BRITISH SOCIETY FOR SURGERY OF THE HAND

Instructional Courses in Hand Surgery

NERVE INJURY & COMPRESSION, PAIN, ANAESTHESIA

16 - 17 JUNE 2017

Series 7, Course 2
Organising Committee:
L Muir
G E B Giddins
D J Brown
V Bhalaik
G Bourke
F Iwuagwu
Z Naqui
M Pickford
G D Smith
M Calcagni, FESSH Representative
S Fleming, BOTA Representative

9 June 2017

Dear Speaker/Participant,

Welcome to the second meeting of the seventh series of the Instructional Courses in Hand Surgery and to the Manchester Conference Centre.

The trade stands will be located in the Pioneer Room, where refreshments and lunch will be served on both days.

We would be most grateful if all participants visit the exhibition stands. I am sure you will appreciate that their attendance helps support these courses.

Since the introduction of online registration for the Instructional Courses, we no longer send out the programme and other details in advance. Information about the course can be found at:

http://www.bssh.ac.uk/about/events/2112/ichs_72_nerve_injury__compression_pain_anaesthesia

I will be present throughout the two days and if you have any queries, please do not hesitate to contact me.

Yours faithfully,

Charlotte Smith
Events and Committee Coordinator
## Friday 16 June

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Speaker(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>08:00</td>
<td>Registration and refreshments</td>
<td></td>
</tr>
<tr>
<td>08:50</td>
<td>Welcome and introduction</td>
<td>Lindsay Muir</td>
</tr>
</tbody>
</table>

### Session I: Nerve Injuries and Compression
Chair: Mark Pickford

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Speaker(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>09:00</td>
<td>Refresher on nerve anatomy and physiology</td>
<td>Andrew Hart</td>
</tr>
<tr>
<td>09:10</td>
<td>Nerve injury – pathophysiology (pre-reading)</td>
<td>Andrew Hart</td>
</tr>
<tr>
<td>09:25</td>
<td>Assessment of nerve function – after injury and after treatment: decision making and timing of surgery</td>
<td>Mikael Wiberg</td>
</tr>
<tr>
<td>09:40</td>
<td>Pathology of nerve compression</td>
<td>Andrew Hart</td>
</tr>
<tr>
<td>09:55</td>
<td>Neurophysiology made simple</td>
<td>Joe Dias</td>
</tr>
<tr>
<td>10:10</td>
<td>Compression neuropathy</td>
<td>Susan Mackinnon</td>
</tr>
</tbody>
</table>

- Introduction to compression neuropathy
- Ulnar nerve compression neuropathy; median nerve compression neuropathy
- Questions and discussion

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Speaker(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>11:00</td>
<td>Refreshments and exhibition</td>
<td></td>
</tr>
</tbody>
</table>

### Session II: Nerve Injuries and Compression
Chair: Maurizio Calcagni

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Speaker(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>11:30</td>
<td>Other compressive neuropathies – AIM, PIN, Guyon’s canal</td>
<td>Mark Pickford and Fortune Iwuagwu</td>
</tr>
<tr>
<td>11:45</td>
<td>Clinical cases discussion with Susan Mackinnon</td>
<td>Susan Mackinnon</td>
</tr>
</tbody>
</table>

- Strategies for peripheral nerve surgery
- Nerve transfers that work
- The future in nerve repair and regeneration | Dominic Power                 |

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Speaker(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>13:00</td>
<td>Lunch and exhibition</td>
<td></td>
</tr>
</tbody>
</table>

### Session III: Neurology
Chair: Grainne Bourke

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Speaker(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>14:00</td>
<td>Nerve transfers for ulnar nerve (supercharge)</td>
<td>Susan Mackinnon</td>
</tr>
<tr>
<td>14:00</td>
<td>Nerve transfers for radial nerve; nerve transfers for median nerve</td>
<td>Susan Mackinnon</td>
</tr>
<tr>
<td></td>
<td>Miscellany</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Participant and faculty cases (if time permits)</td>
<td>Anuj Mishra</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Speaker(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>15:00</td>
<td>Refreshments and exhibition</td>
<td></td>
</tr>
</tbody>
</table>

### Session IV: Neurological miscellany
Chair: Zaf Naqui

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Speaker(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>15:25</td>
<td>Neurological disease that can mimic compression neuropathy, hereditary liability to palsy, double crush syndrome</td>
<td>David Gosal</td>
</tr>
<tr>
<td>15:40</td>
<td>Radiation plexopathy, Parsonage Turner syndrome</td>
<td>Maurizio Calcagni</td>
</tr>
<tr>
<td>15:55</td>
<td>Hand arm vibration syndrome</td>
<td>Tim Davis</td>
</tr>
<tr>
<td>16:10</td>
<td>Iatrogenous nerve lesions and neuromata: causes and management</td>
<td>Esther Vögelin</td>
</tr>
<tr>
<td>16:25</td>
<td>Therapy in nerve injury; re education</td>
<td>Sarah Turner</td>
</tr>
<tr>
<td>16:45</td>
<td>Clinical cases on neuroma with Esther Vögelin</td>
<td>Esther Vögelin</td>
</tr>
<tr>
<td>17:15</td>
<td>End of day one</td>
<td></td>
</tr>
</tbody>
</table>
### Saturday 17 June

#### Session V: Anaesthesia and CRPS

**Chair:** Lindsay Muir

<table>
<thead>
<tr>
<th>Time</th>
<th>Topic</th>
<th>Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>08:00</td>
<td>Anaesthesia evidence for preventing CRPS and anaesthesia in CRPS</td>
<td>Nicholas Fraser</td>
</tr>
<tr>
<td>08:15</td>
<td>CRPS and neurogenic pain [pre-learn Budapest criteria]</td>
<td>Richard Haigh</td>
</tr>
<tr>
<td>08:35</td>
<td>Questions and discussion</td>
<td></td>
</tr>
</tbody>
</table>

#### Session VI: Brachial plexus

**Chair:** Zaf Naqui

<table>
<thead>
<tr>
<th>Time</th>
<th>Topic</th>
<th>Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>09:00</td>
<td>Brachial plexus</td>
<td>Andrew Hart</td>
</tr>
<tr>
<td>09:25</td>
<td>Brachial plexus: Mechanism of injury, classification, relation to prognosis</td>
<td>Grainne Bourke</td>
</tr>
<tr>
<td>09:30</td>
<td>Neurophysiology, MRI and other special investigations</td>
<td>Maurizio Calcagni</td>
</tr>
<tr>
<td>09:45</td>
<td>Brachial plexus: clinical examination – how I do it; including examination of muscle groups</td>
<td></td>
</tr>
<tr>
<td>10:45</td>
<td>Refreshments and exhibition</td>
<td></td>
</tr>
</tbody>
</table>

#### Session VII: Brachial plexus

**Chair:** Fortune Iwuagwu

<table>
<thead>
<tr>
<th>Time</th>
<th>Topic</th>
<th>Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>11:15</td>
<td>The acute injury: priorities, options, timing of surgery, realistic goals</td>
<td>Andrew Hart</td>
</tr>
<tr>
<td>11:30</td>
<td>Nerve reconstruction of C5/C6 and partial plexus injuries</td>
<td>Dominic Power</td>
</tr>
<tr>
<td>11:45</td>
<td>Later reconstruction of adult pan-plexus injury – microsurgical, tendon transfer, fusions</td>
<td>Andrew Hart</td>
</tr>
<tr>
<td>12:05</td>
<td>Does thoracic outlet system exist? Clinical findings, treatment and outcome</td>
<td>Colin Chan</td>
</tr>
<tr>
<td>12:20</td>
<td>Clinical cases</td>
<td></td>
</tr>
<tr>
<td>12:45</td>
<td>Hand diploma and course preview</td>
<td>Lindsay Muir</td>
</tr>
<tr>
<td>12:50</td>
<td>Lunch and exhibition</td>
<td></td>
</tr>
</tbody>
</table>

#### Session VIII: Obstetric brachial plexus

**Chair:** Mr Lindsay Muir

<table>
<thead>
<tr>
<th>Time</th>
<th>Topic</th>
<th>Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>13:45</td>
<td>Obstetrical BPP: cause, natural history, assessment and early management</td>
<td>Gürsel Leblebicioğlu</td>
</tr>
<tr>
<td>14:15</td>
<td>Surgical treatment of shoulder abnormalities in OBPP</td>
<td>Grainne Bourke</td>
</tr>
<tr>
<td>14:30</td>
<td>Questions and discussion</td>
<td></td>
</tr>
</tbody>
</table>

#### Session IX: Fractures and nerve tumours

**Chair:** Alexandra Hazelrigg

<table>
<thead>
<tr>
<th>Time</th>
<th>Topic</th>
<th>Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>14:45</td>
<td>What do I do with a radial nerve palsy and a humeral shaft fracture</td>
<td>Esther Vögelin</td>
</tr>
<tr>
<td>15:00</td>
<td>Nerve tumours: pathology and management</td>
<td>Chris Duff</td>
</tr>
<tr>
<td>15:20</td>
<td>Questions and discussion</td>
<td></td>
</tr>
<tr>
<td>15:40</td>
<td>Closing remarks</td>
<td>Lindsay Muir</td>
</tr>
<tr>
<td>15:45</td>
<td>End of session</td>
<td></td>
</tr>
</tbody>
</table>

**Continued Medical Development:** Friday - 6.5 points; Saturday - 6.0 points; **Total** – 12.5 points
British Society for Surgery of the Hand

Instructional Courses

Series Seven 2017 – 2019

Meeting 7.2

Nerve Injury & Compression, Pain, Anaesthesia

[12.5 CME points]

Manchester Conference Centre

16 & 17 June 2017

International Faculty

Dr Maurizio Calcagni
Professor Gürsel Leblebicioğlu
Dr Susan McKinnon
Professor Esther Vögelin
Professor Mikael Wiberg

Zürich, Switzerland
Ankara, Turkey
St Louis, USA
Bern Switzerland
Umeå, Sweden

National Faculty

Miss Grainne Bourke
Mr Colin Chan
Professor Tim Davis
Professor Joe Dias
Mr Chris Duff
Dr Nicholas Fraser
Dr David Gosal
Dr Richard Haigh
Professor Andrew Hart
Mr Fortune Iwuagwu
Mr Anuj Mishra
Mr Lindsay Muir
Mr Mark Pickford
Mr Dominic Power
Mrs Sarah Turner

Leeds
Wirral
Nottingham
Leicester
Manchester
Stockport
Salford
Exeter
Glasgow
London
Liverpool
Manchester
East Grinstead
Birmingham
Manchester
British Society for Surgery of the Hand  
Instructional Courses Series Seven 2017 – 2019 
Meeting 7.2: Nerve Injury Decompression, Pain, Anaesthesia 
Manchester Conference Centre 
16 & 17 June 2017 

**Delegates**

<table>
<thead>
<tr>
<th>Name</th>
<th>Hospital</th>
<th>Town</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mrs Anne Alexander</td>
<td>OUH NHS Trust</td>
<td>Oxford</td>
</tr>
<tr>
<td>Miss Riffat Aslam</td>
<td>Northern General Hospital</td>
<td>Sheffield</td>
</tr>
<tr>
<td>Mr James Bedford</td>
<td>Leeds General Infirmary</td>
<td>Leeds</td>
</tr>
<tr>
<td>Miss Ruminia Begum</td>
<td>Norfolk and norwich</td>
<td>Norwich</td>
</tr>
<tr>
<td>Mr Onur Berber</td>
<td>Oxford University Hospital</td>
<td>Oxford</td>
</tr>
<tr>
<td>Miss Anna Berridge</td>
<td>Queens Hospital</td>
<td>London</td>
</tr>
<tr>
<td>MD Tadeusz Bilnicki</td>
<td>Zespol Szpitali Miejskich w Chorzowai</td>
<td>Chorzów</td>
</tr>
<tr>
<td>Dr Ricard Blomstedt</td>
<td>Måtarsjukhuset Ortopediska kliniken</td>
<td>Eskilstuna</td>
</tr>
<tr>
<td>Dr Karen Brandt Rosing</td>
<td>University Hospital Aalborg</td>
<td>Aalborg</td>
</tr>
<tr>
<td>Miss Anca Breahna</td>
<td>Pulvertaft Hand Centre</td>
<td>Derby</td>
</tr>
<tr>
<td>Ms Katherine Browne</td>
<td>St James’s Hospital</td>
<td>Dublin</td>
</tr>
<tr>
<td>Dr Paul Ceuterick</td>
<td>Clinique Europe - site St Elisabeth</td>
<td>Brussels</td>
</tr>
<tr>
<td>Mr Kaushik H Chakrabarty</td>
<td>University Hospital South Manchester</td>
<td>Manchester</td>
</tr>
<tr>
<td>Mr Martin Coady</td>
<td>Tees Valley Treatment Centre</td>
<td>Middlesbrough</td>
</tr>
<tr>
<td>Mr Chris Coapes</td>
<td>The James Cook University Hospital</td>
<td>Middlesbrough</td>
</tr>
<tr>
<td>Mr Michael David</td>
<td>The Royal Orthopaedic Hospital</td>
<td>Birmingham</td>
</tr>
<tr>
<td>Dr Claire Cheeran Davidson</td>
<td>Mater Hospital</td>
<td>Dublin</td>
</tr>
<tr>
<td>Dr Pawel Dec</td>
<td>Pomeranian Medical University / Department of Hand Surgery</td>
<td>Szczecin, Poland</td>
</tr>
<tr>
<td>Dr Ger Duff</td>
<td>Uniervisty Hospital Galway</td>
<td>Galway</td>
</tr>
<tr>
<td>Dr Jamila Eriksen</td>
<td>Nordsjællands Hospital</td>
<td>Hillerød</td>
</tr>
<tr>
<td>Miss Nicola Fine</td>
<td>Royal Devon and exeter</td>
<td>Exeter</td>
</tr>
<tr>
<td>Mr Simon Fleming</td>
<td>Great Ormond Street Hospital</td>
<td>London</td>
</tr>
<tr>
<td>Mr Andras Gal</td>
<td>Southend University Hospital</td>
<td>Southend-on-Sea</td>
</tr>
<tr>
<td>Mr Michael R Gale</td>
<td>Pulvertaft Hand Centre</td>
<td>Derby</td>
</tr>
<tr>
<td>Mr Sam Gidwani</td>
<td>Guy’s &amp; St Thomas’ Hospitals</td>
<td>London</td>
</tr>
<tr>
<td>Miss Aurora Gonzalez</td>
<td>Luton &amp; Dunstable NHS Trust</td>
<td>Luton</td>
</tr>
<tr>
<td>Mrs Jennifer Greenhowe</td>
<td>Ninewells Hospital</td>
<td>Dundee</td>
</tr>
<tr>
<td>Dr Xavier Gueffier</td>
<td>Clinique Saint Vincent de Paul</td>
<td>Bourgoin jallieu</td>
</tr>
<tr>
<td>Mr Simon J Harrison</td>
<td>Harrogate District Hospital</td>
<td>Harrogate</td>
</tr>
<tr>
<td>MD Martin Bille Henriksen</td>
<td>Aalborg Universitetshospital</td>
<td>Aalborg</td>
</tr>
<tr>
<td>Miss Catherine Hernon</td>
<td>Leeds Teaching Hospitals NHS Trust</td>
<td>Leeds</td>
</tr>
<tr>
<td>Miss Louise Heylen</td>
<td>Addenbrooke’s Hospital</td>
<td>Cambridge</td>
</tr>
<tr>
<td>Mr Anthony J Heywood</td>
<td>Stoke Mandeville</td>
<td>Stoke Mandeville</td>
</tr>
<tr>
<td>Dr Øyvind Håberg</td>
<td></td>
<td>Kristiansund, Norway</td>
</tr>
<tr>
<td>Mr Edward Jeans</td>
<td>Royal Victoria Infirmary</td>
<td>Newcastle</td>
</tr>
<tr>
<td>Mr Prash Jesudason</td>
<td>Ysbyty Gwynedd</td>
<td>Bangor</td>
</tr>
<tr>
<td>Mrs Riem Johnson</td>
<td>Chelsea and Westminster</td>
<td>London</td>
</tr>
<tr>
<td>Name</td>
<td>Hospital</td>
<td>Town</td>
</tr>
<tr>
<td>------------</td>
<td>---------------------------------------------------------------------------</td>
<td>-----------------------------</td>
</tr>
<tr>
<td>Mrs Sigridur Karlsdottir</td>
<td>Haukeland University Hospital</td>
<td>Bergen, Norway</td>
</tr>
<tr>
<td>Dr Anne marie Kennedy</td>
<td>Mater Misericordiae hospital</td>
<td>Dublin</td>
</tr>
<tr>
<td>Mr Preetham Kodumuri</td>
<td>Nottingham University Hospitals</td>
<td>Nottingham</td>
</tr>
<tr>
<td>Mr Ioannis Konstantinidis</td>
<td>Måalarsjukhuset</td>
<td>Eskilstuna, Sweden</td>
</tr>
<tr>
<td>Mr Michael Lamyman</td>
<td>Oxford University Hospitals NHS Foundation Trust</td>
<td>Oxford</td>
</tr>
<tr>
<td>Doctor Morten Bo Larsen</td>
<td>Hvidovre Hospital</td>
<td>Copenhagen</td>
</tr>
<tr>
<td>Miss Tamsin Lees</td>
<td>St Mary’s Hospital</td>
<td>London</td>
</tr>
<tr>
<td>Dr Anna Luccardi</td>
<td>Royal London</td>
<td>London</td>
</tr>
<tr>
<td>Mr John Mcfarlane</td>
<td>RJAH</td>
<td>Oswestry</td>
</tr>
<tr>
<td>Mrs Mary-Clare Miller</td>
<td>Queen Victoria Hospital</td>
<td>East Grinstead</td>
</tr>
<tr>
<td>Mr Rory J Norris</td>
<td>University Hospital Coventry and Warwickshire</td>
<td>Coventry</td>
</tr>
<tr>
<td>Mrs Suzanne Oxley</td>
<td>Leeds Teaching Hospitals</td>
<td>Leeds</td>
</tr>
<tr>
<td>Mr Vishal Palial</td>
<td>Lincoln County Hospital</td>
<td>Lincoln</td>
</tr>
<tr>
<td>Mr Oscar Peñuela</td>
<td>Hospital de Mataro</td>
<td>Barcelona</td>
</tr>
<tr>
<td>Miss Victoria Rainey</td>
<td>Chelsea and Westminster Hospital</td>
<td>London</td>
</tr>
<tr>
<td>Dr Monica Ramirez</td>
<td>Heinrich Heine Universitat. Unfall und Handchirurgie</td>
<td>Düsseldorf</td>
</tr>
<tr>
<td>Mr Eric Raven</td>
<td>Gelre Hospital</td>
<td>Apeldoorn</td>
</tr>
<tr>
<td>MD Karolina Rosolowicz</td>
<td>Department of Pediatric Surgery and Oncology PUM</td>
<td>Szczecin</td>
</tr>
<tr>
<td>Mrs Camilla Ryge</td>
<td>Gentofte hospital</td>
<td>Copenhagen - Denmark</td>
</tr>
<tr>
<td>Miss Soha Sajid</td>
<td>Robert Jones and Agnes Hunt Orthopaedic Hospital</td>
<td>Oswestry</td>
</tr>
<tr>
<td>Dr Mette Schandorff Skjaerbaek</td>
<td>Hospitalsenhed midt Regionshospitalet Viborg</td>
<td>Viborg</td>
</tr>
<tr>
<td>Dr Lars Solgaard</td>
<td>Gentofte Hospital</td>
<td>Copenhagen</td>
</tr>
<tr>
<td>Miss Lynette Spalding</td>
<td>Addenbrooke’s Hospital</td>
<td>Cambridge</td>
</tr>
<tr>
<td>Mr Kian Tan</td>
<td>University Hospital of South Manchester NHS Foundation Trust</td>
<td>Manchester</td>
</tr>
<tr>
<td>Miss Sarah Taplin</td>
<td>Leeds teaching hospitals NHS trust</td>
<td>Leeds</td>
</tr>
<tr>
<td>Mr Daniel J A Thornton</td>
<td>Leeds General Infirmary</td>
<td>Leeds</td>
</tr>
<tr>
<td>Miss Vi Vien Toh</td>
<td>Sheffield Teaching Hospitals</td>
<td>Sheffield</td>
</tr>
<tr>
<td>Mr Alexander Vaughan</td>
<td>Royal Sussex County Hospital</td>
<td>Brighton</td>
</tr>
<tr>
<td>Dr Pernille Vedel</td>
<td>Bispebjerg Hospital</td>
<td>Copenhagen</td>
</tr>
<tr>
<td>Miss Danielle Wharton</td>
<td>University Hospital of South Manchester (Wythenshawel)</td>
<td>Manchester</td>
</tr>
<tr>
<td>Miss Paulina Witt</td>
<td>Wythenshawe &amp; Salford Royal Hospitals</td>
<td>Manchester</td>
</tr>
<tr>
<td>Mr Fizan Younis</td>
<td>Royal Blackburn Hospital</td>
<td>Blackburn</td>
</tr>
<tr>
<td>Mr stuart mckirdy</td>
<td>Lancashire Teaching Hospitals</td>
<td>Preston</td>
</tr>
<tr>
<td>Mr Arunas Šliužas</td>
<td>Vejle Hospital</td>
<td>Vejle</td>
</tr>
</tbody>
</table>
British Society for Surgery of the Hand
Instructional Courses Series Seven 2017 – 2019
Meeting 7.2: Nerve Injury & Compression, Pain, Anaesthesia
Manchester Conference Centre
16 & 17 June 2017

The British Society for Surgery of the Hand would like to thank the following companies for supporting the Instructional Courses in Hand Surgery:

**Trade Exhibitors**

**ARTHREX LTD**
Unit 5, 3 Smithy Wood Drive, Smithy Wood Business Park, Sheffield, S35 1QN
Telephone: 0114 232 9180, E-mail: info@arthrex.co.uk

**BOLTON SURGICAL LTD**
Churchill House, 16 Churchill Way, Chapeltown, Sheffield, S35 2PY
Telephone: 0114 240 4400, Email: sales@boltons.co.uk

**HOSPITAL INNOVATIONS**
Concept House, Talbot Green Business Park, Pontyclun, CF72 9FG
Telephone: 01443 719 555, Email: info@hospitalinnovations.co.uk
JEWEL MANAGEMENT LTD
Crown House, Waller Road, Devizes, Wiltshire, SN10 2GH
Telephone: 01380 734 990, Email: info@generalmedical.co.uk

MEDARTIS LTD
17A St Christopher Way, Pride Park, Derby DE24 8JY
Telephone: 01924 476 699, Email: mai.widdowson@medartis.com

STRYKER UK LTD
Stryker House, Hambridge Road, Newbury, Berkshire, RG14 5AW
Telephone: 01635 262400 Email: enquiries@stryker.com
Dr Maurizio Calcagni

Plastic and Hand Surgeon, University Hospital Zurich, Switzerland

Short CV of PD Dr. med. Maurizio Calcagni

12.03.1965 Born in Turin [Italy]
1983 – 1989 Medical School at the University of Torino [Italy]
1990-1991 Military Service, Lieutenant of the Sanitary Corp, Turin [Italy]
1994 Specialisation Plastic, Reconstructive and Aesthetic Surgery, University of Turin [Italy], Swiss Recognition as FMH Plastic, Reconstructive and Aesthetic Surgery 2004
1992-1995 Resident Div. of Plastic Surgery and Burn Centre, Orthopedic and Trauma Centre, Turin [Italy]
1996 Resident Institut Français de la Main, Paris [France]
1997-1999 Staff Surgeon Div. of Plastic Surgery and Burn Centre, Orthopedic and Trauma Centre, Turin [Italy]
1990-2000 Visiting Fellowships in Europa and in USA (Innsbruck [AT), Glasgow [GB), New York [USA), Paris [F), Bordeaux [F), Zürich [CH])
1999-2001 Senior Surgeon, Div. of Hand Surgery, Chair of Plastic Surgery, University of Milan [Italy]
2001-2004 Vice-Chairman, Div. of Hand Surgery, Chair of Plastic Surgery, University of Milan [Italy]
2002-2004  Lecturer in Hand Surgery, School of Plastic, Reconstructive and Aesthetic Surgery, University of Milan (Italy)
2004-2007  Vice-Chairman, Dept. of Surgery, head of hand Surgery, Regional Hospital Winterthur (CH)
2004-2009  Examiner of the European Board Examination of the Federation of Societies for Surgery of the Hand
Since 2007  Vice-Chairman, Div. Plastic Surgery and hand Surgery, Head of Hand Surgery and Head of Research Unit, University Hospital Zürich (CH)
2007-2012  Lecturer in Plastic and Hand Surgery, Medical School, University of Zurich (CH)
2007      Specialised in Hand Surgery, Switzerland (FMH Handchirurgie)
2012-2014  Chairman Skin Coverage Committee, International Federation of Societies Surgery of the Hand
2013      Venia Legendi (Privat Dozent) in plastic surgery and hand surgery, University of Zurich
Since 2010  Chairman of European Examination in Hand Surgery of the Federation of European Societies for Hand Surgery
Since 2010  Member of the Board of the Federation of European Societies of Hand Surgery
Short Biography

**DOB:** 20-12-1963  
**Medical School:** 1980-1896 University of Istanbul Cerrahpaşa Medical School, İstanbul, Turkey.  
**Fellowship:** 1987-1992 University of Hacettepe Medical School, Department of Orthopaedic Surgery and Traumatology, Ankara, Turkey  
**Academic Position:** 1995-2017 University of Hacettepe Medical School, Department of Orthopaedic Surgery and Traumatology, Ankara, Turkey  

**Current Tasks**  
- Council Member: Federation of European Societies for Surgery of the Hand, Chair of the JHS-E Committee  
- Member of the Management Committee, J. Hand Surg European  
- Member of the FESSH European Board of Hand Surgery Examination Committee  

**Areas of interest in hand surgery**  
- Obstetrical brachial plexus surgery  
- Musicians hand problems  
- Endoscopic surgery, wrist arthroscopy  
- Kienböck’s disease  
- Limb salvage oncologic surgery
Senior Consultant Hand, Plastic and Reconstructive Surgeon
Head, Section for Hand and Plastic Surgery, Umeå University, Sweden
Director of Research and Education, University Hospital of Northen Sweden and County Council of Västerbotten

Member of several national and national boards and committees for example Board Member of The Swedish National Board of Health and Welfare, Board Member of The Swedish National Board of Donation and Transplantation
Published around 150 scientific publications, review articled and bookchapters – mainly within the field of nerve biology and reconstruction.
Dr. Mackinnon graduated from medical school at Queen’s University in Kingston, Ontario, Canada, in 1975. She trained in general surgery at the same institution and completed her plastic surgery training at the University of Toronto. She then completed a year of peripheral nerve research at the University of Toronto in the Division of Neurosurgery and a hand surgery fellowship in Baltimore at the Raymond Curtis Hand Centre. Dr. Mackinnon joined the surgical staff at the University of Toronto in 1982. In 1988, she was awarded the Medal Prize in Surgery from the Royal College of Physicians and Surgeons for her work on nerve regeneration.

In 1991, Dr. Mackinnon joined the Department of Surgery at the Washington University School of Medicine. Her research work in St. Louis has been funded through the National Institute of Health since 1993 and has investigated nerve allotransplantation and nerve regeneration. This work has resulted in new strategies of nerve transfers to reconstruct nerve injuries. She has been Chief of Plastic Surgery since 1996.

She has published a classic textbook, Surgery of the Peripheral Nerve, 493 peer reviewed publications, 171 book chapters and the new comprehensive text Nerve Surgery. Her open access website provides surgical videos with all the “secret ingredients” and are used globally to improve the care of nerve injured patients. Dr. Mackinnon is Past President of the
American Society for Peripheral Nerve, American Association of Hand Surgery, American Association of Plastic Surgery, and Plastic Surgery Research Council and is a member of the National Academies of Medicine. She is the recipient of the Jacobson Innovation Award from the American College of Surgeons in 2013 for her pioneering work on nerve transfers.

Dr. Mackinnon married Dr. Alec Patterson in 1972 and has four talented children. Lachlan, a James Beard award winning Chef, Megan, an Orthopaedic hand surgeon, Brendan is an Orthopaedic resident and Caitlan, a hospital administrator. She has ten adorable grandchildren.
<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>YEAR(s)</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>University of Basel, Switzerland</td>
<td>MD</td>
<td>1986</td>
<td>Medicine</td>
</tr>
<tr>
<td>University of Basel, Switzerland</td>
<td>FMH (Diploma)</td>
<td>1986</td>
<td>Thesis in Medicine</td>
</tr>
<tr>
<td>University of Basel, Switzerland</td>
<td>FMH (Diploma)</td>
<td>1994</td>
<td>Plastic and Reconstructive Surgery</td>
</tr>
<tr>
<td>University of Bern, Switzerland</td>
<td>PhD</td>
<td>2001</td>
<td>Hand Surgery</td>
</tr>
<tr>
<td>University of Bern, Switzerland</td>
<td>Associate Professor</td>
<td>2006</td>
<td>Plastic, Reconstructive and Hand Surgery</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2013</td>
<td>Hand Surgery</td>
</tr>
</tbody>
</table>
Positions and Honors

Employments:
1994-1995: Registrar in Plastic Surgery, Mount Vernon Hospital, Northwood, Middlesex, UK
1996-1997: Research Fellowship in Hand and Microsurgery, UCLA, Los Angeles, CA, USA
1998-1999: Training in Hand Surgery, University of Bern, Switzerland
1999-2002: Consultant in Hand Surgery University of Bern, Switzerland
2002-2006: Locum chief position in Hand Surgery, University of Bern, Switzerland
2007 to presence: Chief and Co-director in Hand and Plastic Surgery, Department of Orthopaedic, Plastic and Hand Surgery, Inselspital, University of Bern, Switzerland

Memberships:
Swiss Society of Hand Surgery (Board member 2007 to presence, president 2010)
Swiss Society of Aesthetic, Plastic and Reconstructive Surgery
European Society of Plastic Surgery [EURAPS]
Swiss Society of Ultrasound Medicine (SGUM)
Trauma Committee FESSH [Federation of European Societies for Surgery of the Hand] 2005 to presence
Swiss Delegate of IFSSH [International Federation of Societies for Surgery of the Hand] 2007 to presence

Associate editor of the European Journal of Surgery of the Hand: 2006 to presence

Awards:
- Resident Research Conference 1997, UCLA, Los Angeles, CA, June 1997
  Vascularized Knee joint allografts immunosuppressed with FK-506, RS-61443 and SDZ-RAD
- 9th Annual Meeting of the European Association of Plastic Surgeons (EURAPS), Verona, Italy, May 28-30, 1998 Best Paper Award of the Research category


- Best paper award, 16th Annual Meeting of the European Association of Plastic Surgeons (EURAPS), Marseille, May 26-28, 2005 The Subcostal Perforator Flap – An Anatomical Study
  M.A. Constantinescu, D. Feinendegen, T. Niederhäuser, E. Vögelin, A. Banic.


- 49. SGH Kongress, Fribourg, 5.-6.11.15: Greatest Future Clinical Application Award: Regional Immunosuppression with Sirolimus Encapsulated In Situ Forming Implants in Vascularized Composite Allograft. Jonas Schnider1, Damian Sutter1, Julie Denoyelle1, Esther Vögelin1, Jan Plock2, Jean-Christophe Leroux3, Paola Luciani2 (Bern1; Zurich3)

A. Peer-reviewed publications


2. The impact of anxiety and depressive symptoms on chronic pain in conservatively and operatively treated hand surgery patients. Egloff N,


5. [The bionic hand]. Surke C, Ducommun Dit Boudry P, Vögelin E. Ther Umsch 2015 Aug 72(8);487-93


42. [Hand transplantation - fiction or reality?]. Vögelin E. Ther Umsch. 2011


Publications available with this link:
http://www.ub.unibe.ch/content/forschen__publizieren/boris_repository/index_ger.html
University of Bern: https://www.boris.unibe.ch/

Bookchapters and other articles


E.Vögelin. Soft tissue defects around the wrist. In Primary Care of Complex Injuries of the Hand & Wrist: Current Concepts. FESSH Instructional Course


E. Vögelin, L. Haug. Thumb Replantation in Microsurgery. FESSH 2016 Instructional Course. B.Battiston
Dr. Susan Mackinnon

SESSION I
10:10-11:00 (50 Minutes)
PREZI 1 - Ulnar Nerve Compression Neuropathy (20 minutes)
https://prezi.com/uml-qda71htv/
PREZI 2 - Median Nerve Compression Neuropathy (20 minutes)
https://prezi.com/ttidsf08qkqs/
PREZI 3 – Surgical Cases (10 Minutes)
https://prezi.com/q8kofmfe8yq1/

SESSION II
11:45-12:45 (1 Hour)
PREZI 1 - Strategies for Peripheral Nerve Surgery (30 minutes)
https://prezi.com/qirrtisfxp/>
PREZI 2 - Nerve Transfers that Work (30 minutes)
https://prezi.com/wglag_s49v73/

SESSION III
14:00-15:00 (1 Hour)
PREZI 1 - Nerve Transfers for Ulnar Nerve [Supercharge] - (20 minutes)
https://prezi.com/15f-kr4qmfjb/
PREZI 2 - Nerve Transfers for Radial Nerve - (20 minutes)
https://prezi.com/tvasalbssnya/
PREZI 3 - Nerve Transfers for Median Nerve - (20 minutes)
https://prezi.com/sj1wpui4ih8/
Nerve injury: Clinical assessment and Timing of surgery
Mikael Wiberg

All patients with a potential nerve injury should be judged individually and all cases with any form of dysfunctional nerve after injury should be regarded to have a transected nerve until proven otherwise. Using mainly clinical assessment and in some cases additional techniques (ie electrophysiology, MRI), an early decision to explore or not, has to be made to get the best functional restitution if the nerve is transected. Both experimental and clinical data supports the opinion that the earlier a nerve injury is repaired the better to restore function, as many neuronal reactions after injury will persist until the damaged nerve ends are joined together, with or without nerve grafts. Periods longer than three months between injury and repair have been demonstrated to have devastating consequences for the final functional outcome. The factors behind neuronal reactions after injury and the importance of timing will be discussed in the lecture.
The future in nerve repair and regeneration
Dominic Power Consultant Hand and Peripheral Nerve Surgeon, Birmingham UK

The field of surgery is changing from the traditional aims of repair and microsurgical advances facilitating reconstruction to the era of stem cell research, regenerative surgery and rejuvenation. Advances in the laboratory have been slow to be translated into wider clinical practice and clinicians continue to try and refine the techniques of microsurgical repair and reconstruction when results dictate the need for a fresh approach.

Little has changed in acute nerve repair and gap reconstruction using sensory nerve autograft from the techniques described by Otfrid Foerster during the First World War although the advent of practical surgical microscopy allowed Millesi and others to refine the surgical techniques. The development of conduits and nerve allografts in nerve gap reconstruction have been largely driven with commercial aims rather than a desire to improve on the mediocre results of autologous sensory nerve bridging of gaps in mixed nerve trunks.

The paradigm shift towards nerve transfer surgery has followed from the promising results of reconstruction of paralysis in non-reconstructable nerve root avulsion injuries in the management of the brachial plexus. Following a peripheral nerve injury, nerve transfers may be utilized for distal salvage of failed proximal reconstruction, late presentation cases, as an adjunct to nerve graft repair in mixed nerve trunks and use as a primary alternative reconstruction technique. Nerve transfer however is not new. Many of the techniques rediscovered in the last three decades were developed and practiced more than a century ago by Adolf Stoffel and others.

Improved outcomes after peripheral nerve injury will come from a number of areas including earlier and more accurate diagnosis, neuroprotective agents to prevent axonal cell population decline through apoptosis, more accurate methods of repair, the prevention of Wallerian degeneration, enhancing neurotropism and improved methods of rehabilitation including the prevention of end organ decline, non-invasive measures of axonal regeneration and harnessing the plasticity of the central nervous system.
Guyon’s canal compression (ulnar nerve at the wrist)

Guyon’s canal compression syndrome is the variable clinical spectrum of signs and symptoms that is caused by irritation or compression of the ulnar nerve at the wrist. It is named after French Urologist – Guyon (1861).

The canal is approximately 4 cm long and extends from the proximal extent of transverse carpal ligament to the aponeurotic arch of hypothenar muscles. The depression between pisiform (medially) and hook of hamate (laterally) is converted into a fibro-osseous tunnel by the pisohamate ligament. The roof of the canal is formed by the volar carpal ligament and pisohamate ligament and the floor is formed by the posterior leaf of the transverse carpal ligament.

The contents include the deep branch (radial and motor), superficial branch (ulnar and sensory) of the ulnar nerve and the ulnar artery (radial).

Common causes of guyon’s canal compression include: Trauma e.g. acute or repetitive trauma, hook of the hammate fracture; Anomalous muscles; Degenerative – osteoarthritis e.g. pisotriquetral; Space occupying lesions - ganglion, tumours, aneurysm of the artery ; metabolic disorder e.g. diabetes mellitus

Clinical features include altered sensation in the little and ring fingers and later marked wasting of the small muscles of the hand. Signs of ulnar nerve palsy such as Froment’s sign, clawing etc. become demonstrable. But the signs of more proximal compression of the ulnar nerve such as loss of sensation on the ulnar dorsum of the hand and wasting of ulnar sided forearm muscles are absent.

There are three zones of compression and this determines the predominant clinical manifestation.

Zone 1 compression extends from the proximal edge of the palmar carpal ligament to the bifurcation of the ulnar nerve (sensory and motor); zone 2 compression extends from the bifurcation of the ulnar nerve just distal to the fibrous arch of the hypothenar muscles (deep motor branch) and; zone 3 compression contains the superficial sensory branch of the ulnar nerve.

Diagnosis is primarily clinical. And confirmed with neurophysiology. Other investigations needed may include specific provocative tests such as hook of the hamate pull test, x-rays, MRi etc

Treatment include patient education, splints and surgery. Indications for surgery include failed conservative treatment, severe symptoms at presentation and treatment of the cause e.g. aneurysmectomy, osteophysectomy in osteoarthritis.
References

Hand/Arm Vibration Syndrome (Vibration White Finger)

Tim Davis

Lumberjacks, grinders, car mechanics, platers, road workers and welders, and others who regularly use power hand tools at work, are at risk of developing the hand-arm vibration syndrome (HAVS). Vibration can cause a vasospastic hand condition (20%), a peripheral neuropathy (48%) or a combination of both (32%). The latency between the start of the vibration exposure and the development of symptoms can be as short as 6 months or as long as 17 years, but the symptoms must develop during, or a few months after, the period when the sufferer was using vibrating tools. Symptoms which develop more than a year after the cessation of the vibration exposure are probably due to another factor.

The vasospastic condition (vascular component of HAVS) presents as Raynaud’s phenomenon with intermittent cold and vibration-induced skin blanching of the fingers which extends proximally from their tips and has a well localised proximal border. The fingers involved should be those exposed to the most vibration and the attacks usually last for 20-60 minutes and are followed by a period of hyperaemia which is characterised by finger redness and tingling which may be very painful. In contrast the more common spontaneous Raynaud’s phenomenon usually occurs in females, is usually symmetrical and involves all the fingers. The vascular component of HAVS is usually graded according to severity by the Stockholm classification:-

<table>
<thead>
<tr>
<th>The Stockholm Classification of Hand/Arm Vibration Syndrome</th>
<th>Vascular Component</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 1</td>
<td>Skin blanching distal to the distal interphalangeal joint</td>
</tr>
<tr>
<td>Grade 2</td>
<td>Skin blanching distal to the proximal interphalangeal joint</td>
</tr>
<tr>
<td>Grade 3</td>
<td>Skin blanching distal to the metacarpophalangeal joint</td>
</tr>
<tr>
<td>Grade 4</td>
<td>Skin blanching distal to the metacarpophalangeal joint and trophic skin changes</td>
</tr>
</tbody>
</table>

Vibration can also damage peripheral nerves in the hand and cause finger paraesthesia and numbness and hand weakness and pain. The sensory symptoms are frequently caused by vibration-induced digital nerve or skin sensory receptor damage which in severe cases may cause permanent, disabling numbness and tingling. This vibration induced neuropathy (sensorineural component of HAVS) often diffusely affects the hands and frequently is not localised to the median or ulnar nerve sensory territories. It has been graded:-

<table>
<thead>
<tr>
<th>The Stockholm Classification of Hand/Arm Vibration Syndrome</th>
<th>Vibration Neuropathy Grades</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1 (1SN)</td>
<td>Intermittent numbness, with or without tingling</td>
</tr>
<tr>
<td>Stage 2 (2SN)</td>
<td>Intermittent or persistent numbness, reduced sensory perception</td>
</tr>
<tr>
<td>Stage 3 (3SN)</td>
<td>Intermittent or persistent numbness, reduced tactile discrimination and/or manipulative dexterity</td>
</tr>
</tbody>
</table>

Carpal tunnel syndrome is also found in vibration-exposed, but whether vibration causes the pathology of carpal tunnel syndrome to develop or aggravates a carpal tunnel syndrome which is developing for other reasons is uncertain.

The results of carpal tunnel decompression in carpal tunnel syndrome associated with vibration exposure are thought to be good.

There is no cure for either the vascular or the sensorineural component of HAVS and the symptoms of neither subside with the passage of time, even if further vibration exposure is avoided. However avoidance of further use of hand-held vibrating machinery will prevent worsening of both conditions and their symptoms.
Iatrogenous nerve lesions and neuromata: causes and management
Esther Vögelin

Iatrogenic nerve injuries are probably not as uncommon as one might expect. They probably account for around 20% of the traumatic nerve lesions seen at special centers. [1] They usually are the result of surgical procedures and are devastating injuries for the involved patient.

Possible causes include:

- Direct damage (sharp, traction, pressure injury) during surgical procedures
- Compression or traction injury (positioning, Tourniquet, casts, orthotic devices)
- Injection of neurotoxic substances
- Radiation

One series of 722 patients accounted 17.4% traumatic nerve lesions [1], 94% of which were associated directly with a surgical procedure. In earlier series post-operative lesions were more common. [2] Large series indicate that 25.2% of sciatic nerve lesions are secondary to medical interventions, 60% of femoral nerve lesions and 94% of accessory nerve lesions are iatrogenic. [2] In more recent study by Khan and Birch, 47.5% of 612 patients with iatrogenic nerve injuries respectively 57.6% of 26 patients following shoulder surgery failed to improve spontaneously and required surgery. [3, 4]

During surgery nerves can be cut, crushed, tied off, penetrated and twisted by screws, or traumatized and stretched during osteosynthesis or removal of devices. There is even literature on the fact that nerves are mistaken for a tendon or a vessel and therefore cut. [2] Finally, they can be removed accidentally along with a nerve sheath tumor or a lymphnode. The latter is the most common cause for damage to the accessory nerve during neck dissections involving the posterior triangle. Not recognized nerve sheath tumors are an additional cause for iatrogenous nerve injuries despite the wide-spread availability of magnetic resonance imaging and neurosonographic studies. With appropriate surgical technique, they can be removed without causing a functionally relevant neurological deficit. [5] In a recent series of 340 patients [2] with iatrogenic nerve lesions (operated between 1990-2012) the following causes were identified:

- 45% major procedures (trauma, abdominal surgery, orthopaedic procedures)
- 27% minor procedures (lymph node biopsies)
- 15% neurosurgical procedures (endoscopic, open carpal tunnel release)
- 4% non-surgical causes (casts, a.v. punctures, positioning)
- 9% no attributable cause

In this series, the median nerve was most commonly affected (17%), followed by the accessory nerve (16%), the radial and common peroneal nerve (13%), the ulnar nerve (8.5%) and the femoral nerve (5%).

Diagnosis
If a previously asymptomatic patient develops a neurological deficit following a medical intervention, generally after surgery, then this deficit is usually the result of
the intervention. Patients may complain of a sharp pain “like an electrical shock” or a deficit in sensory and or motor function.

- What type of injury has occurred (transection, pressure, stretching)?
- Where is the site of lesion (sensory, motor or mixed deficit?)
- How old is the patient and is neurogenic pain a major complain?

Electrophysiological examination to precisely identify the level of lesion in combination with modern imaging procedures such as Magnetic Resonance Imaging [MRI], MR Neurography [6 ] and or sonography [7] is recommended to plan conservative or surgical therapy. Surgical interventions such as neurolysis, nerve repair or reconstruction should be performed within 6 months, because nerve recovery is usually significantly reduced afterwards. [8] Unfortunately, in those cases diagnosis and therapy is delayed due to the fact, that nerve damage is identified but one hopes for spontaneous improvement or one fails to acknowledge the nerve damage. More complex is the situation with the main symptom of neuropathic pain rather than sensorimotor deficits. In those patients, it is crucial to evaluate whether the pain driver lies in the dorsal root ganglion or in the central nervous system rather than in the peripheral site of nerve injury, probably surgical treatment of a neuropathy is less promising.

**Therapy**

If it is noted during surgery that a nerve has been severed, it should be repaired or reconstructed immediately during the same surgery (primary repair) or within 2-3 weeks (early secondary repair). If there are clinically no signs (no or no progressive Tinel sign) of nerve regeneration after 3 months, electrophysiological and or nerve imaging [MR Neurography in proximal or mixed sensorimotor nerves and or sonography [6,7]] is repeated. In case of neuroma in continuity or severe scarring, secondary exploration is recommended.

In young patients, a severed nerve should be reconstructed even with a long nerve graft. Intraoperative nerve conduction studies may help, to assess how functional a nerve may be. If there is no conductivity after > 3 months, a nerve with scarred or thickened consistency, the nerve needs to be replaced by an auto- or allograft. In elderly patients with motor nerve lesions, a simultaneous tendon transfer is performed and the nerve is only replaced if severe neurogenic pain is present. In patients with neuropathic pain the nerve lesion may be an end neuroma, a neuroma in continuity or a scar-tethered nerve. Depending on the location and type of neuroma (terminal versus in continuity) surgical techniques can be divided in these categories: resection alone (including containment) or translocation of the nerve, resection and endtoend nerve repair, resection with subsequent nerve grafting or primary repair, neurolysis and nerve wrap. [9-12] In the rare cases, when conductivity studies are promising, a neurolysis from the surrounding scarred tissue is performed.

Postamputation pain is another condition due to painful neuroma affecting for example 60% of transfemoral amputees. Preemptive coaptation and collagen nerve wrapping is reported to have better outcome than traction neurlectomy alone. [13] Targeted nerve implantation either primarily at the time of acute amputation or
secondarily for the treatment of neuroma related pain has been shown to be effective in reducing neuroma pain by providing a distal target for regenerating axons. [14] Regenerative peripheral nerve interfaces prevent neuroma formation by providing free muscle grafts as physiological targets for peripheral nerve ingrowth and offers a simple effective treatment for symptomatic neuromas. [15]

Certain groups perform intraoperative neuro-sonography to distinguish a neuroma in continuity without or with residual individual possible functional nerve fascicles. [16] A key factor in improving the prognosis is physical and hand therapy with conservative management or postoperatively until re-innervation of affected muscles and or skin has occurred. Electric stimulation is worthwhile in our opinion even its scientific evidence is controversial, the patient is involved in the therapy and may help to maintain denervated muscle structures.

**Results**

In the series of Kretschmer et al. [1], 70% of 126 patients with surgically treated iatrogenic nerve damage showed postoperative improvement after 18 months. Only one third of patients was referred within 6 months of injury. Lesions of the accessory, radial and tibial nerve had a better prognosis common peroneal nerves. The accessory nerve is an exception, in that repairs after 6 months often produce good functional results. [17] Patients with improper positioning or compression neuropathies generally do not require surgery, because the lesions seem commonly incomplete. 90% will heal over months spontaneously.
Conclusion
If a neurological deficit is noticed immediately after surgery and the involved nerve had not been explored during the procedure, secondary early re-exploration is recommended. If there are reasons to “a wait and see policy” the patient should be closely monitored with clinical, neurophysiological and/or sonographic methods. If no improvement occurs after 3 months and or imaging demonstrates neuroma in continuity or scarred tissue around the injured nerve, re-exploration and required nerve repair should be performed.

References
Question
A 27 year old female presents with the following problem after a posterior cervical lymph node biopsy. This functional deficit is due to injury of which anatomical structure? What is true with regards to reconstruction?

A) Thoracodorsal nerve  
B) Long thoracic nerve  
C) Spinal accessory nerve  
D) The prognosis after nerve reconstruction is satisfactory even after a delay of 12 months  
E) The functional deficit of this condition is usually more symptomatic than a neuroma formation of this nerve.

Correct answer: A-, B-, C+, D-, E+
Therapy Rehabilitation in Peripheral Nerve Injury
Sarah Turner

By means of a case study, this presentation will cover the main aspects of rehabilitation employed by hand therapists for patients following nerve injury. Therapy based on generic and specific post-operative goals will be discussed including splinting, mobilisation, sensory assessment and sensory re-education. The aim is to emphasize the importance of comprehensive therapy and to convey the variety of techniques employed with these patients.
Clinical Cases on Neuroma
Esther Vögelin

1. 60 year’s old lady with sciatic nerve lesion postoperatively after hip replacement
   a) Orthopedic College calls immediately the next day

   b) You get involved 3 or 6 months later, no tinel sign, no clinical sciatic nerve function
   c) When re-exploration in a young patient?

2. 59 year old Lady with devastating neuropathic pain after surgery of a radius fx.
   No improvement after conservative treatment with somatosensory re-education.
   a) Repair or reconstruction?
   b) When reconstruction: nerve allo- or autograft? Additional nerve wrap around the neurorrhapy?
   c) When additional soft tissue padding with flap?

3. 30 year old Male with Neuroma of the palmar cutaneous branch of the median nerve.
4. Patient with pain and little functional deficit of the ulnar nerve (dysaesthesias, M4 of all ulnar nerve innervated muscles).

   a) Anterior transposition? Vascularity?
   b) Protection of the nerve by wrap? Autologous or artificial

5. 25 year old patient after below knee amputation with phantom and neuropathic pain.

   a) Traction neurectomy and translocation into muscle?
   b) Centrocentral coaptation and or collagen wrap?
   c) Creating regenerative peripheral nerve interfaces with free muscle grafts?
   d) Targeted nerve implantation?

6. 27 year old patient with k-wire injury and previous attempted neurolysis of superficial radial nerve at wrist.

   a) Second neurolysis? Nerve wrap?
   b) Resection and grafting? When resulting defect <10mm → what graft?
   c) Resection and proximal translocation?
Complex Regional Pain Syndrome
Richard Haigh

Complex Regional Pain Syndrome (CRPS) is a highly disabling condition characterised by severe limb pain, reduced movement, and swelling, associated with dramatic changes in hair, nail growth, skin temperature and colour changes. Although many patients do recover rapidly, 25% of patients will have unrelenting pain for more than 1 year after the onset of symptoms. Depression, work loss and health-related quality of life is significant and is reported as worse than other chronic conditions (diabetes; chronic lung disease). Intensive, multi-disciplinary rehabilitation is required. There is no evidence for a psychological basis of this condition. Sadly, many patients experience delayed diagnosis, inappropriate immobilisation and suffer a ‘meandering pathway of care’ with multiple consultations and poor outcomes. I hope to be able to help you spot the warning signs of impending CRPS, diagnose the full blown syndrome and start your patient off on the path to recovery.
Brachial Plexus – Mechanism of injury, Classification and relation to prognosis
Gráinne Bourke, Leeds Teaching Hospitals Trust.

Brachial plexus injuries have serious physical and psychological sequelae. The highest incidence of injury is in a young working population. For example, injuries to the Brachial plexus occur in up to 5% of cases of Polytrauma and 4% of severe winter sports injuries (Kaiser R, Mencl L, Haninec P).

The injuries can be classified into Open or Closed injuries. There is a broad spectrum of injury mechanism and thus a variable prognosis and outcome but all injuries cause significant functional disability for a time and require treatment at specialist centres with rehabilitation.

Lacerations including iatrogenic injuries carry the most favourable prognosis. This is related to both the pattern for early referral due to the open injury and thus the possibility or early nerve repair or reconstruction. It is also a reflection of the localised area of trauma to the nerve and its vascular supply. It is important that cases of iatrogenic injury to encourage early referral for assessment and treatment.

Gunshot injuries to the Brachial Plexus have extensive patterns of injury. In 2004 and 2006, Kim DH, Murovic JA, Tiel RL, Kline DG reviewed 118 cases explored at surgery and produced guidelines for managing these cases.

The most serious injuries are a result of rapid high energy traction. These are usually closed injuries and the result of motor bike accidents (63.2%) Kaiser R, Waldauf P, Haninec P. These can result in either Supraclavicular injuries with nerve root avulsion or infraclavicular injuries with associated vascular injury and bony disruption to the shoulder girdle. A similar pattern of infraclavicular injury can occur in industrial work accidents.

The mechanism of injury dictates the pattern of nerve root involvement and thus the prognosis. In road traffic injuries the most common injury is Upper trunk Avulsion which occurred in 45% of the series reviewed by Kaiser et al. Total root avulsion which carries a much poorer prognosis was seen in 16% of cases. In a series of 100 cases by Dubuisson AS, Kline DG, was present in 59 cases.

Soldado F, Ghizoni MF, Bertelli J reviewed the injury mechanism of 150 cases of supraclavicular injury. In 57 cases of upper root injury the patients described the impact as direct vertical to the shoulder while cases of lower root palsy was anterior compression of the shoulder.

Prognosis for recovery is dependent on the injury pattern, timing for reconstruction and patient’s age. Pain, psychology and social support also have significant influence on the longterm function outcome.
References


Types and severity of operated supraclavicular brachial plexus injuries caused by traffic accidents.
Kaiser R1, Waldauf P, Haninec P.


Penetrating injuries due to gunshot wounds involving the brachial plexus.
Kim DH1, Murovic JA, Tiel RL, Kline DG.


Gunshot wounds involving the brachial plexus: surgical techniques and outcomes.
Kim DH1, Murovic JA, Tiel RL, Kline DG.


Injury mechanisms in supraclavicular stretch injuries of the brachial plexus.
Soldado F1, Ghizoni MF2, Bertelli J3


Injuries associated with serious brachial plexus involvement in polytrauma among patients requiring surgical repair.
Kaiser R1, Mencl L, Haninec P.

Brachial plexus injury: a survey of 100 consecutive cases from a single service.
Dubuisson AS1, Kline DG.


Fracture of the shoulder girdle in multiply injured patients - an imperative for a high level of suspicion for associated neurovascular injuries.
Krasnici S1, Schmidt J, Reimann K, Ertel W, Topp T.
Nerve reconstruction of C5/6 and partial plexus injuries
Dominic Power Consultant Hand and Peripheral Nerve Surgeon, Birmingham UK

Closed traction injury to the brachial plexus may result in combinations of nerve root avulsion, nerve rupture and lower grade continuity lesions. The partial plexus injury is defined by complete loss of some nerve root function with integrity or early spontaneous recovery of others. Typically the upper plexus injury involving C5/6 dysfunction with integrity of the lower nerve roots has great potential for reconstruction due to the proximity of the target muscles of shoulder and elbow to the site of injury. Reconstruction can be affected through nerve grafting of ruptured roots or through the use of combinations of intraplexal and extraplexal motor nerve transfers and selective fascicle transfer from the intact lower plexus in cases of root avulsion injury, failed recovery after nerve grafting or as a primary procedure in late presenting cases where the time-distance phenomenon renders recovery through a graft unpredictable.

Nerve transfers are performed close to target muscles without grafts and reinnervation distances are short. The use of a primarily motor donor to a motor target eliminates the axonal misdirection that is an inevitable consequence of mixed nerve grafting and facilitates rehabilitation without the risks of co-contraction. Surgery is performed peripheral to the lesion and is through a bed without scar.

Reconstruction of partial lower plexus injuries is more challenging because the denervated target muscles are more distally placed in the limb and the potential donors too proximal to allow direct nerve co-aptation and rapid reinnervation. Prompt recognition of these lower plexus injuries and early nerve transfer as a primary procedure without delays from proximal nerve grafts can still produce useful hand function but the hand will still be intrinsically deficient and therefore dextrous function is not currently achievable.

Following a C5/6 injury reconstruction involves a quadruple transfer with a medial XI branch transfer to the suprascapular nerve through a posterior approach, triceps branch transfer to the axillary nerve and either a single or double nerve transfer for elbow flexion using ulnar and median fascicles to transfer to the motor branches to biceps and brachialis.

In a lower plexus injury the supinator to posterior interosseus nerve transfer provides finger and thumb extension whilst transfer of brachialis nerve motor fascicles to the anterior interosseus nerve can restore finger and thumb flexion.
Thoracic Outlet Syndrome: Does it exist? Clinical Features, Investigation & Management
Mr C W M Chan, Consultant Vascular Surgeon.
Wirral University Teaching Hospital, and South Mersey Arterial Network

Thoracic Outlet Syndrome (TOS) refers to neurovascular compressive symptoms arising in the superior thoracic outlet. The pathology arises from compression or distraction of the brachial plexus (nTOS) and/or subclavian artery (aTOS) and/or vein (vTOS) giving rise to symptoms of plexopathy (upper, lower or mixed), ischaemia or aneurysmal swelling, or venous obstruction.

The clinic-pathological anatomy can be subdivided into 3 distinct syndromes: scalenius anterior (anticus) syndrome, costoclavicular syndrome and pectoralis minor syndrome. The scalenius anterior syndrome arises from neurovascular compression by the first rib, scalenius anterior and scalenius medius muscles. Anatomical aberration (e.g. interdigitating muscle heads) or pathological change (rib fractures, scar tissue) are common findings. The presence of scalenius minimus muscle can compress the brachial plexus too. Cervical ribs are present in <3% of most case series and can be non-ossified or associated with other musculo-tendinous abnormalities.

The costoclavicular syndrome arises between the first rib, clavicle and the insertion of scalenius anterior. The pincer effect produced by these structures and aggravated by the costo-clavicular ligament and subclavius muscle place the subclavian vein at risk. The pectoralis minor muscle insertion can sometimes compress the axillary vessels giving rise to that syndrome. This is sometimes seen in sportsmen (e.g. baseball players) or body builders.

Diagnosis is clinical and the absence of radiological findings does not exclude TOS. Useful investigations to exclude other conditions and to aid treatment planning include plain radiographs, Duplex ultrasound and CT angiography in neutral and stressed positions. MRI and nerve conduction studies may be required to exclude other nerve pathology. Extended arm stretch testing is a useful provocative test. Objective measures of severity using the DASH score or SVS reporting standard may also be useful to document progress.

Surgical treatment is indicated when symptoms are moderate-severe. This involves creating sufficient space for the neurovascular structures by removing the osseous and muscular boundaries. SuprACLavicular rib resection with anterior scalenectomy and neurolysis is the commonest procedure. Transaxillary resection is helpful in removing the anterior portion of the rib in the costo-clavicular syndrome. Infraclavicular exposure is helpful in reconstructing the subclavian vein. Thoracoscopic resection is not routinely performed although this may play a role in
revisional cases. A thoracoscopic sympathectomy may be considered if there is evidence of sympathetic nerve involvement.

Results are dependent on case selection but success rate ranges from 66-88%. Complications are usually minor or transient and include intercostobrachial neurapraxia, winging of the scapula or the limited requirement for intercostal chest drainage.

Physiotherapy does not cure TOS but has an important adjunctive role to play in the control of symptoms and post-operative rehabilitation. Late interventions when the brachial plexus is atrophied and/or neuritic has a limited prognosis.

References:


Test question:

Which of the following are false in relation to thoracic outlet syndrome?

a. Symptoms attributable to C8-T1 are pathognomonic of the syndrome.
b. The absence of radiological findings in a stressed position rules out TOS.
c. Scalenius anticus syndrome is always due to the scalenius anterior and/or first rib.
d. TOS can manifest itself years after fractures of the clavicle or first rib.
e. Physiotherapy cannot cure thoracic outlet syndrome.
Obstetrical Brachial Plexus Injury

Gürsel Leblebicioğlu

Key points

- The force generated on the brachial plexus during birth with cephalic vertex presentation can cause extraforaminal ruptures of the C6 and C7 roots, and preganglionic injuries of the C8 and T1 roots.
- The most proximal motor branches of the brachial plexus are the dorsal scapular nerve and the contributors of the long thoracic nerve.
- Babies presenting with persistent Horner’s sign and findings of lower trunk injury may have a poor prognosis.
- Persistent denervation of the elbow flexors beyond the age of 4 months, scapular winging, and Horner’s sign may be the indications of surgical exploration.
- Shortness of the involved extremity, glenohumeral dysplasia, radiocapitellar dysplasia, and deformities of the hand are the serious complications of brachial plexus birth injury.

Reanimation Procedures in OBPP

The majority of a brachial plexus birth injury may recover spontaneously and do not require surgery. Infants that present with a flail extremity and a Horner syndrome, indicating a complete injury, frequently require exploration and reconstruction of the brachial plexus. Absence of elbow flexion and loss of biceps function at the age of four months may be used as indicator for operative treatment. When assessment of elbow, wrist, finger and thumb extension is added, the prediction of poor recovery may be more precise. However, no single classification and algorithm are applicable to all patients. The decision for a surgical intervention must be based on examination of the function over time in the individual infant. Clark et al have developed an algorithm for treatment that includes motor assessment at birth, and at the age of three, six, and nine months. At the age of three months, a score less than 3.5 is an indication for surgical treatment. In patients with higher scores, the evaluations are repeated regularly. Failure to achieve higher scores at the follow up is an indication for surgical management. The major determinant for the timing of any surgical intervention is the changes in the target organ in the denervated extremity. Reconstruction of the brachial plexus is a complex and lengthy procedure. The patient’s general medical condition must be prepared for a long procedure. Restricted
respiratory function and systemic conditions, such as coagulopathies and muscular dystrophies, may be contraindications for surgery.
Surgical treatment of shoulder abnormalities in OBPP

Gráinne Bourke. Leeds Teaching Hospitals Trust.

Neonatal Brachial Plexus injury is rare occurring in 0.42 per 1000 live births in the U.K. However the longterm outcome for those who do not have early recovery of limb movement can result in significant disruption of shoulder joint development and subsequent movement and function.

Injuries are defined by the number and site of the roots that are involved in the injury pattern. Broadly these are divided into upper trunk, upper and middle trunk, global palsies and those involving only the lower roots. In all but the last group, which is the most rare, the shoulder is affected.

The pattern of development on the shoulder if untreated can lead to hypoplasia and flattening of the humeral head combined with a retroverted, biconcave glenoid. This joint deformity combined with the variable pattern of reinnervation of the shoulder girdle musculature results in a deficiency of abduction and rotation of the glenohumeral joint and visual displacement of the scapula.

The functional impact of this shoulder involvement is apparent throughout growth and into adult life affecting the individuals in work, daily living and leisure activities.

Non operative strategies such as physiotherapy are important for both maintaining passive range of motion and encouraging active range. Other non operative strategies include the use of splinting and botulinum toxin to weaken the medial rotational muscle force across the shoulder joint early in childhood.

Serial Ultrasound to evaluate the shoulder joint congruency along with clinical examination of nerve recovery can select those cases of early shoulder joint subluxation.

Operative strategies include anterior soft tissue release including release or lengthening on the subscapularis, the coraco-humeral ligament and adjustment to the coracoid length. This can be done in isolation or in combination with external rotation transfer of the Latissimus dorsi and or Teres major posteriorly when active external rotation is absent. Arthroscopic and open releases have been described in the literature.

Skeletal surgery includes both techniques to alter and augment the glenoid by opening wedge or bone block to prevent subluxation and dislocation. Rotational
osteotomies of the humerus either to change the active range in medial or lateral rotation are also well described.

References


Evaluation and management of brachial plexus birth palsy.

Abzug JM, Kozin SH.

Prevention of brachial plexus injury-12 years of shoulder dystocia training: an interrupted time-series study.


Current concepts in the management of brachial plexus birth palsy.

Hale HB, Bae DS, Waters PM.

Management strategies for shoulder reconstruction in obstetric brachial plexus injury with special reference to loss of internal rotation after surgery.

Sibinski M, Hems TE, Sherlock DA.


Upright MRI of glenohumeral dysplasia following obstetric brachial plexus injury.

Nath RK, Paizi M, Melcher SE, Farina KL.


Correlation between external rotation of the glenohumeral joint and deformity after brachial plexus birth palsy.

Kozin SH.
Posterior subluxation and dislocation of the shoulder in obstetric brachial plexus palsy.

Kambhampati SB¹, Birch R, Cobiella C, Chen L.


Posterior shoulder dislocation in infants with neonatal brachial plexus palsy.

Moukoko D¹, Ezaki M, Wilkes D, Carter P.


Management of Shoulder Problems Following Obstetric Brachial Plexus Injury.

Nixon M¹, Trail I².


Arthroscopic release and latissimus dorsi transfer for shoulder internal rotation contractures and glenohumeral deformity secondary to brachial plexus birth palsy.

Pearl ML¹, Edgerton BW, Kazimiroff PA, Burchette RJ, Wong K
What do I do with a radial nerve palsy and a humeral shaft fracture?

Esther Vögelin

The unique anatomic location of the radial nerve in the arm puts it in danger of injury during humeral shaft fractures and surgical intervention. As many as one third of radial nerve palsies associated with humeral fractures occur at the time of surgery [1,2]. Although preoperative radial nerve paralysis was reported in 10–18% of cases, with spontaneous recovery in the majority [3-5], faster and more complete recovery has been associated with early ORIF [6]. Post-operative radial nerve paralysis occurs in 4–6.5% of patients, with spontaneous recovery in 80–90% [4, 5, 7]. Finally, nonunion and infection may complicate humeral ORIF in 2.8–5.8% and 1–5% of cases, respectively [4]. The overall incidence of radial nerve injury after humeral shaft fractures is reported with 11.8%-13% representing the most common peripheral nerve injury associated with bone fractures. [7] Nerve palsies occur most commonly with spiral fractures, but direct trauma or traction mechanisms in transverse or oblique fractures of the distal third can result in nerve palsies as well.

The high percentage of radial nerve injury associated with bone fracture is attributable to the intimate contact with the periosteum of the humerus on the posterior mid-shaft and, its poor mobility piercing the lateral intermuscular septum (12.5 cm proximal of the olecranon fossa). [8]

Causes of post-operative radial nerve paralysis are poorly documented in the literature. This complication is generally thought to be related to laborious reduction manoeuvres, inappropriate placement of bone holders and retractors, overstretching of the nerve over the plate, unintended compression of the nerve underneath the plate and inadvertent direct injury from the surgical blade. The usual application of the plate underneath the radial nerve may cause overstretching, related to plate thickness and width. Elongation of the nerve by 5 mm has been reported over a lateral humeral narrow plate 3.2-mm thick and 13-mm wide in a cadaveric study by El Ayoubi et al. [9]; these authors recorded 11-mm length gain in the nerve when it was experimentally transposed through the fracture site to the medial aspect of the humerus. Transfracture transposition of the radial nerve during lateral and posterior approaches has therefore be recommended by some authors. [9-11] The ideal situation for transposition includes a fracture location that lies between 2 cm proximal and 7 cm distal to the midshaft of the humerus, a comminution that results in humeral shaft shortening and soft tissue disruption with traumatic spontaneous dissection of the radial nerve. [9] In those series a pre-operative radial nerve palsy was seen between 30-50% all of which recovered after transfracture medial transposition of the radial nerve. A disadvantage may be the potential of additional devascularization of the humerus related to this more extensive dissection.

Controversies still exists in the treatment of humeral shaft fractures associated with radial nerve injuries. There are two distinct entities – the preoperative and secondary postoperative radial nerve palsy – with different pathophysiological mechanisms, i.e. trauma-related versus iatrogenic. When it is useful or necessary to explore the radial nerve in patients with pre- and
postoperative palsy? A personal view supported by the literature to define a management strategy of radial nerve palsies in humeral fractures.

Diagnosis
Active extension of the wrist, fingers, and thumb at the metacarpophalangeal joints suggests an intact motor function of radial nerve. Radial nerve sensation to light touch and pinprick can be tested in the dorsal radial aspect of the hand toward the thumb/index web space. Tinel’s sign is helpful in indicating the progression of nerve recovery. If the brachioradialis or extensor carpi radialis longus (ECRL) is not functioning, the injury is expected at the level of the humeral shaft. If the lesion is distal to the origin of the posterior interosseus nerve, ECRL function will be intact and the wrist will be pulled into radial deviation with attempts at extension. Neurophysiologic tests are helpful after 3-4 weeks to determine the level and extent of radial nerve injury to get a baseline of function. Intraoperative nerve stimulation and somatosensory evoked potential monitoring may be helpful during surgery to monitor the status of the nerve. [8] Sonography [12] and MR Neurography [13] are excellent to visualize nerve morphology but allow no prognosis for functional recovery except in Sunderland IV lesions.

Treatment options
Conservative management or early surgical exploration including different algorithms are still controversial. [8, 14-17] Closed humeral shaft fractures with complete motor and sensory radial nerve injury tend to recover in 73-92% [8]. Most patients with spontaneous recovery begin to demonstrate recovery during the first few months. Brachioradialis and ECRL are the first muscles to be reinnervated, and the extensor indicis proprius is the last muscle to recover. Complete recovery typically occurs within 6-12 months, because axons may regenerate with a speed of 1.7 mm per day distally to the site of injury. [8] If a follow-up electrodiagnostic study at the 12-week point shows similar findings as the baseline, then exploration may be indicated.

Early exploration must be recommended in open fractures, associated soft- and vascular injuries, radial nerve deficit after surgery, intractable neurogenic pain suggesting nerve entrapment or compression [18]. The risk of radial nerve injury during plate and screw fixation is reported as 0-10%, during intramedullary nailing is reported between 0-5% [8]. Although the latter is less invasive, there is a risk of radial nerve injury during intramedullary nailing. Early exploration within 2-3 weeks from trauma allows a more accurate determination of nerve injury than later. Neuroma in continuity Sunderland I-III lesions are usually treated by neurolysis, IV and V require resection of nerve lesion and grafting the resulting defect. Surgical intervention for radial nerve injury can be classified into neurorrhaphy, nerve grafting, nerve transfers, and tendon transfers. If transection of the radial nerve is found during immediate surgical exploration, a primary nerve repair is indicated. Adequate resection of surrounding scar tissue and neuromas should be performed until healthy nerve fibers are seen under the microscope in order to facilitate recovery. Nerve grafting is preferred in cases of tension at the neurorrhaphy site or
if there is a nerve gap that has to be bridged. Results after nerve grafting are in line with primary nerve repair with excellent results and functional restoration after nerve grafting.[8]

Factors that influence the outcome after grafting include, the violence of the injury, the length of the defect, that should be bridged and the denervation time. Another main predictor of outcome in radial nerve injury remains the age, independent of primary or secondary injury with or without exploration [19]. However, after fracture fixation and stabilization is achieved, a neurolysed or repaired nerve will potentially benefit from a better environment for recovery with less tension, motion, or callus formation to impede nerve healing.

In young patients, additional distal nerve transfers [20,21] may be an option while in elderly patients, early tendon transfers may accelerate the functional use of the involved hand. Tendon transfers [22] may always be performed in patients with incomplete recovery, and/or failed nerve reconstruction. The patient’s level of function, anatomy, and level of radial nerve injury are three factors routinely used to decide on the appropriate tendons to use for transfer. Wrist extension may be achieved with transfer of pronator teres into extensor carpi radialis brevis, whereas for finger extension, flexor carpi radialis (FCR) is usually used. Additionally, we prefer to perform a palmaris longus tendon (PL) and or flexor digitorum superficialis (FDS III or IV) transfer, to restore thumb extension (extensor pollicis longus, EPL) and radial abduction (abductor pollicis longus and or extensor pollicis brevis, APL and EPB).

Conclusions
Exploration of radial nerve is indicated in open fractures, irreducible fractures, vascular or severe soft tissue injuries, high-energy trauma, sharp or penetrating injury. Early exploration of the radial nerve in secondary nerve palsy with Holstein–Lewis, oblique or transverse fracture of the humeral diaphysis is recommended when the radial nerve had not been exposed during the initial surgery. Another reason for exploration at any time postoperatively is intractable neurogenic pain. Even without a visible nerve damage during the primary approach of fracture repair a secondary palsy up to 45% recovery may be expected but total recovery rate of 75% in secondary nerve palsy is reported after early re-exploration. [17]
Surgical exploration may be initially deferred for fracture with low risk of radial nerve injury. Radial nerve palsy is regularly monitored by neurophysiologic and clinical examination after 3 weeks following the trauma. The clinical improvement of nerve function is the key. If there are no electrophysiological changes at 12 weeks, nerve imaging and surgical exploration is recommended, although functional results of late surgical exploration and repair deteriorate. Surgical interventions include neurolysis or nerve grafting and or tendon or nerve transfers depending on the age of the patient.
In summary, early nerve exploration for a potentially compressed or damaged radial nerve is recommended in secondary radial nerve palsy in which the nerve had not been exposed during initial surgery.
References


Li YL, Ning GZ, Wu Q, Wu QL, Li Y, Feng SQ. Review of literature of radial nerve injuries associated with humeral fractures: an integrated management strategy. Department of Orthopaedics, Tianjin Medical University General Hospital, Tianjin, PR China. Plos One (2013) 8(11)


Question
A 25 year-old man sustains a closed humerus fracture as a result of a fall. He is treated successfully treated with closed reduction. Immediately after reduction, he is unable to actively extend his wrist. In the absence of clinical recovery, the most appropriate time to obtain an EMG to evaluate reinnervation of the ECRL is:

A) 1 week  
B) 6 weeks  
C) 3 months  
D) 6 months  
E) 9 months

Correct answer: A-, B-, C+, D-, E-