Completed stroke, weakness in L side of face, arm and leg	4	Improvement in power, speech, and walking ability	0/-/0	0/-/+2
6	57 M	Completed stroke after weakness in R arm	7	Improvement in power, speech and walking ability	+1/-/+2	+1/-/-2
7	67 M	Completed stroke, weakness R arm and leg, poor memory	6	No change	+3/+6/+2	-1/-/-6
8	62 M	Completed stroke, weakness R arm and leg, severe dysphasia	12	No change		
9	57 M	Intellectual deterioration, R carotid occlusion	3	Further deterioration	-4/-/-2	-4/-/0

postoperative scores, and the units are not comparable across categories. All of the patients who had a left-sided procedure showed some degree of improvement in verbal abilities, while both the patients who had a right-sided operation in whom visual perception was tested showed improvement in this function.

Comment

These preliminary results suggest that extracranial to intracranial bypass may produce an improvement in some patients with long-standing moderate completed ischaemic stroke. As some of our patients had a longstanding total paralysis of arm before surgery, these deficits may have been regarded as too severe to fall within the criteria for entry into the Canadian co-operative study.⁵

¹ Jacobson, J H, and Suarez, E L, *Surgical Forum*, 1960, **11**, 243.

² Donaghy, R M P, in *Microvascular Surgery*, ed R M P Donaghy and M G Yasargil, p 75. Stuttgart, Georg Thieme, 1967.

³ Yasargil, M G, *Microsurgery Applied to Neurosurgery*, p 105. Stuttgart, Georg Thieme, 1969.

⁴ Greenhalgh, R M, and Illingworth, R D, in *Progress in Stroke Research 1*, ed R M Greenhalgh and F Clifford Rose, p 393. London, Pitman Medical, 1979.

⁵ McDowell, F H, *Stroke*, 1977, **85**, 545.

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How quickly can haemoglobin A₁ increase?

The proportion of glycosylated haemoglobin (HbA₁) correlates with blood and urine glucose measurements, especially those made one to two months before.¹ HbA₁ is particularly valuable in assessing diabetic control, since its concentration is independent of short-term fluctuations in blood glucose. There is evidence that HbA₁ decreases one to two months after improved regulation of blood glucose,² though the time it takes to increase after development of hyperglycaemia is uncertain. A rise in HbA₁ was observed four weeks after development of hyperglycaemia in mice³ and six weeks after relaxation of control in postpartum diabetics.⁴ A significant increase in HbA₁ was also observed in normal people about one month after a glucose load.⁵ We decided to find how quickly a period of hyperglycaemia can lead to increased HbA₁ concentrations.

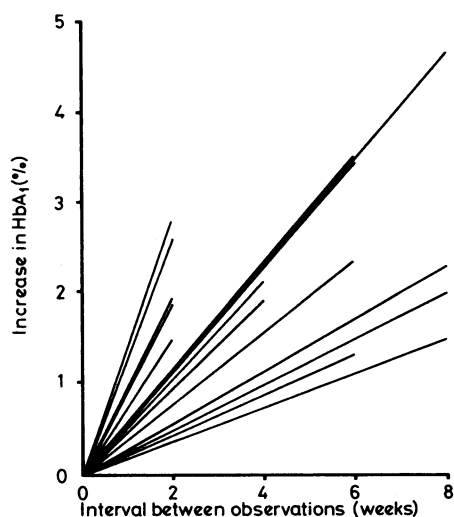
Patients, methods, and results

Twenty-nine insulin-dependent diabetics were studied. In each case HbA₁ was estimated at intervals of two to eight weeks over eight months by macrocolumn chromatography. Diabetic control was assessed with regular urine tests and morning blood glucose estimations. Of the 131 HbA₁ estimations performed, only those in which HbA₁ was increased are reported here.

Comparing each observation with the immediately preceding one we found an increase greater than 1% in 15; of these, six had increased more than 2% and three more than 3%. Five showed an increase in HbA₁ by two weeks (see figure). HbA₁ increased from a range for well-controlled diabetes⁴ in three patients (at intervals of two, four, and six weeks respectively). Before 11 of the 15 estimations hyperglycaemia had worsened: before six of these 11 patients developed persistent 2% glycosuria (four with symptoms), while five of the estimations were from postpartum diabetics whose control had been deliberately relaxed after delivery.

Comment

Most patients showed little change in their HbA₁ concentrations over two months. Some, however, showed large increases within even two weeks, and in two patients HbA₁ increased from a range for well-



Percentage increases in haemoglobin A₁ in 15 subjects according to interval between observations.

controlled diabetes by four weeks. Thus a single HbA₁ estimation may relate to a shorter period of hyperglycaemia than has been suggested.¹ Such rapid changes in HbA₁ limit its value in assessing diabetic control retrospectively, and conclusions about diabetic control drawn from single HbA₁ estimations should be treated with caution.

¹ Koenig, R J, *et al*, *New England Journal of Medicine*, 1976, **295**, 417.

² Ditzel, J, and Kjaergaard, J-J, *British Medical Journal*, 1978, **1**, 741.

³ Koenig, R J, and Cerami, A, *Proceedings of the National Academy of Sciences of the United States of America*, 1975, **72**, 3687.

⁴ Leslie, R D G, *et al*, *Lancet*, 1978, **2**, 958.

⁵ Maquert, F X, *et al*, *Lancet*, 1978, **2**, 431.

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Upper airways obstruction after Dettol ingestion

The following case report describes a hitherto unreported effect of Dettol ingestion which may be important in the management of Dettol poisoning.

Case report

A 22-month-old girl was admitted to hospital 40 minutes after drinking a maximum of 125 ml Dettol. She complained of a sore mouth but had not vomited. Her birth weight had been 1960 g at 33 weeks' gestation. She had been asphyxiated at birth and developed the idiopathic respiratory distress syndrome, from which she had recovered quickly. There was no record of tracheal intubation or controlled ventilation during this period. She had not been prone to upper respiratory tract infections subsequently and had had no hoarseness or noisy breathing. She had had no respiratory symptoms or contact with infectious illnesses immediately before ingesting Dettol.

On arrival in hospital she was conscious and alert. Her temperature was 36.5°C, pulse 112/min, and respiration 28/min. She had mild inspiratory stridor and erythema of the chin and anterior chest wall. Her buccal mucosa was inflamed. Vomiting was induced with syrup of ipecachuana. Thereafter she became progressively more stridulous with subcostal and intercostal retractions. She did not appear to have inhaled vomitus, and there were no signs suggestive of aspiration on a chest radiograph. Within two hours of vomiting she had pronounced stridor with severe retractions and was in imminent danger of complete upper airway obstruction. A nasotracheal tube was passed under general anaesthesia, which immediately relieved her