Nerve Compression Injuries Due to Traumatic False Aneurysm

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Experience with 17 patients with delayed onset of compression neuropraxia due to hemorrhage following nonoperative treatment of penetrating arterial injuries is presented. Fifteen cases involved the arteries of the neck shoulder girdle and upper extremity and two the gluteal vessels. This resulted in dysfunction of components of the brachial plexus, median ulnar, and sciatic nerves. Follow-up extended from 3 to 18 months. Of 10 brachial plexus lesions two recovered fully, five partially, and three not at all. Of seven peripheral nerve injuries, full recovery occurred in two patients and none in five. Adverse prognostic factors for neurological recovery are sepsis, involvement of intrinsic hand innervation and the sciatic nerve. An improved prognosis may be expected for upper trunk lesions of the brachial plexus and radial nerve lesions. The complication is essentially avoidable and a careful appraisal of the circulatory status must be made in all patients with penetrating trauma in the neck and shoulder girdle and buttock.

THERE HAS BEEN INCREASING AWARENESS in recent years of the phenomenon of neurological dysfunction complicating compression by a false aneurysm.[1-7] This invariably follows initial misdiagnosis of penetrating arterial trauma, usually in the shoulder girdle area, and the patient presents with a late onset false aneurysm and a peripheral neurological deficit some time after the original injury.

The morbidity created is considerable and prognosis for recovery guarded. The purpose of this paper is to report a relatively large experience with the problem with particular reference to prognostication of neurological recovery.

Patients

At King Edward VIII Hospital, Durban, South Africa, 17 patients have been treated on the Vascular and Orthopaedic Services for compression nerve injuries complicating false aneurysm. Their ages ranged from 18 to 59 years (average 27.7) and all were men.

The sites of arterial injury are summarized in Table 1. Fifteen injuries involved arteries in the root of the neck and upper extremity. The most frequently injured artery was the axillary (nine patients). Two patients had a neurological deficit in the lower extremity complicating laceration of a gluteal vessel.

In every instance the original injury followed penetrating trauma; knife stabs in 14 and handgun missile wounds in three patients. There was a consistent pattern of clinical presentation in all patients. All were seen soon after the initial trauma, and the diagnosis of major arterial disruption was not made at that time. Clinical neurological assessment revealed no deficits initially or within the following 24-hour period, and all patients were quite definite that they had full use of the involved limb at that time. Several had returned to work.

Following an interval that ranged from 1 to 6 weeks (average 3.3), the patients returned to hospital with some degree of neurological dysfunction. In eight patients there was an acute, dramatic onset of false aneurysm formation and neurological deficits involving the shoulder girdle and arm, occurring within 10 days of injury. The remaining nine described a more gradual and progressive onset of neurological dysfunction. The pattern of nerve injury involving the upper limb is shown in Table 2.

Six patients presented with total loss of function of the upper limb, which was flail and anesthetic (total brachial plexus lesion).

In one patient neurological disability was confined to the distribution of the upper trunk of the brachial plexus with inability to abduct the shoulder and flex the elbow. Wrist movements and intrinsic hand function remained intact (upper trunk type lesion). Two patients had, in addition to upper trunk type lesions, inability to dorsiflex the wrist and extend the fingers due to radial nerve compression. In one patient shoulder, elbow, and wrist movement remained intact but all intrinsic hand function was lost (lower trunk type lesion). Five patients with injuries of the distal axillary or proximal brachial arteries had peripheral nerve dysfunction involving the radial in two and the median in three. Total sciatic nerve lesions occurring within 3 weeks of a stab in the buttock area were seen in two patients.

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TABLE 1. *Arteries Injured*

	Number
Common carotid (proximal)	1
Subclavian (distal)	1
Axillary	9
Brachial	4
Gluteal	2
Total	17

Management and Results

All patients were submitted to selective arteriography followed by urgent surgical exploration. Arterial ligation was necessary in the two patients with gluteal artery lesions and in three in whom a false aneurysm arising from the axillary artery had become overtly septic. Arterial reconstruction followed standard principles in the remaining 12 patients (Table 3) and all regained a normal peripheral pulse. Exploration of the involved nerves confirmed that they were in anatomical continuity and had been compressed by the false aneurysm.

All patients have been carefully followed-up over a period ranging from 3 to 18 months, with regular clinical and electromyographic reassessments and every patient is presently involved in a full physical and occupational therapy rehabilitation program.

The neurological results in ten patients with brachial plexus lesions are summarized in Table 4. Two recovered fully, five had partial return of neurological function, and three show no signs of recovery.

Full recovery occurred within 6 months in two patients who had a lesion in which the predominant neurological dysfunction was in the distribution of the upper trunk of the brachial plexus; one had additional radial nerve involvement.

Of the six patients who initially presented with total involvement of the brachial plexus, five have regained full shoulder girdle and elbow function (upper trunk distribution), but have shown no signs of return of intrinsic hand function (lower trunk distribution) after 6 months or more.

TABLE 2. *Pattern of Nerve Injury*

	Number
Brachial plexus distribution	
Total	6
Upper trunk	1
Upper trunk and radial	2
Lower trunk	1
Peripheral nerves	
Radial	2
Median	3
Sciatic	2
Total	17

TABLE 3. *Treatment of Arterial Injury*

	Number
Ligation	5
Patch angioplasty	2
Vein graft	10
Total	17

In the three patients who show no sign of neurological recovery, all initially presented with an infected false aneurysm and ran a protracted postoperative course complicated by gross local sepsis. One patient had originally presented with a total brachial plexus lesion and showed no signs of recovery after 18 months. Another patient's initial lesion was confined to the distribution of the upper trunk and radial nerve but within the following month the neurological lesion had progressed to total brachial plexus involvement. Uncontrollable sepsis eventually necessitated amputation. The third initially had a lesion confined to the lower trunk and showed no improvement 3 months later.

Of the seven patients with peripheral nerve compression lesions, full recovery was noted within 3 months in two with radial involvement (Table 5). No recovery has been recorded more than 3 months after injury in three patients with median nerve involvement. Sciatic nerve function has failed to recover more than 6 months after injury in two patients.

Discussion

The morbidity created by nerve compression caused by a false aneurysm is considerable. Only four of the 17 patients in this series have recovered full neurological function over a period ranging from 3 to 18 months. No visible nerve lesion was noted in any patient at surgical exploration and the pathology is best described as a lesion in continuity.[8] However, in the traction-type brachial plexus lesion, which may be similar, functional recovery may be expected for at least a 12-month period after injury.[9]

TABLE 4. *Neurological Result—Brachial Plexus Compression Injury (3—18 Months)*

Presenting Deficit (N = 10)		Residual Deficit		Follow-up (Months)
Total	6	Lower trunk	5	<6
		No recovery (sepsis)	1	18
Upper trunk	1	Full recovery	1	6
Upper trunk and radial	2	Full recovery	1	3
		Total flail (sepsis)	1	1 (amputation)
Lower trunk	1	No recovery (sepsis)	1	3

TABLE 5. *Neurological Result—Peripheral Nerve Compression Injury (3—18 months)*

Presenting Deficit (N = 7)		Residual Deficit		Follow-up (Months)
Radial	2	Full recovery	2	3
Median	3	No recovery	3	>3
Sciatic	2	No recovery	2	>6

Local sepsis augers badly for neurological recovery as shown in three patients with brachial plexus lesions who show no signs of improvement. In one the neurological lesion progressed from partial to total involvement of the plexus. Other lesions that appear to have a poor prognosis are those that involve intrinsic hand innervation, namely the lower trunk of the brachial plexus, the median, and ulnar nerves. Similarly, more than 6 months after injury, neither patient with sciatic nerve injury shows any sign of recovery.

Based on the observations made in this group of patients it would appear that an improved prognosis may be expected for compressive injuries involving the upper trunk of the brachial plexus and the radial nerve. Other factors that may have some influence on the degree and extent of neurological injury are the rate at which compression developed and the duration of the compression. There are too few patients in this series for any meaningful analysis to be made. However, it is our general impression that prolonged compression contributes to irreversible nerve damage.

With regard to operative management an additional consideration is the possible role of epineural fibrosis in perpetrating the compressive element. We have as far as possible practiced neurolysis at the time of vascular reconstruction, but feel that this should be meticulously done under magnification in order to reduce the danger of iatrogenic damage and to ensure adequate dissection of the nerve.

The complication of late onset false aneurysm is essentially an avoidable one. Careful reappraisal of the case notes at the time of initial presentation to hospital indicates that every patient in the present series had some evidence of a major arterial injury. Overt signs of ischemia rarely occur with traumatic arterial disruption in the neck, shoulder-girdle, or buttock area due to the rich collateral network. Signs of arterial injury are therefore more subtle and may be missed if the diagnosis is not carefully considered.[1-7] In most busy trauma services, routine selective arteriography for all penetrating wounds in these anatomical areas is impracticable. However, this investigation should be performed in the presence of a distal pulse deficit, local hematoma, bruit or anatomically related nerve injury. Additional important considerations are active hemorrhage, a history of recent excessive hemorrhage, unexplained hypovolemia, and low hemoglobin and hematocrit levels.

The complications described serve to emphasize the importance of routine repair of major arterial disruption due to penetrating trauma, even if there is no active hemorrhage or peripheral circulatory compromise at the time of presentation.

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