Neurovascular complications in displaced extension-type supracondylar fractures in children. Outcome of conservative management

By

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Ethics

This study has University of Cape Town Research Ethics Committee approval REC REF 345/2008.

Declaration

I, Frederik Marthinus Louw, hereby declare that the work on which this dissertation is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university.

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Date: 16/08/2010

Acknowledgement

To Prof Teddie Hoffman, my supervisor. This thesis, amongst other things, once more humbles me in light of the immense energy, dedication, time and knowledge you so freely part with. I still, as always, have so much to learn.

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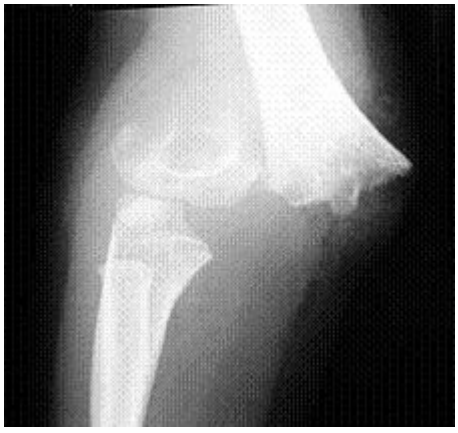
Introduction

Supracondylar humerus fracture is the most common fracture of the elbow in children.[1,2,3,4,5,6] 95% are extension-type fractures, mostly from a fall on an outstretched arm.[7,8,9] It is also the most common upper limb injury requiring surgery.[10] Closed reduction and internal fixation with kirschner-wires is the gold standard of treatment.[3, 4,11,12,13,14,15] The ideal configuration of pins however, remains controversial.[1,3,5,8,11,12,14,16]

The distal humerus has a unique anatomy in the child. The bony supracondylar region is the weakest point, with ligaments and tendons being relatively stronger, thus making it susceptible to fracture[6,8,11,17] The neurovascular structures lie close to bone. The brachialis muscle is all that separates the neurovascular bundle anterior from bone. This renders neurovascular structures prone to injury with fracture.[18]

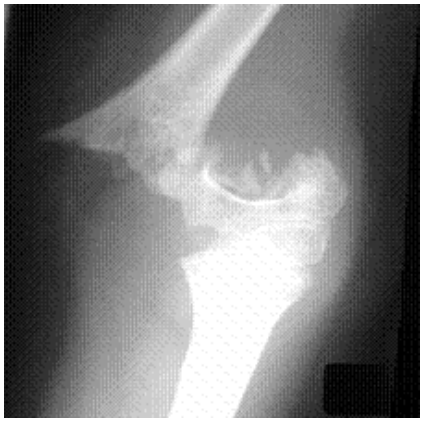
Neurovascular complications are common, with an incidence of 10-20%.[3,4,7,9,11,15,19,20,21,22,23,24,25] Most injuries are found in displaced/ Gartland type III fractures.[26,27,28] Common displaced fracture patterns correlate with specific neurovascular injuries.[7,18,24,29] Posterolateral (PL) fracture displacement correlates with median nerve (MN) and arterial injury. Posteromedial (PM) fracture displacement correlates with radial nerve (RN) injury.

Posterolateral = MN + Vasc injury



Picture 1

Posteromedial = RN injury



Picture 2

Nerve injury is the most common complication in supracondylar fractures.[1] The anterior interosseous nerve (AIN) is the most common injured nerve (34%).[1,7,17,30,31,32,33,34,35] The ulnar nerve has the highest incidence of iatrogenic injury.[1,18,24,36,37] Most authors agree that neurological injuries are neuropraxic and shall resolve spontaneously.[4,7,11,13,15,16,17,19,20,21,22,23,32,33,38,39,40,41] Some authors however feel nerve injury requires surgical exploration.[18,42,43,44] Similar controversy exists over iatrogenic ulnar nerve injury management. Several authors recommend ulnar nerve exploration post pinning.[7,18,42,43,44]

Vascular compromise occurs in up to 20% after supracondylar fracture.[7,14,24,26,31,33,45] All children presenting with an absent or diminished pulse require urgent closed reduction and internal fixation (CRIF) with k-wires. The vascular status is then re-evaluated. If the hand remains pulseless, ischaemic and cold post CRIF urgent vascular exploration with open reduction and internal fixation (ORIF) is required.[7,15,19,22,24,26,40,46,47]

Compartment syndrome similarly requires exploration, ORIF with k-wires and fasciotomy.[7,35,47,48,49,50,51] Great controversy exists over the management of the pink, pulseless hand with good capillary filling post CRIF. Griffin et al[6] refers to this as, “the most difficult aspect of managing children with supracondylar fractures…is the question of how to proceed when an apparently satisfactory attempt at closed reduction has failed to re-establish a palpable pulse…”. Two schools of thought exist. Some authors manage the pink, pulseless hand conservatively with observation, maintaining the pulse returns over time.[3,6,7,15,21,23,24,28,29,32,33,35,40,45,53,54,55] Several other authors feel an absent pulse denotes vascular injury which must be explored.[2,9,11,14,18,26,27,31,38,47]

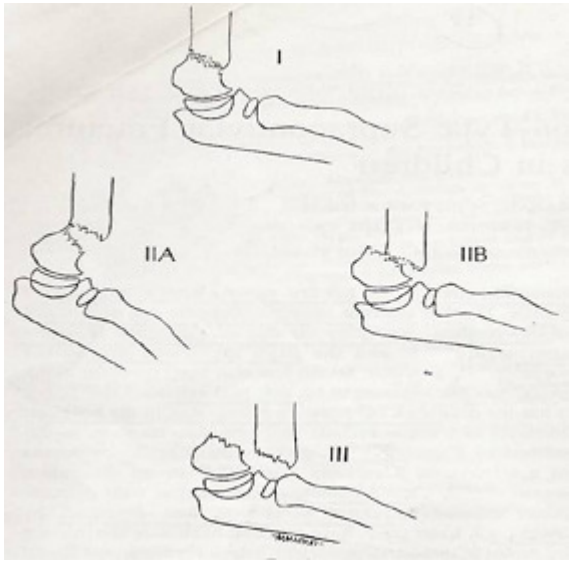
Aims of Study

The aim of our study was to review our conservative management of neurovascular complications in displaced extension-type supracondylar fractures of the humerus in children. We critically analysed the outcomes. Our results shall aim to clarify the management of this contentious issue.

Patients and Methods

After obtaining ethics approval, a retrospective review was done on all displaced supracondylar humerus fractures seen at Red Cross Children's Hospital over a 5 year period 2002-2006. All children's notes and radiographs were reviewed. A total of 508 children were identified. 10 were flexion-type injuries and excluded. 498 extension-type fractures were managed in theatre. The average age was 6 years and 3 months (range 2-13 years). Mechanism of injury: 493 fall on level. 332 male: 166 female (2:1) children were seen. The left arm was most often injured L: R 300: 198 (1.5:1). Average time from injury to surgery: 7 hours (see table 2). Fractures were classified according to Pirone's[11] modification of Gartland[28] and Wilkins[7] classification.

Pirone's Classification – Picture 3



It was chosen as it corresponded with other classifications and for its interobserver reliability[7]. All type I fractures were managed in POP backslab and were not operated on and thus excluded. 168 (34%) were type IIA and managed with manipulation under anaesthesia and Plaster of Paris (POP). None of these patients developed neurovascular complications and were thus excluded. 43 were type IIB and 287 were type III (330 combined- 66%). All type III and 38 type IIB these were managed with CRIF. All complications came from this group. 5 type IIB were treated with MUA + POP. We saw 3 open fractures which were managed with exploration, washout and ORIF. 6 fractures were irreducible, all type III and managed with straight lateral traction (SLR). There were 14 associated ipsilateral fractures of the forearm, all k-wired (see table 2).

TABLE 1

PATIENTS AND METHODS TABLE

Average age	6 yrs 3 months	Range 2- 13 years
Extension fractures	n = 498	
Type IIA	168	34% Excluded
Type IIB	43	Type IIB + III= 330= 66%
Type III	287	
Posteromedial displacement	146	51%
Posterolateral displacement	141	49%
Mechanism of injury	493	Fall on level
Mechanism of injury	5	Other
Flexion fractures	n= 10	Excluded
Type II Flex	3	Excluded
Type III Flex	7	Excluded
Complications flexion type	2 ulnar nerve palsies	Recovered day 21 Excluded
Male	332	
Female	166	
Male: Female	2:1	
Left: Right	1.5:1	
Open fractures	3	All type I Gustilo + A
Irreducible fractures	6 type III	SLR
Associated fractures	14	3%
Time from injury to surgery	n=469	7 hrs (6-18)
	n=23	18-48 hrs
	n=6	48 hrs- 7 days

Surgical Technique

All fractures were first seen at the Red Cross Children's Hospital Trauma Unit by the residing Casualty Officer. The child was examined and neurovascular status of the limb documented. Bruising, ecchymosis, open wounds, pucker sign and compartment syndrome were looked for. Ipsilateral forearm injuries were documented. The arm was splinted in 20° flexion and AP and lateral x-rays taken of the affected limb as well as an AP view of the opposite arm (for later comparison of Baumann's angle). The orthopaedic registrar was then called to assess the injury and neurovascular status once more. All fractures (type IIA/B and III) were booked as emergencies.

All children received general anaesthesia. Type IIA were treated with closed reduction and backslab POP with forearm in 100° flexion and pronation. Reduction was performed as described by Wilkins[7]. Baumann's angle post-reduction was measured in theatre with image intensification. Thirty eight type IIB and all type III fractures were managed with closed reduction and internal fixation with crossed k-wires (5 type IIB had MUA + POP). Parenteral antibiotics were given as prophylaxis (1st generation cephalosporin). Fractures were reduced and then held in flexion and pronation with adhesive tape (Wilkins[7]). Sterile drape was then performed and 2 parallel 1.6mm lateral k-wires were inserted under image intensifier guidance. A medial 1.6mm wire was then inserted. The adhesive tape was then removed and the elbow flexed to less than 90° and the vascular status re-evaluated. This was done by combined assessment of the radial pulse, capillary filling, colour, warmth and pulse oximetry as well as compartment palpation. If the pulse returned the child was put into a backslab POP with less than 90° elbow flexion to allow further compartment and neurovascular evaluation in the ward[33]. If the pulse did not return, but the hand remained pink, warm and with good capillary filling a similar backslab was applied and the child sent to the ward for neurovascular and compartment checks (pink, pulseless hand). All children were kept overnight and seen by professor or senior consultant on the morning round who repeated the neurovascular assessment. In cases of the pink, pulseless hand the patient was kept for a further day of neurovascular and compartment checks to see if the pulse would

return. When iatrogenic ulnar nerve palsy was discovered post-op the medial pin was removed. All children were then discharged.

Follow up visits were at one, three weeks, three months and until full range of motion was regained. K-wires were removed at routine three week clinic follow up. Neurovascular assessments were repeated at follow up and x-rays taken. Bauman's angle and carrying angle were assessed as well as range of motion. Results were assessed according to the Flynn[4] criteria with any varus given a poor outcome.

TABLE 2

Criteria for Grading Results

Result	Cosmetic Factor: Loss of Carrying Angle °	Functional Factor: Loss of Motion °
Excellent	0-5	0-5
Good	6-10	6-10
Fair	11-15	11-15
Poor	>15	>15

All neurovascular complications were followed up until full return of pulse and neurological function. No nerve conduction studies, electromyography (EMG), angiograms or specialised vascular studies were done.

All ipsilateral forearm fractures were manipulated and k-wired. Open reduction and percutaneous k-wire fixation (ORIF) was used exclusively for open fractures. Straight lateral traction was employed for all failed reductions. Bedside skin traction was applied as described by Dunlop[56] and Piggot[35]. No skeletal traction was used. The carrying angle was assessed clinically during traction period. At three weeks when callus appears, and the child can actively flex, traction was discontinued and child discharged without any further immobilization.

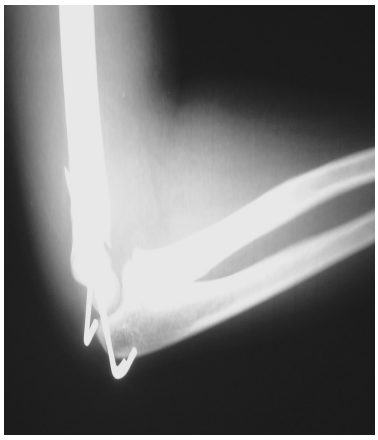
Results

We found 60 neurovascular complications (60/498- 12.1%). 39 neurological complications and 21 vascular. There were 4 combined neurovascular injuries. No compartment syndromes or Volkmans ischaemic contractures were observed. As found in the literature, the majority of our injuries were type III fractures (287/330- 86%).[11,22,26,27,33,58]

Neurological complications

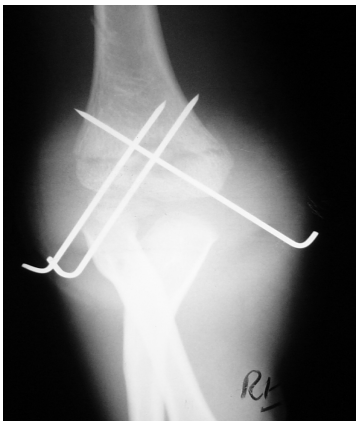
All neurological injuries were managed conservatively with an expectative approach. Seven radial nerve injuries were recorded (2%). All were preoperative. Six were in isolation. One radial nerve injury was associated with an iatrogenic ulnar nerve injury. All were type III fractures with 7/7 posteromedially (PM) displaced. Radial nerve average time to recovery: 68.3 days (range 21-106 days).Seven median nerve palsies (2%) were noticed on admission, only two of which were AIN palsies (much lower than reported in literature). All median nerve injuries were type III fractures and posterolaterally (PL) displaced, corresponding with the literature[7,18,24,29]. Two median nerve injuries were associated with vascular compromise. Median nerve average time to recovery was 53.6 days (range 21-180). One MN injury was only noticed at 21 days. ? iatrogenic injury to MN as UN was also pinned in this combined injury. Twenty-five (7.6%) ulnar nerves were iatrogenically injured with pinning. Two had associated vascular compromise, a further two had associated median nerve injuries and one an associated radial nerve injury. Three were type IIB and 22 type III fractures, all managed with CRIF. 22/25 injured ulnar nerves were diagnosed on day one post surgery. A further three were diagnosed within a week post surgery, suggesting a delayed onset. Ulnar nerve average time to recovery: 76.3 days (range 18-193 days). This corresponds with literature findings that the ulnar nerve takes the longest of all injured nerves to recover.[33] Review of x-rays revealed three common medial pin placement errors with iatrogenic ulnar nerve palsy. Medial pins in ulnar nerve injuries were placed either: (a) too posterior in the ulnar groove, (b) too lateral on medial epicondyle or (c) too vertical >40°

Wire too posterior



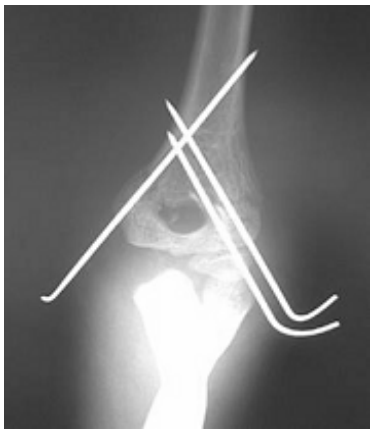
Picture 4

Wire too lateral



Picture 5

Wire too vertical >40°



Picture 6

All children but one (38/39) had full neurological return of function by 6 months. One nerve had normal function by 193 days. No residual nerve defects were present after 193 days. No surgical explorations or neurolysis was required.

Vascular complications

All vascular compromised injuries were managed with urgent closed reduction and cross k-wire pinning after which the vascular status was re-evaluated. An absent or reduced pulse was noticed in 21 (6.3%) of children on admission. “Pucker sign” and ecchymoses were seen in 3 children on admission, a sign of fracture penetration of the brachialis and commonly seen with vascular injuries.[59,60] These 3 reduced with CRIF and the pulse returned in all 3 by day 2. All vascular cases were type III fractures with 18 posterolaterally displaced, which also corresponds with literature findings[7,18,24,29]. Associated neurological injuries were found in 4 cases. Median nerve injury was found in only 2 of these cases. The ulnar nerve was iatrogenically injured in the other 2 cases. The pulse returned in 13 children post CRIF. One fracture was irreducible and managed in straight lateral traction. A residual pulse deficit was found in 8 patients. All these patients had pink, pulseless hands

with normal capillary filling. Return of radial pulse was observed in three children by day three post-op. The other 5 pulseless, pink hands had normal radial pulses at three week follow up. Average time to return of pulse: 6.3 days (range 0-21 days). No vascular explorations were done. No compartment syndromes or Volkmann's ischaemic contactures were encountered.

Table 3

Average time to return of neurovascular function

Injured	Average time	Range
Radial Nerve	68.3 days	21-106 days
Median Nerve	53.6 days	21-180 days
Ulnar Nerve	72.3 days	18-193 days
Pulse	6.3 days	0-21 days

Irreducible fractures

Six fractures were irreducible in theatre. All were managed with straight lateral traction. Two of these children were late presenters arriving at 34 and 192 hours post injury respectively. They were noted to be swollen but had no evidence of compartment syndrome despite one having no radial pulse. This child had an absent pulse but well perfused hand (pink, pulseless and good capillary filling) and an AIN palsy. The pulse returned on day 2 in traction, the AIN function on day 21. All went on to uneventful union. There were 14 ipsilateral forearm fractures. Ten were Salter-Harris 2 distal radius fractures. All 14 were k-wired and united without complications.

Open fractures

Open fractures were seen in 3 children. All explored, debrided, washed out and wired (ORIF). All open fractures were Gustilo and Anderson[57] type 1 fractures, with anterior cubital fossa wounds. None had neurovascular complications and all fractures united.

Varus malunion

Varus malunion was found in 11 cases.

Type IIA: 3/168= 2%

Type IIB: 2/43 = 4%

Type III : 6/287= 2%

All 11 with varus had a poor outcome according Flynn's criteria.

Statistical analysis

Statistical analysis was done by McNemar 2 by 2 table for paired categorical data. The 13/21 absent pulses which returned post closed reduction and internal fixation (MUA + CRIF) had a P value= 0.02. This was statistically significant ($P<0.05$) for return of pulse post MUA + CRIF which corresponds with literature findings that this is the ideal initial treatment. All radial nerve palsies were seen with type III posteromedially displaced fractures corresponding with literature. We found similar correspondence of results in median nerve palsies: all posterolaterally displaced type III fractures. 18/21 vascular injuries were type III posterolateral displacement, the most common displacement associated with vascular compromise.

TABLE 4

RADIAL NERVE INJURY

	DOB	Sex	Side	#	Pre Op Assoc Injuries	Time from injury to surgery	Manage-ment	Post Op Neurology	Baumann's Angle° L	R	Assoc Fractures/ Complicatio	Time to recovery
1	21/4/94	M	R	T3PM	RN palsy	11 hours	CRIF	RN palsy UN palsy	80	85	Iatrogenic ulnar nerve palsy	90 days RN + UN recovery
2	28/8/00	F	L	T3PM	RN palsy	4	CRIF	RN palsy	78	76		21 days
3	13/6/95	F	L	T3PM	RN	5	CRIF	RN	80	78		90 days
4	14/3/98	F	R	T3PM	RN	7	CRIF	RN	75	75	R forearm rad/ulna # k-wired	106 days
5	25/7/97	M	R	T3PM	RN	8	CRIF	RN	75	76		101 days
6	16/10/94	F	L	T3PM	RN	4	CRIF	RN	80	78		21 days
7	5/9/92	M	L	T3PM	RN	4	CRIF	RN	81	70	CA 15°	49 days

TABLE 5

MEDIAN NERVE INJURY

	DOB	Sex	Side	#	Pre Op Assoc Injuries	Time from injury to surgery	Manage-ment	Post Op Neurology	Baumann's Angle° L	R	Assoc Fractures/ complication	Time to recovery
1	12/2/99	M	R	T3PL	MN palsy	4 hours	CRIF	MN	75	75		22 days
2	26/2/95	F	L	T3PL	MN	3	CRIF	MN	62	62		21 days
3	12/7/95	M	L	T3PL	MN	6	CRIF	MN UN palsy	68	70	Iatrogenic ulnar nerve palsy	180 days MN + UN Recovery
4	23/4/97	M	L	T3PL	AIN	7	CRIF	AIN	65	65		21 days
5	12/2/94	M	L	T3PL	MN No pulse	3	CRIF	MN Pink, pulseles Hand	70	70	Vascular compromise	21 days for pulse + MN recovery
6	11/7/02	M	L	T3PL	AIN No pulse	3	Irreducable SLR	AIN Pink, pulseles Hand	70	70	Vascular compromise Irreducable	2 days for pulse + 21 days for AIN
7	9/10/97	M	L	T3PL	?MN	21	CRIF	UN palsy MN diagnosed at 21 days	82	78	Iatrogenic ulnar nerve palsy	90 days UN + MN recovery. ? iatrogenic MN injury

TABLE 6

ULNAR NERVE INJURIES

	DOB	Sex	Side	#	Pre Op Assoc Injuries	Time from injury to surgery	Manage-ment	Post Op Neurology	Baumann's Angle° L	R	Assoc Fractures/ Complications	Time to recovery
1	1/9/00	F	L	T3PL		6	CRIF	D1 post op UN palsy diagnosed	80	75		90 days
2	15/2/02	M	L	T3PM		6	CRIF	D1 UN palsy	75	70		90 days
3	18/5/00	F	L	T3PM		5	CRIF	DI UN palsy	70	72	L wrist #, Salter-Harris 2 k-wired	150 days
4	14/11/98	M	L	T3PM		12	CRIF	D1 UN palsy	70	73		90 days
5	8/12/97	M	L	T3PM		15	CRIF	DI UN palsy	78	75		56 days
6	6/7/94	M	R	T3PM		18	CRIF	D1 UN palsy	70	70		180 days
7	10/10/00	M	L	T2B		15	CRIF	D1 UN palsy	62	70	No varus. Carrying angle 10°	18 days

	DOB	Sex	Side	#	Pre Op Assoc Injuries	Time from injury to surgery	Manage-ment	Post Op Neurology	Baumann's Angle° L	R	Assoc Fractures/ Complications	Time to recovery
8	21/4/94	M	R	T3PM	RN	11	CRIF	D1 UN palsy	80	85	Pre op RN palsy	90 days for UN + RN recovery
9	21/5/99	M	R	T3PM		7	CRIF	D4 UN palsy Diagnosed	70	75		21 days
10	23/12/99	M	R	T3PM		5	CRIF	D1 UN palsy	70	70		21 days
11	28/5/99	M	R	T2B		5	CRIF	D1 UN palsy	70	70		56 days
12	12/7/95	M	L	T3PL	MN	6	CRIF	DI UN palsy	68	70	Pre op MN palsy	180 days for UN + MN recovery
13	23/2/00	F	R	T3PM	No pulse	7	CRIF	D1 UN palsy Pink, pulseless hand	79	74	Vascular compromise	193 days for UN recovery. 3 days for pulse
14	11/8/99	M	R	T3PM		4	CRIF	D1 UN palsy	80	80		21 days
15	9/10/97	M	L	T3PL	?MN	21	CRIF	D1 UN palsy. MN diagnosed at 21 days	82	78	?iatrogenic MN injury	90 days UN+ MN recovery

	DOB	Sex	Side	#	Pre Op Assoc Injuries	Time from injury to surgery	Manage-ment	Post Op Neurology	Baumann's Angle° L	R	Assoc Fractures/ Complications	Time to recovery
16	18/7/98	F	L	T3PL		8	CRIF	D1 UN palsy	65	65		21 days
17	27/6/98	F	L	T3PM	No pulse	5	CRIF	D1 UN palsy Pulse Returned post CRIF	90	80	Vascular compromise No varus , carrying angle 12°	21 days UN recovery. Pulse returned post CRIF
18	12/5/94	M	L	T3PL		6	CRIF	D1 UN palsy	68	67		21 days
19	2/2/95	M	L	T3PL		2	CRIF	D1 UN palsy	76	76		156 days
20	22/11/95	M	L	T3PL		10	CRIF	D1 UN palsy	73	76		21 days
21	13/6/95	F	L	T2B		5	MUA Lost position K-wired	D1 UN palsy	74	80	MUA + POP failed. CRIF at 1 week	80 days
22	2/7/01	F	L	T3PL		9	CRIF	D6 UN palsy diagnosed	70	72		72 days. UN palsy diagnosed at D6.
23	25/2/00	F	R	T3PL		6	CRIF	D3 UN palsy diagnosed	74	72		90 days

	DOB	Sex	Side	#	Pre Op Assoc Injuries	Time from injury to surgery	Manage-ment	Post Op Neurology	Baumann's Angle° L	R	Assoc Fractures/ Complications	Time to recovery
24	27/6/98	F	R	T3PM		5	CRIF	D1 UN palsy	70	70		21 days
25	30/6/02	F	L	T3PL		4	CRIF	D1 UN Palsy	70	72		78 days

TABLE NO 7

VASCULAR INJURIES

No	DOB	Sex	Side	#	Vascular status pre-op	Time from injury to surgery	Manage-ment	Vascular status post op	Baumann's angle° L	Baumann's angle° R	Assoc injuries	Time to recovery
1	13/6/95	M	R	T3PL	Weak pulse Capillary filling (CF) normal	5 hours	CRIF	Pulse returned	70	70		Post-op normal
2	31/5/01	F	R	T3PL	Absent pulse CF normal Soft tissue "tented" over fracture ? pucker sign	6	CRIF	Absent pulse CF normal	74	70		Pulse returned day 2
3	8/3/00	F	L	T3PL	Absent pulse CF normal	7	CRIF	Pulse returned	70	70		Post-op normal
4	5/12/96	F	R	T3PM	Absent pulse Doppler normal	4	CRIF	Pulse returned	70	70		Post-op normal
5	5/3/03	M	L	T3PL	Weak pulse CF normal	13	CRIF	Pulse returned	72	72		Post-op normal

No	DOB	Sex	Side	#	Vascular status pre-op	Time from injury to surgery	Manage-ment	Vascular status post op	Baumann's angle° L	Baumann's angle° R	Assoc injuries	Time to recovery
6	15/5/00	M	L	T3PL	Weak pulse CF normal	10	CRIF	Pulse returned	78	70	CA 10°	Post-op normal
7	15/3/96	M	L	T3PL	Weak pulse CF normal	4	CRIF	Weak pulse CF decreased but <4 sec	74	74		Pulse returned day 21. ?entrapment
8	13/5/98	F	L	T3PL	Absent pulse Absent Doppler	5	CRIF	Pulse returned	72	74		Post-op Normal
9	12/11/9[illegible]	M	R	T3PL	Pucker sign Swollen. CF + pulse normal	7	CRIF	Pulse returned	80	78		Post-op Normal
10	29/10/9[illegible]	M	L	T3PL	Weak pulse CF decreased	4	CRIF	Pulse returned	82	82		Post-op Normal

No	DOB	Sex	Side	#	Vascular status pre-op	Time from injury to surgery	Manage-ment	Vascular status post op	Baumann's angle° L	Baumann's angle° R	Assoc injuries	Time to recovery
11	11/7/02	M	L	T3PL	Absent pulse CF normal AIN palsy	3	CRIF failed. SLR	Absent Pulse in traction CF normal Pink, pulseless	70	70	AIN palsy	Pulse returned day 2 in traction AIN day 21
12	16/12/99	M	R	T3PL	Weak pulse CF normal	3	CRIF	Pulse returned	70	70		Post-op Normal
13	12/11/96	M	L	T3PL	Weak pulse CF normal	3	CRIF	Pulse returned	75	75		Post-op Normal
14	27/6/98	M	L	T3PM	No pulse CF not noted	5	CRIF	Pulse returned	90	80	CA 12° UN k-wired	Post-op normal pulse UN day 21
15	20/9/96	F	L	T3PL	Weak pulse Ecchymosis ? pucker sign	2	CRIF	Pulse returned	70	72		Post-op Normal

No	DOB	Sex	Side	#	Vascular status pre-op	Time from injury to surgery	Manage-ment	Vascular status post op	Baumann's angle° L	Baumann's angle° R	Assoc injuries	Time to recovery
16	4/12/96	M	L	T3PL	Absent pulse Swollen No compartment syndrome	8	CRIF	Absent Pulse CF normal Pink, pulseless	74	72		Pulse returned day 21
17	21/2/94	M	L	T3PL	Absent pulse MN palsy	3	CRIF	Absent Pulse CF normal Pink, pulseless	70	70	MN palsy	Pulse returned day 21. MN day 21
18	23/2/00	F	R	T3PM	Absent pulse	7	CRIF	Absent Pulse CF normal Pink, pulseless	79	74	UN k-wired	Pulse returned day 3. UN day 193
19	10/5/00	M	R	T3PL	Weak pulse CF normal	10	CRIF	Pulse returned	70	74		Post-op Normal

No	DOB	Sex	Side	#	Vascular status pre-op	Time from injury to surgery	Manage-ment	Vascular status post op	Baumann's angle° L R	Assoc injuries	Time to recovery
20	16/7/00	F	R	T3PL	Absent pulse CF normal	9	CRIF	Absent Pulse CF normal Pink, pulseless	67 68		Pulse returned day 21
21	14/2/98	M	L	T3PL	Absent pulse CF normal	6	CRIF	Absent Pulse CF normal Pink, pulseless	78 78		Pulse returned day 21

Discussion

Neurovascular injury is a common occurrence in extension-type supracondylar fractures of the humerus.[3,4,7,9,11,15,37,38,39,40,41,42,43] The close proximity of neurovascular structures to bone renders them at risk to injury.[18] Type III severely displaced fractures have the highest incidence of neurovascular complications.[11,22,26,27,33,58] The management of supracondylar fractures is also associated with many complications.[11] Neurological injury is either due to the initial injury or iatrogenically due to fracture manipulation and k-wire fixation. The ulnar nerve is most commonly injured iatrogenically.[1,8,36,44] The management of vascular compromise causes most distress in surgeons. Fortunately the pulse returns post manipulation and k-wiring in more than 80% of cases.[9,24,61] The management of these rare remaining cases of absent pulse is widely debated. Great controversy exists over the ideal management of neurovascular complications.

I have embarked on a literature review to ascertain where there was consensus in the management of neurovascular complications following supracondylar fractures. I have provided an outline for the treatment of these injuries. Contentious issues with regard to neurovascular complications and their management have been dealt with separately and I have compared the management of these complications with our own conservative outcomes.

Outline of management

In 1959 Gartland[28] noted, “It is interesting to observe the trepidation with which men, otherwise versed in trauma, approach a fresh supracondylar fracture”.[28,55]

The initial clinical assessment of the supracondylar fracture is critical. A careful and thorough neurovascular examination is necessary.[18,43] This is best to judge subsequent changes in neurovascular status against.[55] Some authors also feel a neurological injury at presentation is a red flag, with high risk for soft tissue injury and compartment syndrome.[48]

The neurovascular examination is difficult in the injured child.[17,33] Great care should be taken as injuries are often missed.[17,24,26,55,62]

Meticulous neurological examination of the ulnar, radial and median nerves should be performed. The anterior interosseous nerve (AIN) should be specifically examined. An AIN injury has subtle signs which are oft overlooked.[1,19,21,30] Vascular examination involves evaluating in combination the radial pulse, capillary filling, temperature, colour, swelling, oxygen saturation monitoring and Doppler if no pulse felt. Careful examination of the cubital fossa for ecchymosis, pucker sign and bruising should be done. These signs indicate penetration of brachialis muscle by proximal fracture end and are associated with neurovascular compromise.[18,59,60] Pre-operative angiography in vascular cases remains controversial.[2,6,14,26] A high index of suspicion for compartment syndrome should be maintained.[35,47,48,50] The elbow should be splinted in less than 90° flexion to minimize compartment pressures.[51] If in doubt compartment pressures should be measured. Ipsilateral forearm fractures occur in 10-15% and the careful forearm examination is neccessary.

The aim of treatment is an anatomically reduced and united fracture with full range of motion and a normal cosmetic appearance, as well as being free of complications. [11] All type III fractures should be managed as emergencies. Closed reduction and k-wire fixation (CRIF) is associated with the best outcomes.[11,12,13,14] No consensus exists over the ideal pin configuration with proponents of both crossed k-wires and lateral wires respectively.[36] CRIF however, is not easy. A very thin area of bony contact exists at the fracture site. A small amount of rotation leads to a large amount of varus.[63] It is also extremely difficult to k-wire a poorly reduced fracture. [7] Accurate reduction of the fracture is vital. The fracture may be irreducible/ poorly reduced for a number of reasons. Soft tissue / brachialis muscle interposition, neurovascular entrapment[59,60] or delayed presentation with swelling. Management once more is controversial with some authors advocating ORIF whilst others use straight lateral traction (SLR). [35,46,56,64] Post CRIF, if the pulse remains absent without capillary filling and the hand is ischaemic the exploration of the brachial artery and ORIF should be undertaken.[11,14,22,40] All compartment syndromes should be explored with

fasciotomy. The management of the pulseless, well-perfused arm (the so called pink, pulseless hand) with good capillary filling remains controversial.

Careful post-operative neurovascular assessment and compartment checks are mandatory. Delayed onset neurovascular injuries and compartment syndrome should be vigilantly looked for.[62] Iatrogenic injuries may also be documented.

Neurological complications

The management of neurological complications in supracondylar fractures continues to elicit debate. Two broad groups of injuries exist: 1) Median and radial nerve palsies 2) Iatrogenic ulnar nerve palsies.

Some authors feel median and radial nerve injuries require surgical exploration. Aronson et al[65] feel if the fracture is irreducible it requires surgical exploration to remove obstruction to reduction which may be neurological. Louahem et al[33] explore all complete radial nerve palsies post-op as it denotes nerve discontinuity, as well as nerve lesions with partially reduced fractures where nerve entrapment is suspected. Luria et al[26] and Rasool[18] explore all median nerve injuries associated with vascular compromise as they feel entrapment is the likely cause.

Many authors however feel median and radial nerve injuries are neuropraxias and shall resolve spontaneously.[4,7,11,13,15,16,17,19,20,21,22,23,32,38,39,40,41] We agree with Culp et al[23] that immediate exploration is of little help. There exists the potential for iatrogenic injury during surgical exploration and open surgery is certainly not free of complications.[6,13,24,26] Most nerves recover over 3-6 months.[21,24,33] Furthermore studies show that late exploration and neurolysis of nerves in continuity renders good results.[19,23,33] Our study had 14 radial and median nerve injuries. All recovered uneventfully on conservative management by six months. We agree with the literature and feel these nerve injuries were all neuropraxias and thus recovered. We also feel that should the nerve injury not recover by six months exploration and neurolysis should be undertaken, with good results. This approach will also spare a large number of children unnecessary and potentially dangerous operations.

Iatrogenic ulnar nerve injuries were responsible for most of our nerve injuries (25/39-64%). The nerve is classically injured by the medial pin. Anatomically the nerve runs in the ulnar groove posterior to the medial epicondyle. Here it is bridged by the cubital retinaculum, which creates a fibro-osseous tunnel for the nerve. During flexion the biomechanics of the tunnel change. The tunnel size decreases, the ulnar nerve and retinaculum stretches and the nerve may sublux anteriorly.[18,44]The hyperlax/ subluxing ulnar nerve has a prevalence of 6-18%.[66,67] The ulnar nerve is thus very vulnerable to injury during flexion and medial pinning.[6,36,44] The majority of iatrogenic injuries are due to pinning in flexion, and tightening of the retinaculum which traps the nerve in this position injuring it.[10.16.36.44] Interestingly biomechanical studies also show the ulnar nerve falls posteriorly with extension of the elbow.[8,18,36,66,67]

Rasool[18,44] , Brown et al[42], Schoeneker et al[14] and Wilkins[7] surgically explore all iatrogenic ulnar nerve injuries. They feel this allows you to address the pathology, release the retinaculum constricting the nerve and to safely resite the medial pin. They believe this offers the nerve its best chance at recovery. We managed all 25 ulnar nerve palsies conservatively. We believe the injury to be neuropraxic and shall recover spontaneously. If diagnosed early the medial pin was removed. All 25 nerves returned to normal function by 193 days. We agree with the literature that recovery takes longest in injured ulnar nerves (average 72.3 days).[33] Review of our x-rays revealed 3 common medial pin placement errors. The pin was a) too vertical >40°, b) too lateral and c) in the ulnar groove.[18,44] More accurate and careful placement of the medial pin is necessary. Conservative management of iatrogenic ulnar nerve injuries yields good results, with all our 25 injured ulnar nerves making a full recovery. The injury is mostly neuropraxic and heals spontaneously. Lyons et al[20] also believe the injury to be neuropraxic and had full recovery of the nerve despite not removing the medial pin in certain cases. We feel surgical exploration with neurolysis is only necessary if no improvement by 6 months. [23]

Lateral pinning is proposed as a solution to iatrogenic ulnar nerve injuries.[11,16,68,69] We have some reservations about using this technique over cross-pinning. Lateral pinning

biomechanically has less stability in comparison to cross-pinning.[70,71] The instability is largely rotational which is concerning as internal rotation classically leading to most varus deformities. Lateral pinning is also more difficult to perform.[16,36] Brauer et al[8] in her exhaustive study found lateral pinning to have a 2% incidence of ulnar nerve palsy (thus not free of iatrogenic palsy). Moreover, Babal et al[1] felt there was underreporting of nerve injuries with lateral pinning. Furthermore, when faced with pin tract sepsis (2% incidence) the treatment is removal of the pin. In this case stability would be better held with cross-pinning once wire is removed. We thus prefer cross-pinning as our choice of fixation despite a slightly higher rate of iatrogenic nerve palsy.

Our high incidence of iatrogenic palsies caused us concern. After critically evaluating our results and surgical technique, we felt more attention to medial pinning technique was necessary. A new policy was thus devised with regards medial pinning as per Eidelman et al[36] with modification. The opposite elbow is palpated pre-op to ascertain if the ulnar nerve subluxes and its position. The lateral 2 k-wires are pinned first after fracture reduction. The adhesive elastoplast tape is then removed, the arm extended allowing the ulnar nerve to fall posteriorly.[36] The medial pin is placed anterior on the epicondyle, watching the hand for any twitches. If twitches are observed the pin is resited.[58] The pin is aimed from anteromedial to posterolateral and wire is advanced at 40° or less. The opposite cortex must be engaged and the wires cross above the fracture.[55] We do not use a mini-open medial incision as Green et al[63] as we feel our new approach is safe and that the mini medial approach still has a risk of nerve injury.[36] This method we feel is safe, reproducable and reliably avoids injury to the ulnar nerve.

Vascular complications

Vascular complications pose a great management dilemma to the orthopaedic surgeon.[53] The spectrum of injury ranges from a decreased pulse to an absent pulse with an ischaemic hand and compartment syndrome. There are several reasons for an absent pulse. These may be due to arterial compression, spasm, laceration, intimal flap with or without a intramural thrombus or entrapment in the fracture site. Most authors agree CRIF should be done

urgently with re-evaluation of the vascular status. We agree that a pulseless, ischaemic hand post-op should be surgically explored. The management of the pulseless, well-perfused hand with good capillary filling (pink, pulseless hand) post-op however causes most controversy. Very little is known of the natural history of pink, pulseless hands.[2,53] One reason is that this presentation is so rare.

Several authors are pro surgical exploration of all pink, pulseless hands.[2,9,11,14,18,26,27,31,38,47] They maintain that the brachial artery is injured.[2,9,26,38] Pre-operative vascular studies and specifically angiography are condemned[2,6,14,22,72] It is felt angiography is invasive, technically difficult and has a high risk for iatrogenic damage. Angiography also causes an unnecessary delay and is not necessary as the level of arterial injury is at the fracture site. This unfortunately leaves the surgeon in the dark as to the cause of vascular compromise. An anterior approach to the cubital fossa to explore the neurovascular bundle is thus advocated.[65] It is felt this approach has a low morbidity and allows the surgeon to address the pathology. Here the damaged artery may be repaired, a thrombus may be removed, and entrapment may be relieved.[18,59,60] By addressing the pathology the authors feel the child is afforded the best chance for vascular recovery. Long-term ischaemic complications of claudication, fatigueablility, cold intolerance and limb length discrepancy are so too best avoided.[2] To support this they quote early return of pulse and imaging studies and pressure studies to confirm successful repair.[2,9,14,26] Potential loss of limb it is felt is prevented, and so too the complications such as thrombus propagation. [2] The conservative management of pink, pulseless hands is frowned upon by these authors. They feel that conservative management ignores potential brachial artery injury and is far too reliant on the collateral blood supply of the elbow to reconstitute the blood flow. White et al[2] refers to this reliance on collateral blood flow as "…to take a calculated gamble…".

The pink, pulseless hand is however managed with success by conservative means by several authors.[3,6,7,15,21,23,24,28,29,32,33,35,40,45,53,54,55] The pulse returned in the pink, pulseless hand with observation in all our cases. It is felt to be due to relief of arterial spasm or compression or the rich collateral blood supply around the elbow which reconstitutes the

blood flow to the arm in cases of arterial injury or occlusion.[15,19,21,23,25,33,40,73] Many pro-surgical exploration authors do not discern between pink, pulseless hands and ischaemic hand exploration. White et al[2] in her large review noted that there was a bias toward surgery in most of the studies she reviewed. ORIF and exploration are not free of complications either. Noamen et al[9] had 7 relooks out of 31 explorations. Gosens[24] reported cases of iatrogenic nerve injury. Luria et al[26] had a false positive duplex scan for no pulse and thus a negative exploration which led to osteomyelitis. Luria[2] also unnecessarily operated on a further 2 patients found to have arterial spasm. Scar complications are common and may lead to decreased motion of the elbow.[26] A blind surgical approach regime to these injuries may reap many unnecessary operations and complications. Exploration and vascular repair is difficult surgery in adults, requiring meticulous technique which is not always replicable in children.[74] Surgical exploration and repair was shown by Subharwal et al[15] to have high asymptomatic reocclusions and stenosis. In this landmark study 45% of patients post surgical brachial artery repair in supracondylar fractures had reocclusion or stenosis. Good collateral blood supply was found supplying the distal forearm in these cases, thus explaining the asymptomatic presentation. These findings were ascertained by magnetic resonance angiography (MRA) combined with duplex colour Doppler and pressure studies. It is interesting to note that very few studies evaluating the post-op patency of the brachial artery repair have been done.[2,15] In the absence of angiography due to iatrogenic complications and other reservations, the ideal evaluation of brachial artery patency is yet to be found. It is with great interest to find many brachial artery repair studies base their success on palpable distal pulses and pressure studies. No other post-op vascular studies have quoted collateral blood supply status bar Subharwal[15] These successes thus, in light of Subharwal's[15] findings, could well be due to collateral blood flow, as seen in the conservative management of the pink, pulseless hand.

Five of our eight pink, pulseless hands regained their pulse within 3 days. The other three had a normal pulse by 21 days. All patients made a full recovery. No compartment syndromes were seen. We feel the importance of long-term ischaemic complications is

overstated. We agree with Choi et al[45] and Robbs[53] that the long-term complications are minimal in literature, with most authors recommending observation. Lally et al[73] further showed the long-term vascular sequelae in children who had brachial artery ligation for insertion of a Scribner shunt were minimal. Collateral blood supply to the elbow was found to sufficiently and consistently reconstitute distal bloodflow. In the absence of conclusive studies providing proof of vascular patency post repair, and in light of our study's outcome, we feel the pink, pulseless hand post-op may be watched and managed conservatively with vigilant neurovascular observation. We believe the rich collateral blood supply to the elbow sufficient to compensate for vascular compromise in this situation. We once more have reservations about the iatrogenic damage caused by surgical exploration as well as unnecessary operations performed on children that would otherwise have done well with observation.

Criticism

Neurological

The AIN is the most commonly injured nerve in supracondylar fractures.[1,7,17,30-35] It was documented in only 2 cases in our study. The AIN palsy is well known in the literature for its subtle signs.[6,30,45,62] Babal et al[1] and McGraw et al[21] noted that the AIN is commonly overlooked in assessment. Babal[1] also mentions that the AIN is regularly referred to as a median nerve injury, further explaining the low incidence in some studies. We agree the reason for our low incidence of AIN palsy was due either to the injury being overlooked or not specifically tested for. We currently insist on documentation and careful examination of the AIN in all cases.

No electromyography (EMG) or nerve conduction studies (NCS) were done. No specialised strength testing or fatigueability tests were done. All patients were followed-up until normal clinical nerve function was noted.

Vascular

Follow-up was until a normal pulse was re-established. No angiograms, Duplex colour Doppler, Magnetic Resonance Angiogram or specialised pressure testing were done. No long-term ischaemic complication follow-up or testing (i.e. cold intolerance, strength and claudication) was performed. We do feel that these complications are minimal and may be managed with observation.

Conclusion

Neurovascular complications in extension-type supracondylar humerus fractures can be managed conservatively if there is good capillary filling post-reduction.

Neurological injuries are mostly neuropraxic which recover spontaneously. If there is no neurological improvement at 6 months we recommend exploration and neurolysis. Vascular compromise as seen in the pink, pulseless hand post CRIF may be managed conservatively with observation.

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Final drafts are in progress for publication of this data in an orthopaedic journal.

Abbreviations

PM- posteromedial

PL- posterolateral

AIN- anterior interosseous nerve

CRIF- closed reduction and internal fixation (percutaneous pinning)

ORIF- open reduction and internal fixation

POP- Plaster of Paris

SLR- straight lateral traction

EMG- electromyogram

RN- radial nerve

UN- ulnar nerve

MN- median nerve

CF- capillary filling

T3PM- type III fracture with posteromedial displacement

T3PL- type III fracture with posterolateral displacement

T2B- type II B fracture

- fracture

CA- carrying angle of the elbow

References

[1] Babal JC, Mehlman CT, Klein G. Nerve injuries associated with pediatric supracondylar humerus fractures: A meta-analysis. *J Pediatr Ortop.* 2010; 30: 253-263

[2] White L, Mehlman CT, Crawford AH. Perfused, Pulseless and Puzzling: A systematic review of vascular injuries in pediatric supracondylar humerus fractures and results of a POSNA questionnaire. *J Pediatr Orthop.* 2010; 30: 328-335

[3] Cheng JCY, Lam TP, Shen WY. Closed reduction and percutaneous pinning for Type III displaced supracondylar fractures in children. *J Orthop Trauma.* 1995; 9: 511-515

[4] Flynn JC, Matthews JG, Benoit RL. Blind pinning of supracondylar fractures of the humerus in children. 16 Years' experience with long term follow up. *J Bone Joint Surg Am.* 1974; 56-A: 263-272

[5] Slobogean BL, Jackman H, Tennant S, Slobogean GP, Malpuri K. Iatrogenic nerve injury after the surgical treatment of displaced supracondylar fractures of the humerus: number needed to harm, A systematic review. *J Pediatr Orthop.* 2010; 30: 430-4

[6] Griffin KJ, Walsh SR, Tang TY, Boyle JR, Hayes PD. The pink pulseless hand: A review of the literature regarding management of vascular complications of supracondylar humeral fractures in children. *Eur J Endovasc Surg.* 2008; 36: 697-702

[7] Wilkins K, Beaty JH .I n Rockwood: *Fractures in Children.* Vol 3. 4th ed. Philadelphia, Pennsylvania: Lippincitt-Raven.1996

[8] Brauer CA, Lee BM, Bae DS, Waters PM, Kocher MS. A systematic review of medial and lateral entry pinning versus lateral entry pinning for supracondylar fractures of the humerus. *J Pediatr Orthop.* 2007; 27: 181-186

[9] Noamen HH. Microsurgical reconstruction of brachial artery injuries in displaced supracondylar fracture humerus in children. *Microsurgery.* 2006; 26: 498-504

[10] Beaty JH, Kasser JR. In Rockwood + Wilkins: *Fractures in children.* Vol. 3 5th edition. Philadelphia: Lippincott, Williams and Wilkins; 2001: 576-624

[11] Pirone AM, Graham HK, Krajbich JI. Management of displaced extension-type supracondylar fractures of the humerus in children. *J Bone Joint Surg Am.* 1988. 70-A:641-649

[12] Hamdi A, Poitras P, Louati H, Dagenais S, Masquito JJ, Kontio K. Biomechanical analysis of lateral pin placements for pediatric supracondylar humerus fractures. *J Pediatr Orthop.* 2010; 30: 135-139

[13] Padman M, Warwick AM, Fernandes JA, Flowers MJ, Davies AG, Bell MJ. Closed reduction and stabilization of supracondylar fractures of the humerus in children: the crucial factor of surgical experience. *J Pediatr Orthop Br.* 2010; 19: 298-303

[14] Schoeneker PL, Delgado E, Rotman M, Sicard GA, Capelli AM. Pulseless arm in association with totally displaced supracondylar fracture. *J Orthop Trauma.* 1996;10: 410-415

[15] Subharwal S, Tredwell SJ, Beauchamp RD, Mackenzie WG, Jakubec DM, Cairns R, LeBlanc JG. Management of pulseless pink hand in pediatric supracondylar fractures of the humerus. *J Pediatr Orthop.* 1997;17: 303-310

[16] Skaggs DL, Cluck MW, Mostofi A, Flynn JM, Kay RM. Lateral-entry pin fixation in the management of supracondylar fractures in children. *J Bone Joint Surg Am.* 2004; 86: 702-707

[17] Shrader M Wade. Pediatric supracondylar fractures and pediatric physeal elbow fractures. *Orthop Clin N Am.* 2008; 39: 163-171

[18] Rasool MN. Neurovascular injuries in supracondylar fractures of the elbow in children- anatomical considerations. *SA Ortopaedic Journal.* 2005 August: 28-40

[19] Lipscombe PR, Burleson RJ. Vascular and neural complications in supracondylar fractures of the humerus in children. *J Bone Joint Surg Am.* 1955; 37: 487-492

[20] Lyons JP, Ashley E, Hoffer MM. Ulnar nerve palsies after percutaneous cross-pinning of supracondylar fractures in childrens' elbows. *J Pediatr Orthop.* 1998;18: 43-4

[21] McGraw JJ, Akbarnia BA, Hanel DP, Keppler L, Burdge RE. Neurological complications resulting from supracondylar fractures of the humerus in children. *J Pediatr Orthop.* 1986;6: 647-650

[22] Shaw BA, Kasser JR, Emans JB, Rand FF. Mangement of vascular injuries in displaced supracondylar humerus fractures without arteriography. *J Orthop Trauma.* 1990;4: 25-29

[23] Culp RW, Osterman AL, Davidson RS, Skirven T, Bora FW. Neural injuries associated with supracondylar fractures of the humerus in children *J Bone Joint Surg Am.* 1990. 72-A; 8: 1211-1215

[24] Gosens T, Bongers KJ. Neurovascular complications and functional outcome in displaced supracondylar fractures of the humerus in children. *Injury.* 2003; 34: 267-273

[25] Mangat KS, Martin AG, Bache CE. The 'pink pulseless' hand after supracondylar fracture of the humerus in children. The predictive value of nerve palsy. *J Bone Joint Surg Br.* 2009. 91-B; 11: 1521-5

[26] Luria S, Sucar A, Eylon S, Pinchas-Mizrachi R, Berlatzky Y, Anner H, Liebergall M, Porat S. Vascular complications of supracondylar humeral fractures in children. *J Pediatr Ortop B.* 2007;16 : 133-143

[27] Copley LA, Dormans JP, Davidson RS. Vascular injuries and their sequelae in pediatric supracondylar humerus fractures: toward a goal of prevention. *J Pediatr Orthop.* 1996;16: 99-103

[28] Gartland JJ. Management of supracondylar fractures of the humerus in children. *Surg Gynaecol Obstet.* 1959; 109: 145-154

[29] Lyons ST, Quinn M, Stanitski CL. Neurovascular injuries in type III supracondylar fractures in children. *Clin Orthop Realt Res.* 2000; 376: 62-7

[30] Spinner M, Schreiber S. Anterior interosseous nerve palsy as a complication of supracondylar fractures of the humerus in children. *J Bone Joint Surg Am.* 1969; 51-A: 1584-90

[31] Dormans JP, Squillante R, Sharf H. Acute neurovascular complications with supracondylar humerus fractures in children. *J Hand Surg Am.* 1995; 20: 1-4

[32] Campbell CC, Waters PM, Emans JB, Kasser JR, Millis MB. Neurovascular injury and displacement in type III supracondylar humerus fractures. *J Pediatr Orthop.* 1995; 15: 47-52

[33] Louahem DM, Nebunescu A, Cavanese F, Dimeglio A. Neurovascular complications and severe displacement in supracondylar humerus fractures in children: defensive or offensive strategy? *J Pediatr Orthop B.* 2006; 15: 51-57

[34] Cramer KE, Green NE, DeVito DP. The incidence of anterior interosseous nerve injury in supracondylar fractures on children. *J Pediatr Orthop.* 1993; 13: 502-5

[35] Piggot J, Kerr Graham H, McCoy GF. Supracondylar fractures of the humerus in children. Treatment by straight lateral traction. *J Bone Joint Surg B.* 1986; 68-B: 577-583

[36] Eidelman M, Hos N, Katzman A, Bialik V. Prevention of ulnar nerve injury during fixation of supracondylar fractures in children by ' flexion-extension cross-pinning' technique. *J Pediatr Orthop B.* 2007; 16: 221-224

[37] Karakurt L, Ozdemir H, Yilmaz E, Inci M, Belhan O, Serin E. Morphology and dynamics of ulnar nerve in the cubital tunnel after percutaneous cross pinning of supracondylar fracture's in childrens elbows: an ultrasound study. *J Pediatr Orthop B.* 2005; 14: 189-193

[38] Korompilias AV, Lykissas MG, Mitsionis GI, Kontogeorgakos VA, Manoudis G, Beris AE. Treatment of pink pulseless hand following supracondylar fractures of the humerus in children. *International Orthopaedics.* 2009; 33: 237-241

[39] Iobst CA, Spurdle C, King WF, Lopez M. Percutaneous pinning of pediatric supracondylar fractures with semisterile technique. The Miami experience. *J Pediatr Orthop.* 2007; 27: 17-22

[40] Garbuz DS, Leitch K, Wright JG. The treatment of supracondylar fractures in children with an absent radial pulse. *J Pediatr Orthop.* 1996; 16: 594-596

[41] Galbraith KA, McCullough CJ. Acute nerve injury as a complication of closed fractures or dislocations of the elbow. *Injury.* 1979; 11: 159-164

[42] Brown IC, Zinar DM. Traumatic and iatrogenic neurological complications after supracondylar humeral fractures in children. *J Pediatr Orthop.* 1995; 15: 440-3

[43] Royce RO, Dutkowsky JP, Kasser JR, Rand FR. Neurological complications after k-wire fixation of supracondylar humerus fractures in children. *J Pediatr Ortop.* 1991; 11: 191-4

[44] Rasool MN. Ulnar nerve injury after k-wire fixation of supracondylar humerus fractures in children. *J Pediatr Orthop.* 1998; 18: 686-690

[45] Choi PD, Melikian R, Skaggs DL. Risk factors for vascular repair and compartment syndrome in the pulseless supracondylar humerus fracture in children. *J Pediatr Orthop.* 2010; 30: 50-56

[46] Dodge HS . Displaced supracondylar fractures of the humerus in children- treatment by Dunlop's traction. *J Bone Joint Surg Am.* 1972; 54(7):1408-1418

[47] Clement DA. Assessment of a treatment plan for managing acute vascular complications associated with supracondylar fractures of the humerus in children. *J Pediatr Orthop.* 1990; 10:97-100

[48] Ramachandaran M, Skaggs DL, Craford HA, Eastwood DM, Lalonde FD, Do TT, Kay RM. Delaying treatment of supracondylar fractures in children. Has the pendulum swung too far? *J Bone Joint Surg Br.* 2008, 90-B: 1228-33

[49] Mubarak SJ, Carroll NC. Volkman's ischaemia in children: aetiology and prevention. *J Bone Joint Surg Br.* 1979;61-B:487-92

[50] Mubarak SJ, Owens CA, Hargens AR, Garetto LP, Akeson WH. Acute compartment syndromes: Diagnosis and treatment with the aid of wick catheter. *J Bone Joint Surg Am.* 1978:60-A: 1090-5

[51] Battaglia TC, Armstrong DG, Schwend RM. Factors affecting forearm compartment pressures in children with supracondylar fractures of the humerus. *J Pediatr Orthop.* 2002: 22:431-9

[53] Robb JE. Annotation. The pink, pulseless hand after fracture of the humerus in children. *J Bone Joint Surg Br.* 2009; 91-B: 1410-12

[54] Gillingham BL, Rang M. Editorial. Advances in children's elbow fractures. *J Pediatr Orthop.* 1995; 15: 419-421

[55] Marquis CP, Cheung G, Dwyer JSM, Emery DFG. Supracondylar fractures of the humerus. *Current Orthopaedics.* 2008; 22: 62-9

[56] Dunlop J. Transcondylar fractures of the humerus in childhood. *J Bone Joint Surg.* 1939; 21: 59-73

[57] Gustilo RB, Anderson JT. Prevention of infection in the treatment of one thousand and twenty-five open of long bones. Retrospective and prospective analysis. *J Bone Joint Surg.* 58-A. 1976:453-8

[58] Bashyal RK, Chu JY, Schoeneker PL, Dobbs MB, Luhmann SJ, Gordon JE. Complications after pinning of supracondylar distal humeral fractures. *J Pediatr Orthop.* 2009; 29: 704-8

[59] Staples OS. Dislocation of the brachial artery: A complication of supracondylar fracture of the humerus in children. *J Bone Joint Surg Am.* 1965; 47: 1525-32

[60] Thomas AP. Entrapment of the proximal fragment of supracondylar fractures. *J Bone Joint Surg Br.* 1990; 72-B: 321-2

[61] Prietto CA. Supracondylar fractures of the humerus. A comparative study of Dunlop's traction versus percutaneous pinning. *J Bone Joint Surg Am.* 1979; 61: 425-8

[62] Got C, Tan TW, Thakur N, Marcaccio EJ, Eberson C, Madom I. Delayed presentation of brachial artery pseudo aneurysm after a supracondylar humerus fracture in a 6-year-old boy: A case report. *J Pediatr Orthop.* 2010; 30: 57-59

[63] Green DW, Widmann RF, Frank JS, Gardner MJ. Low incidence of ulnar nerve injury with crossed pin placement for pediatric supracondylar humerus fractures using mini-open technique. *J Orthop Trauma.* 205; 19: 158-63

[64] Kurer MHJ, Regan MW. Completely displaced supracondylar fracture of the humerus in children: A review of 1708 comparible cases. *Clin Orthopaedics.* 1990; 256: 205-14

[65] Aarondson DC, van Vollenhoven E, Meeuwis JD. K-wire fixation of supracondylar humerus fractures in children: results of open reduction via a ventral approach in comparison to closed treatment. *Injury.* 1993; 3: 179-81

[66] Childress HM. Recurrent ulnar nerve dislocation at the elbow. *Clinical Orthopaedics.* 1975;108: 168-73

[67] Zalz I, Waters PM, Kasser JR. Ulnar nerve instability in children. *J Paediatr Orthop.* 1996; 16: 567-9

[68] Topping RE, Blanco JS, Davis TJ. Clinical evaluation of crossed-pin versus lateral pin fixation in displaced supracondylar humerus fractures. *J Pediatr Orthop.* 1995; 15 : 435-9

[69] Skaggs DL, Hale JM, Bassa J et al, Operative treatment of supracondylar fractures of the humerus in children. The consequences of pin placement. *J Bone Joint Surg Am.* 2001; 83-1: 735-40

[70] Zoints LE, McKellop HA, Hathaway R. Torsional strength of pin configurations used to fix supracondylar fractures of the humerus in children. *J Bone Joint Surg Am.* 1994; 76: 253-6

[71] Lee SS, Mahar AT, Mielson D, Newton PO. Displaced pediatric supracondylar humerus fractures: Biomechanical analysis of percutaneous pinning technique. *J Pediatr Orthop.* 2002; 22: 440-3

[72] Holden CEA. The pathology and prevention of Volkmans ischaemic contracture. *J Bone Joint Surg.* 1979; 61-B: 296-300

[73] Lally KP, Foster CE, C hwals WP, Brennan LP, Atkinson JB. Long-term follow-up of brachial artery ligation in children. *Ann Surgery.* 1990; 212: 194-6

[74] Meek AC, Robbs JV. Are brachial artery injuries significant? *South African Journal of Surgery.* 1983; 21: 227-33