Ischaemia and the pink, pulseless hand complicating supracondylar fractures of the humerus in childhood

LONG-TERM FOLLOW-UP

A series of 26 children was referred to our specialist unit with a ‘pink pulseless hand’ following a supracondylar fracture of the distal humerus after a mean period of three months (4 days to 12 months) except for one referred after almost three years. They were followed up for a mean of 15.5 years (4 to 26). The neurovascular injuries and resulting impairment in function and salvage procedures were recorded. The mean age at presentation was 8.6 years (2 to 12). There were eight girls and 18 boys.

Only four of the 26 patients had undergone immediate surgical exploration before referral and three of these four had a satisfactory outcome. In one child the brachial artery had been explored unsuccessfully at 48 hours. As a result 23 of the 26 children presented with established ischaemic contracture of the forearm and hand. Two responded to conservative stretching. In the remaining 21 the antecubital fossa was explored. The aim of surgery was to try to improve the function of the hand and forearm, to assess nerve, vessel and muscle damage, to relieve entrapment and to minimise future disturbance of growth.

Based on our results we recommend urgent exploration of the vessels and nerves in a child with a ‘pink pulseless hand’, not relieved by reduction of a supracondylar fracture of the distal humerus and presenting with persistent and increasing pain suggestive of a deepening nerve lesion and critical ischaemia.

“The early recognition at the outset of ischaemic injury is a matter of utmost importance for the reversibility of damage depends wholly on the duration of the ischaemia”.

Supracondylar fractures of the distal humerus account for 3% to 18% of all fractures in children. Ischaemia of muscle and nerve is the most serious complication of the injury. Compromise of the brachial artery has been reported in approximately 11% of cases of supracondylar fracture.

There is no dispute regarding the need for emergency treatment of the vascular problems in the patient with a supracondylar fracture and a cool, white hand. However, several authors have suggested that a conservative approach or delayed surgery is appropriate in a child with a supracondylar fracture and a pink, but pulseless hand. We describe the long-term follow-up of a series of children who presented with a supracondylar fracture and a pink, but pulseless hand who were subsequently referred with ischaemic contracture of the forearm muscles.

Patients and Methods

Between 1983 and 2003, 26 children who initially presented with a 'pink pulseless hand' and a supracondylar fracture of the humerus were referred to our specialist unit. Their mean age at the time of injury was 8.6 years (2 to 12). The mean length of follow-up from the time of injury was 15.5 years (4 to 26). All data were collected and coded by diagnosis prospectively. The patients were followed up prospectively by periodic examination at least annually; none was discharged from follow-up. The brachial artery and relevant nerves were explored at the elbow. Some children subsequently required intrinsic muscle release, tendon transfer and in one child free muscle transfer. All underwent lengthy periods of serial splinting and stretching, before and after the salvage operation.

Assessment of nerve outcome was recorded as good, fair or poor. Muscle power was graded according to the Medical Research Council (MRC) scale. Areas of complete and partial sensory loss were mapped and photographed.
Table I. Details of the nerve injuries

<table>
<thead>
<tr>
<th>Nerve affected (56 nerves)</th>
<th>Cause</th>
<th>Death</th>
<th>Motor</th>
<th>Sensory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median</td>
<td>Trapped in fracture</td>
<td>9 Conduction block</td>
<td>6 Good</td>
<td>7 Good</td>
</tr>
<tr>
<td></td>
<td>Ischaemia and compression</td>
<td>14 Axonotmesis</td>
<td>17 Fair</td>
<td>10 Fair</td>
</tr>
<tr>
<td></td>
<td>Transection</td>
<td>1 Neurotmesis</td>
<td>1 Poor</td>
<td>7 Poor</td>
</tr>
<tr>
<td>Ulnar</td>
<td>Trapped in fracture</td>
<td>3 Conduction block</td>
<td>8 Good</td>
<td>7 Good</td>
</tr>
<tr>
<td></td>
<td>Ischaemia and compression</td>
<td>15 Axonotmesis</td>
<td>10 Fair</td>
<td>10 Fair</td>
</tr>
<tr>
<td></td>
<td>Transection</td>
<td>1 Neurotmesis</td>
<td>1 Poor</td>
<td>2 Poor</td>
</tr>
<tr>
<td>Radial</td>
<td>Trapped in fracture</td>
<td>1 Conduction block</td>
<td>10 Good</td>
<td>10 Good</td>
</tr>
<tr>
<td></td>
<td>Ischaemia and compression</td>
<td>12 Axonotmesis</td>
<td>3 Fair</td>
<td>2 Fair</td>
</tr>
<tr>
<td></td>
<td>Transection</td>
<td>0 Neurotmesis</td>
<td>0 Poor</td>
<td>1 Poor</td>
</tr>
</tbody>
</table>

Results

All 26 patients had a ‘pink pulseless’ hand which had been noted at the referring institution and all but three were pink and pulseless on referral. Only four of the 26 patients had undergone immediate surgical exploration of the brachial artery before referral. Of the 26 fractures, 21 had been treated by operative fixation and five by manipulation and casting alone. Three of the four patients in whom the artery had been explored early did well with no evidence of post-ischaemic contracture. Any neurological loss recovered spontaneously without the need for further operation. In a fifth child the artery had been explored and repaired after an interval of 48 hours. The children were referred to our unit after a mean period of three months (4 days to 12 months), except for one who was referred at almost three years.

Established ischaemic contracture of the muscles of the forearm and hand was present in 23 of the 26 children at the time of referral. In two, the median and ulnar nerves had recovered, and residual contractures of the flexor muscles responded to a stretching programme. The antecubital fossa was explored in the remaining 21 children. The aim of surgery was to improve hand and forearm function if possible, to assess the prognosis of the nerves, to perform neurolysis or repair as necessary, to extricate nerves and vessels from the site of the fracture, to restore blood flow to the hand and to minimise future disturbance of growth.

Arterial injury. The radial pulse had been recorded as absent in all 26 children when they presented to the referring hospitals. It was successfully restored in two children by urgent decompression and in one by emergency repair of the artery. In a fourth child in whom the artery was repaired by reversed vein grafting at 48 hours after injury flow was not restored.

A weak radial pulse was noted in nine children when they were discharged from the referring hospital. We found that the radial pulse was absent or scarcely perceptible in 15 of the children when we examined them. The pulse was normal only in the three cases successfully explored.

The brachial artery and the median and ulnar nerves were explored in 21 children. The artery in the child treated by tendon elongation was not explored. In 12 the vessel was found to be constricted by dense scar tissue deep to the bicipital aponeurosis and in nine it was trapped within the fracture. Pulsatile flow returned after decompression of the vessel in all patients. Four of the arteries trapped in the fracture were found to be narrowed to a cord, but pulsation returned about 15 minutes after release of the vessels, which were bathed in a solution of papaverine.

Muscle injury. Post-ischaemic fibrosis of the muscles of the forearm and hand was evident in all children except in the three treated successfully by immediate exploration of the artery.

The flexor compartment was worst affected and palliative operations were necessary in 22 children. These included flexor muscle slide in 17, step elongation of the tendons in four, and a free functioning transfer of gastrocnemius in one. The extensor compartment was least affected and serial stretching and splinting were successful in five patients.

The intrinsic muscles were affected in nine hands, and in four of these the contractures did not respond to splinting or stretching. The contracted adductor pollicis was detached from its origin in three patients and the interosseous muscles were released in one.

Nerve lesions (Table I). The diagnosis of nerve injury was made clinically and surgery was not delayed for nerve conduction studies. There was evidence of 56 injuries to a main nerve. Nerve conduction studies were considered to be unjustified and in this situation unnecessary. Intraoperative nerve stimulation was performed routinely. No child had a lesion confined to the anterior interosseous nerve. In 41 nerves ischaemia of the nerve and compression by swollen or infarcted muscle were thought to be the cause of the lesion. A total of 13 nerves were trapped in the fracture and two had been divided by a Kirschner (K)-wire during internal fixation.

The ulnar nerve was regularly found to be compressed and narrowed by infarcted muscle. The depth of the lesion varied between different groups of nerve fibres and some preservation, or recovery, of modalities of cutaneous sensibility and of vaso- and sudomotor function was noted at
referral in 14 of the median and ulnar nerves. Conduction block was the predominant lesion in ten of the 13 radial nerves. Degenerative lesions (axonotmesis) affected 30 of the main nerves, and repair was necessary in the two cases of neurotmesis.

Recovery of cutaneous sensibility and small muscle function in the hand was generally good after decompression. Recovery into the flexor and extensor muscles of the forearm was determined by the extent of post-ischaemic fibrosis. The nerves recovered much better than the muscles of the forearm after decompression.

Pain. No formal documentation regarding pain was available from the referring institutions. The children or their parents recollected intense pain in the first 12 hours after injury, often resistant to standard analgesia. Pain was felt in the whole forearm and hand and was not confined to the elbow.

Growth and deformity. Quantitative data regarding growth of the affected limb were not formally recorded in all patients. The extent of growth disturbance was evident only at long-term review (case 2). Children with a seemingly good early result after corrective surgery had recurrence of deformity with considerable defects at skeletal maturity.

Illustrative cases
Case 1. (Fig. 1). An eight-year-old boy was referred four weeks after closed treatment of a supracondylar fracture of the left distal humerus. The radial pulse was absent and there was complete radial, median and ulnar palsy. The antecubital fossa was explored and the brachial artery and the median nerve were found to be trapped within the fracture. The artery was a pulseless cord. Flow through the artery returned as the dorsum of the foot was prepared for vein harvesting. A flexor muscle slide was performed eight weeks later.

Case 2. (Fig. 2). A seven-year-old boy was referred at ten weeks with complete lesions of the median, radial and ulnar nerves. The radial pulse was absent. The artery and median nerve were found to be trapped in the fracture which had been fixed by K-wires. Pulsatile flow was restored by release of the artery from the site of the fracture. Ischaemic
changes in the muscles were irreversible and were treated by a flexor muscle slide, adductor pollicis slide and release of the interosseous muscles.

Discussion
Ischaemia of the forearm musculature and nerves in patients with a supracondylar fracture of the humerus may be caused by axial arterial occlusion, compartment syndrome, or both. The result of sustained ischaemia is fibrosis of muscle and nerve. External or internal compression of a rigid compartment causes the collapse of small vessels and increased venous pressure leading to a compartment syndrome. Anoxic damage to the capillary walls increases vascular permeability which in turn increases interstitial swelling. Increased pressure within the closed fascial spaces provokes further ischaemia by blocking the arteriovenous microcirculation. As a result progressive necrosis of muscle tissue and compression and ischaemia of the nerves occur.16,17

It is important to distinguish the different pathological entities of arterial occlusion and compartment syndrome in each patient. Forearm fasciotomies without restoration of arterial flow will not prevent ischaemic injury to the muscle and nerve, or growth disturbance.

Several recent publications have suggested that a pulseless limb which appears to be well perfused can be managed without operation.9-12 Gosens and Bongers9 treated conservatively four patients with supracondylar fractures of the humerus without pulses, but with good capillary refill and a warm hand. No functional deficit was noted at short-term review and all pulses returned spontaneously before discharge. The length of the ischaemic time was not reported or the relation of return of pulses to manipulation of the fracture. In the series reported by Garbuz et al10 five patients with a pink, pulseless hand were managed conservatively. In four a pulse was audible on Doppler examination immediately after reduction and all had a pulse before discharge. Louahem et al11 reviewed a series of 210 patients with severely displaced supracondylar fractures. They described 26 with a pink, pulseless hand. In 21 after closed reduction of the fracture the pulses returned immediately and in two this occurred later at four and six days. Good distal perfusion and adequate oxygen saturation were recorded over this period.

The radial recurrent artery arises distal to the elbow, anastomosing with the radial collateral branch of the profunda brachii. The last part of the profunda brachii accompanies the radial nerve. The other main descending collateral pathway is the superior ulnar collateral artery which runs with the ulnar nerve.18 If either the radial or ulnar nerves are caught or severed by the fracture or the surgeon, the collateral vessels are also disrupted. Poiseuille’s Law19 states that flow through a vessel is affected by three variables, namely, the radius of the cylindrical vessel, the total tension in the wall and the pressure gradient and is described by the following equation:

\[
\theta = \frac{\pi R^4}{8} \frac{\lbrack\Delta P\rbrack}{L}
\]

where flow \( \theta \) is the flow of an incompressible uniform viscous liquid, \( R \) is the internal radius of the tube, \( P \) the pressure difference between the two ends, \( m \) the dynamic fluid viscosity and \( L \) the total length of the tube.19

Wajcberg et al20 stated that in Hispanic children the radius of the brachial artery in a six-year-old was 1.25 mm to 1.5 mm and the rate of flow through the artery at rest was 200 ml per minute. The collateral vessels have a radius of approximately 0.5 mm and therefore blood flow would be reduced to 20 ml per minute, a reduction of 90%. As the tissue pressure increases secondary to increased vascular permeability in an environment of relative ischaemia, transmural pressure will decrease and flow will be reduced further.

Peripheral nerves are extremely sensitive to ischaemia and changes in sensibility should not be ignored. The large \( A_p \) fibres, responsible for touch and vibration sense, are the most sensitive.21 Lewis, Pickering and Rothschild22 demonstrated that early muscle weakness was the result of ischaemia of the nerves. Although injury to a main artery is unlikely to cause complete necrosis of a nerve trunk, increasing loss of sensibility indicates deepening of the nerve lesion.23 When flow through the brachial artery is impeded by tamponade the nerves become compressed by swelling and haematoma around the elbow and are further compromised by swelling and infarction of the muscles of the forearm (Fig. 1b). Deepening of the lesion of the nerve while under observation distinguishes between the conduction block of concussion and the much more serious anoxic lesion. Such deepening of the lesion is a sign of critical ischaemia.

The Society for Vascular Surgery and the International Society for Cardiovascular Surgery has developed a grading system for acute limb ischaemia in the lower limb (Table II).24 These guidelines are also applicable to ischaemia of the upper limb and that resulting from trauma. Our cases would be considered as class IIb in this system.

Pain is a cardinal sign of nerve ischaemia.25 All children in our series experienced severe pain in the first 12 hours, and in many the pain was resistant to opiates. Unfortunately, no formal documentation regarding pain and in particular any improvement after closed or surgical treatment of the fracture was available from the referring institutions. Eadie26 described two types of ischaemia which may follow injury. The first is caused by interruption of the main arterial supply with or without venous damage. The extensor muscles and the small muscles of the hand are affected and this can be distinguished from a true compartment syndrome which chiefly affects the muscles of the flexor compartment. When there is interruption to the arterial supply, release of the fascial compartments will not prevent ischaemic injury to the muscle. Wadsworth27 described the different types of ischaemia of the forearm vasculature as compression obstruction from an external force, haematoma or bone fragment, contusion causing
subadventitial haemorrhage or thrombus, laceration, transection or perforation, false aneurysm, traumatic arteriovenous fistula and spasm. The brachial artery in the child seems to be almost unique in being prone to true spasm but it is unwise to assume this diagnosis. In 1973 Parkes28 stated that “in the absence of peripheral arterial pulses, take immediate steps to relieve pressure on a main artery by reducing any displaced fracture or dislocation and, if still necessary, explore the artery”. Wadsworth27 suggested that “Arterial spasm was usually self-limiting and disappeared within a few minutes after relief of the external local stimulus. In the treatment of trauma persisting ischaemia should be considered arterial injury and not caused by spasm”.

In a patient with a supracondylar fracture of the humerus and absent distal pulses when there is pain and evidence of a deepening nerve lesion urgent exploration can prevent long-term disability. In the pulseless limb when reduction of the fracture results in relief from compression and return of the pulse, exploration of the artery is not required. Our series demonstrates the consequences of prolonged ischaemia, but it is not possible to describe a prevalence of the condition in the population from this study. The seriousness of Volkmann’s contracture is considered more for its irreversibility and devastating consequences rather than for its prevalence, and almost every case is preventable. Only rarely is repair of the vessel needed. Decompression of the artery is usually adequate and allows simultaneous decompression of the accompanying veins. Exploration of a swollen antebrachial fossa in a child is not an easy operation, but this should not be a reason for avoiding such surgery when it is indicated. Careful exploration with direct visualisation should avoid damage to accompanying structures. A solution of 1% papaverine should be available to apply to the vessel locally which may accompany veins. Exploration of a swollen antecubital fossa in this situation. 

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Table II. The Society for Vascular Surgery/International Society for Cardiovascular surgery grading system for limb ischaemia

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Viable limb, without impairment of sensory or motor function and audible Doppler signals</td>
<td>Heparin is given and elective treatment, either conservative or interventional, is arranged</td>
</tr>
<tr>
<td>IIa</td>
<td>Marginally threatened with symptoms limited to mild sensory loss</td>
<td>A delay of nine hours is acceptable before attempting treatment</td>
</tr>
<tr>
<td>IIb</td>
<td>Immediately at risk with pronounced sensory loss, mild to moderate motor loss but audible Doppler signals</td>
<td>Delay is unacceptable and urgent clot extraction is required</td>
</tr>
<tr>
<td>III</td>
<td>Absent Doppler flow, paralysis, total sensory loss and irreversible tissue damage</td>
<td>Attempts to restore blood flow would lead to complications and therefore delayed amputation should be performed after resuscitation</td>
</tr>
</tbody>
</table>

References