Discussion

One of the main aims in the management of head injuries is to ensure that complications are prevented or, if this is not possible, recognised soon enough to institute effective treatment. Even with reasonably prompt operations only a proportion of intracranial haematomas may be successfully treated, but this study indicates that when the operation is substantially delayed after clinical signs have been detected the mortality is increased. Indeed, in this series several intracranial haematomas were first discovered at necropsy.

The commonest reason for failing to recognise an intracranial haematoma is mistakenly attributing the depressed conscious level to a cerebrovascular accident or excess alcohol. When neither of these misdiagnoses confuses the issue the reasons are more difficult to determine, and probably more than one factor often plays a part. Much must depend on the reliable recording of changes in the conscious level and the communication of these from nurse to doctor and from one doctor to another in the general hospital, and from the primary surgeon to the neurosurgeon (usually by telephone). Problems may arise at each of these levels: the primary surgeon may underrate what the nurse reports, or the neurosurgeon may undervalue what the primary surgeon reports. So restricted is the number of neurosurgical beds in many British cities that there is often a reluctance to accept head-injured patients for transfer until the diangosis of haematoma is so definite that deterioration is already advanced[5]; this was certainly a problem in Glasgow at times during the period of this study. When a previously lucid patient deteriorates to the extent of being in coma the diagnosis is easy, but when a patient's conscious level is already altered and it becomes deeper it can be less so, particularly for staff who are not trained or experienced in the observation of acutely brain-damaged patients. It was to facilitate such observations and their communication between nurse and doctor, and doctor and neurosurgeon that the Glasgow Coma Scale[6] was introduced and with it the Glasgow Head Injury Observation Chart.[7]

This study, however, has also defined two clear causes of confusion: the cerebrovascular accident and the drunk. The patient thought to be suffering from the effect of excess alcohol can present a difficult problem, as MacEwan first pointed out in 1879.[8] If there is clear evidence of clinical deterioration or there are focal neurological signs or there is a fractured skull, then traumatic intracranial haematoma must be excluded before the clinical state is ascribed to alcohol. In a patient who has taken alcohol and is in coma without focal signs or fracture some help may be obtained from estimating the blood alcohol concentration; if it is under 43·4 mmol/l (200 mg/100 ml) altered consciousness is unlikely to be due to alcohol alone.[9] Whether a traumatic intracranial haematoma is present, however, can be resolved only by further investigation.

In the patient suspected of having had a cerebrovascular accident the detection of a fractured skull is the best clue to management. All but four of the 33 patients with traumatic haematomas whose conditions were incorrectly diagnosed as vascular accidents had skull fractures. By contrast, in a retrospective study of 1000 consecutive patients with cerebrovascular accidents in a local teaching hospital 132 underwent skull *x*-ray examination and not one had a fracture. If a patient suspected of having had a cerebrovascular accident has a skull fracture his condition is likely to be due to a traumatic intracranial haematoma.

Intracranial haematoma may be difficult to diagnose clinically, and the best chance of recognition rests on an awareness by the doctor of the possibility. Even so, there will always be instances of delay in making the diagnosis and even of discovery at necropsy. The EMI scan appears to be a reliable method of detecting haematoma[10] but it may be some time before it becomes generally available.

References

1 McKissock, W, *et al*, *Lancet*, 1960, **2**, 167.
2 Jamieson, K G, and Yelland, J D N, *Journal of Neurosurgery*, 1968, **29**, 13.
3 McLaurin, R L, and Tutor, F T, *Journal of Neurosurgery*, 1961, **18**, 61.
4 James, T G I, and Turner, E A, *Lancet*, 1951, **2**, 45.
5 Jennett, B, *British Medical Journal*, 1975, **3**, 267.
6 Teasdale, G, and Jennett, B, *Lancet*, 1974, **2**, 81.
7 Teasdale, G, Galbraith, S, and Clark, K, *Nursing Times*, 1975, **71**, 973.
8 MacEwan, W, *Glasgow Medical Journal*, 1879, **11**, 1.
9 Galbraith, S, *et al*, *British Journal of Surgery*, 1976, **63**, 128.
10 Galbraith, S, *et al*, *British Journal of Surgery*, 1976, **63**, 157.

Carpal tunnel syndrome, humeral epicondylitis, and the cervical spine: a study of clinical and dimensional relations

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Summary

Forty-three patients with idiopathic carpal tunnel syndrome, confirmed by nerve conduction studies and treated by surgery, were compared clinically and radiologically with 43 age- and sex-matched control patients. Patients with carpal tunnel syndrome had a significantly greater prevalence of lateral humeral epicondylitis (tennis elbow) (33%) than controls (7%). Randomised reading of the cervical spine radiographs in ignorance of the groups to which they belonged showed no significant difference in the prevalence of either intervertebral disc degeneration or intraforaminal osteophyte protrusion using conventional grading methods. Measurement of the minimum anteroposterior diameter of the cervical spinal canal, the anteroposterior diameters of the cervical vertebral bodies, and the ratio of intervertebral disc height to adjacent vertebral body height in the cervical spine, however, showed a consistent trend to smaller measurements in the carpal tunnel group. Differences were significant at several vertebral levels in each of these dimensions. The narrowing of the intervertebral discs relative to the vertebral bodies in patients with carpal tunnel syndrome may indicate connective tissue changes, which might also occur in the common extensor origin at the elbow or in the contents of the carpal tunnel.

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Introduction

In most cases of the carpal tunnel syndrome a clear cause for the compression of the median nerve at the wrist cannot be shown, although the compression must be due to either an increase in the volume of the contents of the carpal tunnel or a relative inability of the carpal tunnel to accommodate its contents, or both. While there is no evidence to suggest that the volume of the carpal tunnel itself may be reduced, an abnormality of the nine tendons (or their sheaths) that pass through the canal is believed by some to cause the syndrome.[1 6] Hence the reported association between carpal tunnel syndrome and various forms of "non-rheumatoid" tenosynovitis of the wrist and hand is of interest.[4 7 8]

Idiopathic carpal tunnel syndrome has also been noted in association with upper arm and shoulder pain,[1 9–12] cervicobrachial pain,[13–16] and tennis elbow.[1 2 4] Since none of these series of patients were compared with controls, however, it is difficult to know whether any of these reported associations are significant. We describe here a study of patients with idiopathic carpal tunnel syndrome that was designed to investigate certain possible clinical associations of the syndrome, in particular, humeral epicondylitis, and to make certain radiological measurements.

Patients and methods

The hospital records of all patients who had had surgery for the carpal tunnel syndrome in 1970-3 at the General Infirmary at Leeds were reviewed. Patients were invited to return for a clinical and radiological examination provided that (*a*) the median nerve compression had been shown by delayed sensory conduction at the wrist with reference to the normal values determined at Leeds[17]; (*b*) the patient was aged 55 or under at the time of review; and (*c*) there was no apparent underlying cause for the median nerve compression. Of the 70 apparently suitable patients 53 (76%) attended for review, but 10 of these had to be excluded as other causes were found.

The remaining 43 patients (39 women and four men) were examined for the presence of humeral epicondylitis, periarthritis of the shoulder, or tenosynovitis, and their response to surgical decompression of the median nerve was assessed. Tennis elbow was diagnosed when either considerable localised tenderness was found at the lateral humeral epicondyle at review or a history of tennis elbow had been substantiated by the doctor treating it. Periarthritis of the shoulder was diagnosed when either passive glenohumeral abduction was less than 90°, or internal rotation of the shoulder with the arm abducted to 90° was less than 45°, or external rotation of the shoulder with the arm abducted to 90° was less than 45°.

Haemoglobin estimations, blood sedimentation rate measurements, the differential agglutination test for rheumatoid factor, protein-bound iodine estimations, and lateral and oblique radiographs of the cervical spine were also carried out in each patient.

Forty-three control patients matched for age and sex were obtained from a trauma clinic and underwent a similar clinical review and the same radiological examination. All radiographs from both groups were numbered and assessed in randomised sequence for (*a*) intervertebral disc degeneration at each vertebral level on the lateral radiograph using standard epidemiological grades;[18] and (*b*) intervertebral foraminal narrowing by osteophytes on the right and left oblique radiographs with four grades of severity—1 = doubtful, 2 = mild, 3 = moderate, 4 = severe—based on the moderate and severe grades used by others.[19]

The minimum anteroposterior diameter in the sagittal plane of the spinal canal,[20 21] the anteroposterior diameter of the cervical vertebral bodies midway between the vertebral end plates, and the ratio of intervertebral disc height to body height (fig 1) were measured on all the lateral cervical spine radiographs (film focus distance = 180 cm) for both groups of patients.

The interobserver and intraobserver coefficient of variation for each of the measurements was satisfactory in 1480 observations.

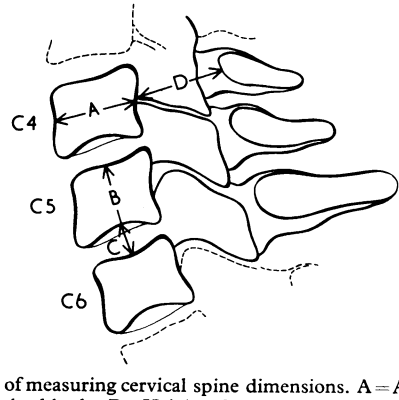

FIG 1—Methods of measuring cervical spine dimensions. A = Anteroposterior diameter of vertebral body. B = Height of vertebral body. C = Intervertebral disc height. D = Minimum anteroposterior diameter of spinal canal of sagittal plane. C:B = Disc: body ratio.

Results

The carpal tunnels of 59 wrists were decompressed in 43 patients, 16 of the patients having the operation on both wrists. In addition to an abnormal delay in sensory conduction in the median nerve at the wrist in every case, there was an abnormal delay in motor latency at the wrist in 43 wrists.

CLINICAL ASSOCIATIONS IN PATIENTS WITH CARPAL TUNNEL SYNDROME

Tennis elbow was confirmed in 14 (33%) patients. In 11 patients evidence of tennis elbow was present at the review examination, and in four patients the wrist extension test was positive. In six patients the tennis elbow had developed a mean of 22·6 months after successful relief of the hand symptoms of median nerve compression on the same side. In two patients the condition had apparently begun while the patient was suffering from the hand symptoms of median nerve compression but had remained unrelieved after section of the flexor retinaculum. In a further patient tennis elbow had developed and resolved early in the development of the hand symptoms of median nerve compression. A further five patients, although having clear evidence of tennis elbow at review, could not remember whether this had predated their wrist operation, which had otherwise been most successful.

Follow-up of patients with tennis elbow diagnosed at the review examination showed that the condition lasted no longer than 15 months, with nine cases resolving spontaneously and five resolving after hydrocortisone injection. No difference could be found between the patients with and without tennis elbow in the electrical severity of the median nerve compression, length of time from surgery to review, or the adequacy of the surgical relief of the median nerve compression.

Medial humeral epicondylitis—A chronic bilateral medial epicondylitis was found in three patients. In two women it was associated with bilateral tennis elbow and in one man it occurred alone and appeared to be caused and perpetuated by occupational stresses.

Periarthritis of the shoulder was found in three women; in two it appeared to be a chronic condition and in the other was acute in onset, lasted 12 months, and resolved spontaneously.

CONTROL PATIENTS

Tennis elbow was diagnosed in three patients (7%). In one patient this elbow tenderness may have been secondary to the use of a crutch. No cases of periarthritis of the shoulder were found.

RADIOLOGICAL FINDINGS

Intervertebral discs—A low and similar prevalence of disc degeneration was found in both groups. When the ratios of the intervertebral disc heights to vertebral body heights were compared in the two groups, however, a trend to smaller values was found in the patients with carpal tunnel syndrome, which reached statistical significance at four levels (fig 2).

Foraminal osteophytes—No significant differences could be found in the frequency of the foraminal osteophytic encroachment between the two groups.

Minimum anteroposterior (sagittal) diameter of cervical spinal canal—Comparison of this dimension at each vertebral level between patients

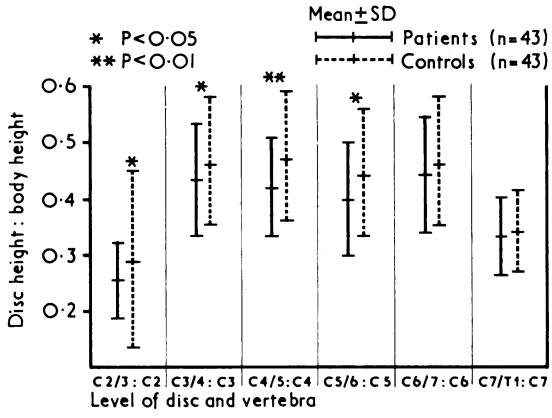

FIG 2—Ratios of intervertebral disc space height to vertebral body height in patients with carpal tunnel syndrome and controls.

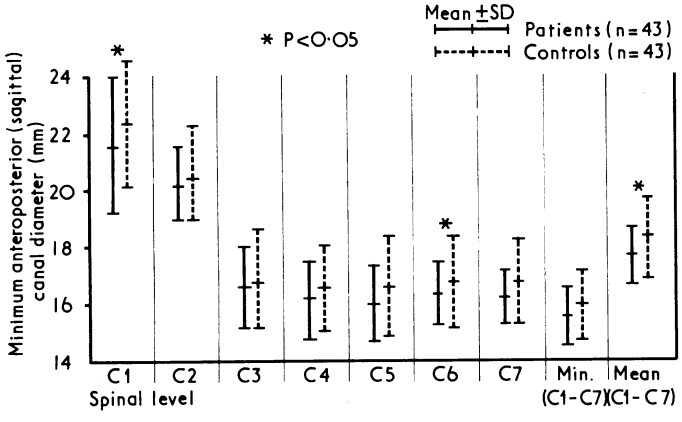

FIG 3—Comparison of minimum anteroposterior (sagittal) canal diameters in patients with carpal tunnel syndrome and controls.

and controls showed a trend to smaller measurements in the former group which reached statistical significance at three levels (fig 3). When these dimensions were compared in the patients with associated tennis elbow and the controls it was apparent that patients who had tennis elbow associated with their carpal tunnel syndrome had the smallest cervical spinal canals (fig 4).

Anteroposterior diameters of cervical vertebral bodies—The same trend towards smaller measurements, which reached statistical significance at two levels, was again found in the patients with carpal tunnel syndrome (fig 5).

Discussion

Our findings showed an association between lateral humeral epicondylitis and idiopathic carpal tunnel syndrome when a closely defined group of patients with the latter condition was reviewed six to 48 months after median nerve decompression. Clinical evidence suggests that the tennis elbow found in these cases is not a symptom of median nerve compression, is not related to the frequently associated forearm pain, but is a distinct rheumatic syndrome occurring in the same patients.

Three sets of measurements were made on the cervical spine radiographs, and the reproducibility of all three was checked within one and between two observers. For all measurements patients in the carpal tunnel group had smaller values than age- and sex-matched controls. Possibly patients with median nerve entrapment at the wrist may have smaller bony carpal tunnels which predispose to this compression when other factors become operative. Nevertheless, our results give no direct evidence for this, as no other bony structures were measured. The carpal tunnel group did not differ in body height or weight from the control group.

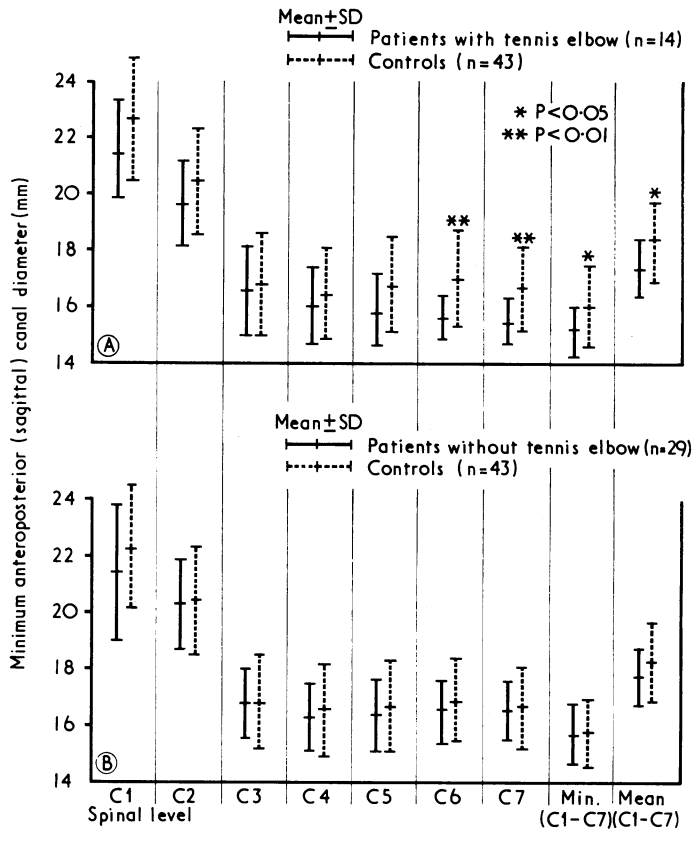

FIG 4—Comparison of minimum anteroposterior (sagittal) canal diameters in (A) patients with carpal tunnel syndrome and tennis elbow and controls and (B) patients with carpal tunnel syndrome without tennis elbow and controls.

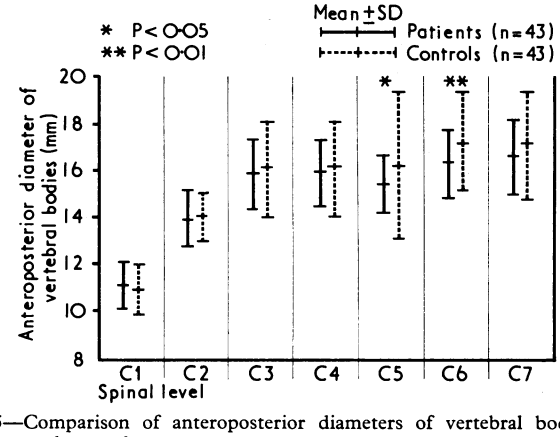

FIG 5—Comparison of anteroposterior diameters of vertebral bodies in patients and controls.

Some authors have suggested that one factor predisposing to carpal tunnel syndrome is compression of some of the cervical nerve roots in the neck, and the finding of smaller cervical spine dimensions in the carpal tunnel group possibly supports this.[16] Nevertheless, since the cross-sectional area of cervical nerve roots occupies only one-fifth to one-quarter of the cross-sectional area of their exit foramina this seems an unlikely explanation.[22] In the context of a cervical spine disorder being related to the carpal tunnel syndrome some workers have noted that humeral epicondylitis is associated with cervicobrachial pain.[23 24]

The narrowing of the cervical intervertebral discs relative to the adjacent vertebral body in those with carpal tunnel syndrome might be evidence of degeneration in soft tissues. This may reflect connective tissue changes which are also present in sites

such as the common extensor origin at the elbow or in the contents of the carpal tunnel.

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References

[1] Phalen, G S, *Journal of Bone and Joint Surgery*, 1966, **48A**, 211.
[2] Yamaguchi, D M, *et al*, *Minnesota Medicine*, 1965, **48**, 22.
[3] Mangini, U, *Bulletin of the Hospital for Joint Diseases*, 1961, **22**, 56.
[4] Lipscomb, P R, *Clinical orthopaedics*, 1959, **13**, 164.
[5] Nissen, K I, *Journal of Bone and Joint Surgery*, 1963, **45B**, 620.
[6] Nissen, K I, personal communication, 1975.
[7] Hombal, J W R, and Owen, R, *Hand*, 1970, **2**, 192.
[8] Conklin, J E, and White, W L, *Surgical Clinics of North America*, 1960, **40**, 431.
[9] Dick, T B S, and Zadik, F R, *British Medical Journal*, 1958, **2**, 288.
[10] Kremer, H, *et al*, *Lancet*, 1953, **2**, 590.
[11] Garland, H, *et al*, *British Medical Journal*, 1957, **1**, 730.
[12] Kummel, B M, and Zazanis, G A, *Clinical Orthopaedics*, 1973, **92**, 227.
[13] Russell, W R, *Proceedings of the Royal Society of Medicine*, 1955, **49**, 197.
[14] Crymble, B, *British Medical Journal*, 1968, **3**, 470.
[15] Semple, J C, and Cargill, A O, *Lancet*, 1969, **1**, 918.
[16] Upton, A R M, and McComas, A J, *Lancet*, 1973, **2**, 359.
[17] Kemble, F, and Peiris, O A, *Electromyography*, 1967, **7**, 127.
[18] Council for International Organisations of Medical Sciences, *Epidemiology of Chronic Rheumatism*, vol 2, Atlas of Standard Radiographs of Arthritis. Oxford, Blackwell, 1963.
[19] Pallis, C, *et al*, *Brain*, 1954, **77**, 274.
[20] Burrows, E H, *Clinical Radiology*, 1963, **14**, 77.
[21] Nurick, S, *Brain*, 1972, **95**, 87.
[22] Wilkinson, N, in *Cervical Spondylosis: Its Early Diagnosis and Treatment*, ed M Wilkinson, p 44. London, Heinemann, 1970.
[23] Silberstein, C E, *Clinical Orthopaedics*, 1965, **40**, 184.
[24] Storey, G O, in *Cervical Spondylosis: Its Early Diagnosis and Treatment*, ed M Wilkinson, p 147. London, Heinemann, 1970.

Transplantation of tumour with a kidney graft

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Summary

A cerebral glioma discovered by angiography and brain biopsy in a kidney donor was subsequently suspected of being a secondary tumour. By this time a biopsy of one of the transplanted kidneys had shown a clump of malignant cells in a glomerulus. Because of the psychological state of this recipient the transplant was not removed, but the recipient of the second kidney was immediately told of the danger of tumour cell transfer, and underwent nephrectomy. The patient remained well on haemodialysis; multiple sectioning of the kidney showed no signs of tumour. The transplant in the first recipient functioned well until his death, six months after operation. At necropsy undifferentiated tumour was found in the pleura, liver, pelvic peritoneum, and transplanted kidney.

All cadaver donors should undergo full laparotomy after removal of the kidneys, particularly those with a high risk of cancer, and a full necropsy should also be performed shortly afterwards to exclude tumour and other unsuspected diseases. Then it is not too late to remove a transplanted kidney should a tumour be found.

Introduction

The possibility of transplanting tumour tissue with a kidney graft is a danger that must always be borne in mind. The tumour may be of renal origin[1 2] or it may arise from structures outside the kidney.[3 4] It is difficult "successfully" to transplant malignant tissue into a normal person, but after immunosuppression host resistance is lowered, with subsequent survival, multiplication, and dissemination of tumour cells. Transplantation of organs from donors with malignant disease should thus not be performed even if the tumour is apparently localised (apart from primary brain tumours), as one cannot be certain whether or not microscopic spread has occurred.

The difficulties encountered in prompt detection of a tumour in the cadaver donor, the advice given to the recipients after transplantation of kidneys at risk, and subsequent management are illustrated in the following report of a donor and two recipients.

Donor

The donor was a 45-year-old male chronic schizophrenic who was transferred to a general hospital because of a fit and the rapid onset of deep unconsciousness. He developed respiratory depression and required ventilation to enable a diagnosis to be made. A cerebral angiogram showed a cerebral tumour, which a brain biopsy showed to be a glioma. On these grounds the patient was deemed to be suitable as a kidney donor. After certification of death both kidneys were removed for transplantation with a warm ischaemic time of 13 minutes. At nephrectomy no intra-abdominal disease was found. A chest *x*-ray picture was normal. A full necropsy was, however, not performed.

After the renal transplants had been performed the radiologist queried the angiographic appearances as being of a secondary rather than a primary brain tumour and the pathologist gave a cautious interpretation of the brain biopsy in view of the necrosis present.

Two patients received kidneys through the National Organ Sharing Scheme on the basis of HLA match. The tissue typing is shown in the table. Preservation was with a cold flush with dextran 40 in dextrose and ice storage. Anatomically the kidneys were normal and no features of pathological significance were seen on the cortical surface or in the hilum.

Tissue typing of donor and two recipients

			HLA-A		HLA-B	
Donor	A	Rh+	2	11	W 5	W 10
Recipient 1	A	Rh+	2	3	W 5	W 10
Recipient 2	A	Rh+	2	W 29	W 5	W 19

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