LESSON OF THE MONTH

Median nerve injury: an underrecognised complication of brachial artery cardiac catheterisation?

Angus M Kennedy, Michael Grocott, Martin S Schwartz, Hamid Modarres, Mark Scott, Fred Schon

Abstract

***Objective*—To describe the local neurological complications associated with cardiac catheterisation via the right brachial artery.**

***Methods*—A follow up study to determine the mechanism of injury and outcome of patients who sustained a high median nerve palsy after this procedure. Five right handed patients were identified in a 24 month period. Each was assessed clinically and electrophysiologically at presentation. All were followed up initally (range six to 22 months) clinically, electrophysiologically, and using components from the Chessington occupational therapy neurological assessment battery (COTNAB) functional hand assessment.**

***Results*—The incidence of this complication was between 0.2 and 1.4%. Three mechanisms of injury were identified. These included direct nerve compression due to formation of antecubital fossa haematoma, direct nerve trauma, and ischaemia secondary to brachial artery occlusion. The initial neurological and nerve conduction deficits improved with time. However, all cases had persistent disability in hand function as documented clinically and on the dexterity and stereognosis subcomponent of the COTNAB test.**

***Conclusion*—This is an uncommon, but probably underrecognised complication. Those performing cardiac catheterisation via the right brachial artery should be aware of the potential risks of damage to the median nerve. They should evaluate hand function after the procedure and take prompt action if median nerve dysfunction is noted. Damage to the median nerve results in appreciable long term disability, which may have medicolegal relevance.**

(*J Neurol Neurosurg Psychiatry* 1997;**63**:542–546)

Keywords: median neuropathy; brachial artery; left heart catherisation; complication

Regional Neuroscience Unit, Atkinson Morley's Hospital, St George's Health Care Trust, Copse Hill, Wimbledon, London SW20 0NE,UK
A M Kennedy
M Grocott
M S Schwartz
H Modarres
F Schon

Department of Plastic and Reconstructive Surgery, Queen Mary's University Hospital, Roehampton, London SW15 5PN, UK
M Scott

Correspondence to:
Dr Fred Schon, Regional Neuroscience Unit, Atkinson Morley's Hospital, St George's Health Care Trust, Copse Hill, Wimbledon, London SW20 0NE, UK.

Received 23 August 1996
and in revised form 18 March 1997
Accepted 25 March 1997

The advent of coronary artery bypass grafting and percutaneous coronary artery angioplasty has led to a considerable increase in coronary angiography. Left heart catheterisation is usually performed via the right common femoral artery, but the right brachial artery may be used when the femoral artery is diseased or when monitoring of aortic valve pressure is required. The most serious, but rare, complications are either due to allergic contrast media reactions or to myocardial infarction in those with severe left main stem disease.[1 2] Neurological complications include an estimated 0.2% risk of CNS thromoboembolism, which characteristically results in posterior cerebral artery territory stroke.[3] Local complications include arterial thrombosis, haematoma formation, and false aneurysm formation.[4] Although peripheral nerve damage associated with arterial cannulation is recognised, little has been written about it. In a recent prospective study of cardiac catheterisation via the femoral artery, damage to the adjacent femoral nerve occurred in 20 out of 9585 cases (0.2%) and, although initially disabling, was reported to be almost completely reversible.[5] There are occasional reports of brachial plexus and median nerve damage complicating the axillary and brachial artery approach, although no follow up data have been provided.[6–9] We present what may be the first comprehensive study of median nerve damage associated with cardiac catheterisation in five patients followed up for between 18 to 34 months.

Patients and methods

The five patients with median nerve damage were referred in a two year period between 1992 and 1994 from two hospitals where brachial artery catheterisation is routinely performed. During this time a total of 350 brachial artery procedures were performed. All patients were examined clinically by a neurologist at presentation and the diagnosis confirmed by neurophysiological assessment. Baseline investigations to exclude predisposing risk factors for peripheral neuropathy were performed. Regular clinical and neurophysiological follow up was made. Motor function for muscles

innervated by the median, ulnar, and radial nerves was quantified using the Medical Research Council's scale for recording muscle power.[10] The individual muscle scores were summed to determine a composite motor score for each nerve. The degree of disability was assessed using three components from the Chessington occupational therapy neurological assessment battery (COTNAB): (*a*) stereognosis and tactile discrimination, (*b*) manual dexterity, and (c) coordination. This test enabled disability in hand function to be quantified and compared with normal control subjects. Three performance grades were identified: 0 normal; 1 borderline performance; and 2 impaired performance.[11]

Results

The results suggest an incidence figure of 1.4% for median nerve damage after brachial artery catheterisation. However, this may be an overestimate as we failed to identify additional cases with this complication in the eight year period preceding this study in one hospital

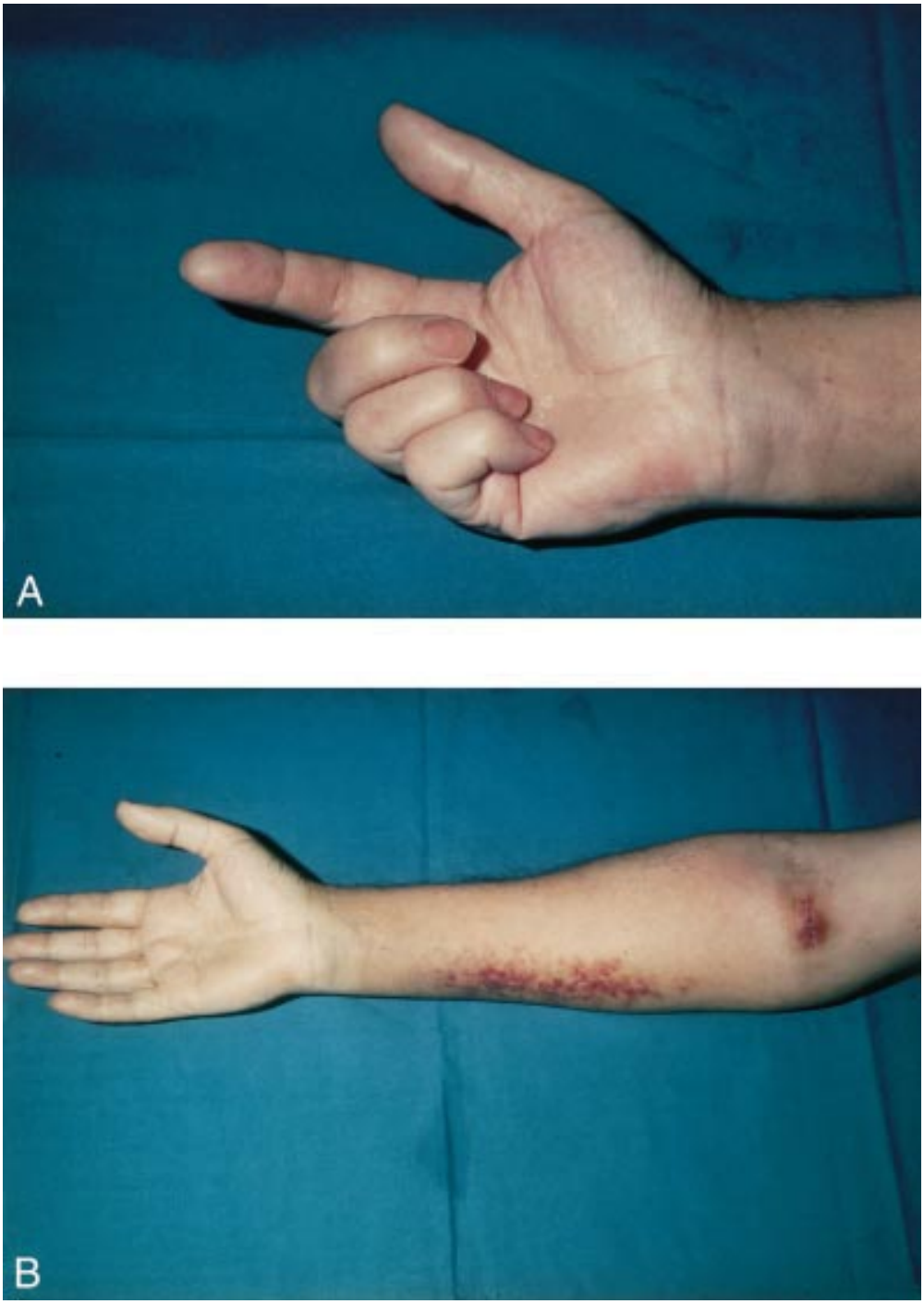

Figure 1 (A) Orator's hand posture: The patient has been asked to make a fist. The hand is held in an "orator's hand" posture. This is typical of a high median nerve palsy, in which there is paralysis of the flexor pollicis longus and the flexor digitorum profundus of the second digit. This leads to an inability to pinch together the thumb and index finger. (B) The extensive bruising of the right forearm was noted two days after the angiogram in patient 1.

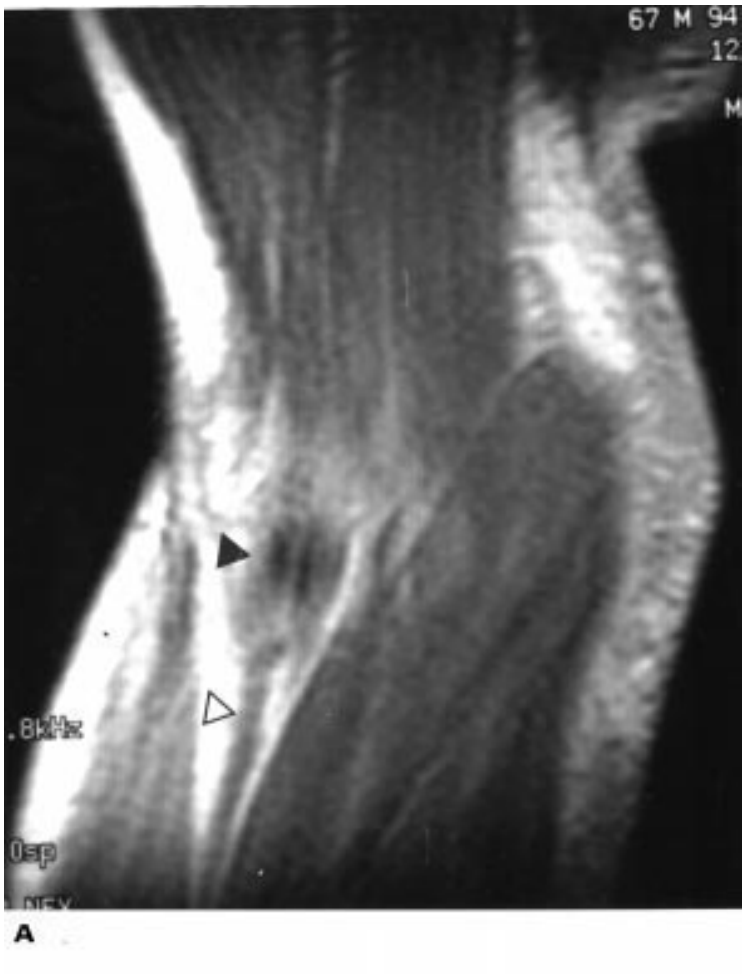

Figure 2 (A) T1 weighed MRI of the forearm of patient 1. The black arrow indicates the haematoma at the antecubital fossa. The white arrow indicates the radial artery as it crossed the antecubital fossa. (B) The haematoma has been removed and the nerve is being explored surgically. The area of focal nerve compression with intraneural haematoma is shown at the tip of the scissors.

where detailed records existed. The exact incidence of this complication remains unclear, although our data show that it occurs in between 0.2 and 1.4% of all brachial artery procedures. Two patients were men and three were women and the age at angiography ranged from 59 to 74 with a mean of 66 years. Two patients had hypertension, but no patient had diabetes mellitus or other systemic disease known to predispose to peripheral neuropathy. Patient 5 had a right hemiparesis, due to an earlier stroke. No patient was on long term oral anticoagulation before the procedure. Various factors causing nerve damage were identified. Patient 1 had a large antecubital haematoma (see below and fig 1). Patient 2 sustained direct median nerve trauma as a result of ligature misplacement. Patient 3 developed symptoms at a delayed interval and was found to have an asymptomatic peripheral neuropathy, in addition to the high median nerve palsy, on nerve conduction studies. Patients 4 and 5 developed brachial artery occlusions which were confirmed by digital subtraction angiography. Patient 4 required three further operations to restore arterial circulation. The medical records did not permit accurate identification of the type or duration of procedures used to secure haemostasis.

Median nerve damage was clinically evident in all cases. Each patient, when asked to make a clenched fist, showed the orator's hand sign (fig 1A) This posture, indicative of high median motor nerve dysfunction, results from paralysis of the flexor pollicis longus muscle, the flexor digitorum profundus muscle of the second digit, and the muscles of the thenar eminence. It results in a serious disability as the patient is unable to pinch together the thumb and index finger. A forearm haematoma as a result of the procedure was seen only in patient 1, who was referred immediately after the procedure (fig 1B). Figure 2 shows the T1 weighed MRI of the right antecubital fossa from this patient. A "black" haematoma in the antecubital fossa was identified (indicated by the arrow in fig 2) and the patient was referred for surgical exploration. This subsequently confirmed that the nerve was in continuity, but compressed by a large extraneural haematoma (fig 2B). The other four patients were referred at a delayed interval and therefore surgical exploration was not possible.

All five patients were followed up for between six and 22 months after angiography. Most patients reported some improvement in their symptoms, although all were troubled to some degree by sensory disturbance or weakness. Patient 5 developed a reflex sympathetic dystrophy only partly responsive to sympathetic nerve blockade. All patients complained of disability when using their right hand, including problems holding cutlery or using a pen. At initial follow up, repeat clinical examination confirmed continuing impairment of median nerve function in all cases. Patient 1, the only patient to undergo nerve exploration, had a satisfactory outcome although his sensory deficit persisted. The COTNAB test established the presence of functional hand disability. Performance was worst on the tests of manual dexterity and all except one patient performed at the impaired level. Two patients scored grade 1 (borderline level) on tests of stereognosis. Surprisingly, stereognosis and dexterity tests of left hand function (unaffected side) were grade 1 (borderline level) in some instances. Patient 3 had an asymptomatic neu-

ropathy which may have explained this, whereas patient 5 had had a previous stroke. Repeat electrophysiological assessment showed evidence of improvement, although all five patients had persistent abnormalities of nerve conduction .

A further telephone follow up was made one year later. Patient 3 was lost to follow up. The recovery interval now ranged from 18 to 34 months. Patients 1, 2, and 5 thought that their hands had subjectively improved; however, patient 4, who had the sympathetic dystrophy, was worse. All patients reported continuing hand pain and disability. All except one patient reported persisting numbness and tingling.

Discussion

This study has identified five patients who sustained a high median neuropathy after right brachial artery left heart catheterisation. Winer *et al* reported a series of 31 patients with iatrogenic nerve injury, of whom six had median nerve damage. In four patients the injury occurred after cardiac catheterisation, but no follow up data were reported. In one patient the injury was due to injection of local anaesthetic into the median nerve and in another due to a forearm haematoma.[6] A single case of median nerve damage was reported by Campion *et al*, but no details were provided.[12] De Bono reports one case of median neuropathy which occurred when the median nerve was mistaken for the brachial artery (DP De Bono, personal communication).

Despite these case reports the true incidence of this complication is not known. Interestingly, the National Angiographic Register has no record of a single case of high median neuropathy among 538 recorded complications of 57 000 diagnostic catheterisations. This is by contrast with our series, in which the incidence of median nerve damage seemed to be between 0.2% and 1.4% of all brachial artery procedures. The incidence of femoral neuropathy complicating cardiac catheterisation has been estimated at 0.21%, and it might be speculated that median nerve damage might occur more often due to the close anatomical relation of the artery and nerve. Some have also suggested that brachial artery cardiac catheterisation is associated with a 3.6-fold higher risk of fatal complications, compared with those who had a femoral artery catheterisation. However, no details about the comparative incidence of nerve damage were provided.[1]

Various mechanisms seem to contribute to median nerve damage. Firstly, as illustrated by patient 1, prolonged haemorrhage at the site of cannulation may cause an expanding haematoma. Pattern speculated that neuropathy induced by haemorrhage was due to a combination of direct nerve compression and focal nerve ischaemia caused by compression of the vasa nervorum.[13] He noted the similarity between the pain found in such cases and in those with ischaemic diabetic mononeuropathy. This mechanism is evident in the patients with median nerve damage, in whom the damage is caused to some degree by local haematoma formation, which results directly from uncontrolled haemorrhage into the anatomically confined antecubital fossa. The importance of haematoma formation has previously been highlighted in cases of median nerve damage secondary to axillary arteriotomy. Surgical exploration of six of these patients disclosed persistent bleeding with haematoma formation in five and pseudoaneursym formation in two. Pseudoaneurysm formation and fibrous scarring may also compress the nerve directly.[14] A further study reported five patients with brachial plexus or median nerve damage after axillary arteriotomies, four of which were associated with haematoma formation.[15] It is important to note that none of our patients received long term oral anticoagulants before angiography. This has been identified as a risk factor for both haematoma formation and femoral neuropathy.[5] Furthermore, this finding provides additional evidence to suggest that the median nerve is at particular risk of damage, even when haemostatic mechanisms are normal.

Secondly, direct nerve damage, as illustrated by patient 2, can be caused by inappropriately placed needle tips, manipulation by arterial forceps, or by the misplacement of arterial ligatures. The experience of the operator may be crucial in this context. Thirdly, nerve damage may be due to nerve ischaemia caused by varying degrees of arterial thrombosis, as illustrated by patients 4 and 5. The methods used to secure haemostasis may be relevant, although we did not identify this as an important factor.

The outcome in our five patients was poor, by contrast with the relatively satisfactory outcome reported by Kent *et al*, in which only six out of 16 patients with femoral neuropathy had persistent symptoms.[5] The reason for this difference is not clear; however, impairment in dominant hand function may lead to more troublesome symptoms than those experienced due to damage to the femoral nerve. Although in all patients some subjective improvement occurred, each reported continuing motor and sensory deficits at follow up. The continuing problems were exemplified by patient 5, who had an ongoing pain syndrome. All patients had significant functional impairment at follow up as documented by the COTNAB test.

It is evident from this study that cardiac catheterisation via the right brachial artery is associated with the risk of appreciable local nerve damage. Although clearly uncommon, this is probably an underdiagnosed complication. Those performing this procedure should be aware of this complication so that (1) patients undergoing brachial artery cardiac catheterisation can be warned of the small risk of median nerve damage; (2) a thorough examination of hand function before and after the procedure is encouraged; (3) a prompt referral for consideration of surgical exploration of patients complaining of median nerve symptoms and signs is ensured, particularly if associated with an appreciable haematoma. High median neuropathy of this type, unlike the more commonly reported femoral neuropathy after cardiac catheterisation, may

cause long term disability of dominant hand function, which may be of medicolegal relevance.

We are grateful to Ms Doreen Rowlands for her instruction in the use of the COTNAB test; our cardiological colleagues for allowing us to report their patients; Dr Michael Johnson and Professor David De Bono for providing the angiographic register data; Dr Juliet Britton, Dr Geoffrey Hart, and Dr Andrew Clifton for their radiological advice; and Dr Charles Pumphrey for his comments about the manuscript.

1 Davis K, Kennedy JW, Kemp H, Judkin M, Gosselin A, Killip T. Complications of coronary arteriography from the collaborative study of coronary artery surgery (CASS). *Circulation* 1979;**59**:1105–11.
2 Stewart J, Ward D, Pumphrey C, Redwood D, Parker D. Major complications of coronary arteriography: the place of cardiac surgery. *Br Heart J* 1990;**63**:74–7.
3 Keilson GR, Schwartz W, Recht L. The preponderance of posterior circulatory events is independent of the route of cardiac catherization. *Stroke* 1992;**23**:1358.
4 Bourassa M, Noble J. Complication rate of coronary arteriography. A review of 5250 cases studied by a percutaneous femoral technique. *Circulation* 1976;**53**:106–14.
5 Kent KC, Moscucci M, Gallagher S, DiMattia S, Skillman J. Neuropathy after cardiac catherization: incidence, clinical patterns, long term outcome. *J Vasc Surg* 1994;**19**: 1008–14.
6 Winer JB, Harrison MJG. Iatrogenic nerve injuries. *Postgrad Med J* 1982;**58**:142–5.
7 Staal A, van Voorthuisen AE, van Dijk LM. Neurological complications following artherial catheterisation by the axillary approach. *Br J Radiol* 1966;**39**:115–6.
8 Littler W. Median nerve palsy: a complication of brachial artery cannulation. *Postgrad Med J* 1976;**52**(suppl 7): 110–1.
9 Macon W, Futrell J. Median nerve neuropathy after percutaneous puncture of the brachial artery in a patient receiving anticoagulation. *N Engl J Med* 1973;**288**:1396.
10 *Aids to examination of the peripheral nervous system*. London: Baillere Tindall, 1986.
11 Tyerman R, Tyerman A, Howard P, Hadfield C. *The Chesington occupational therapy neurological asssessment battery*. Nottingham: Nottingham Rehabilitation, 1986.
12 Campion B, Frye R, Pluth J, Fairburn J, Davis G. Arterial complications of retrograde arterial catherization. *Mayo Clin Proc* 1971;**46**:589–92.
13 Patten BM. Neuropathy induced by haemorrhage. *Arch Neurol* 1969;**21**:381–6.
14 Wescott J, Taylor P. Transaxillary, selective four vessel arteriography. *Radiology* 1972;**104**:277–81.
15 Molnar W, Paul D. Complications of axillary arteriotomies: an analysis of 1762 consecutive studies. *Radiology* 972;**104**: 269–76.