Ultrasound

Jeremy D.P. Bland, MBChB
Michael S. Cartwright, MD, MS
Craig Zaidman, MD
Victor H. Flores, MD
Please be aware that some of the medical devices or pharmaceuticals discussed in this handout may not be cleared by the FDA or cleared by the FDA for the specific use described by the authors and are “off-label” (i.e., a use not described on the product’s label). “Off-label” devices or pharmaceuticals may be used if, in the judgment of the treating physician, such use is medically indicated to treat a patient’s condition. Information regarding the FDA clearance status of a particular device or pharmaceutical may be obtained by reading the product’s package labeling, by contacting a sales representative or legal counsel of the manufacturer of the device or pharmaceutical, or by contacting the FDA at 1-800-638-2041.
# Ultrasound

## Table of Contents

- Course Committees & Course Objectives ........................................ 4
- Faculty .......................................................................................... 5
- Prognosis in Peripheral Nerve Disease
  *Jeremy D.P. Bland, MBChB* ......................................................... 7
- Neuromuscular Ultrasound Outside the Wrist: Other Entrapment Neuropathies
  *Michael S. Cartwright, MD, MS* .................................................. 13
- Clinical Uses of Muscle Ultrasound
  *Craig Zaidman, MD* .................................................................... 19
- Musculoskeletal Ultrasound Guided Procedures
  *Victor H. Flores, MD* ................................................................. 25
- CME Questions .............................................................................. 31

---

*No one involved in the planning of this CME activity had any relevant financial relationships to disclose.*

*Authors/faculty have nothing to disclose*

*Chair: Francis O. Walker, MD*

*The ideas and opinions expressed in this publication are solely those of the specific authors and do not necessarily represent those of the AANEM.*
Objectives

Objectives - Participants will acquire skills to (1) describe key sonographic and EDX factors that help provide a prognosis for the treatment of CTS, (2) list three common entrapment neuropathies that can be imaged with US and discuss transducer orientation and techniques for doing so, (3) identify two common changes in muscles with neurogenic/myopathic disorders and describe the typical distribution of findings in neuropathy and myopathy, and (4) explain two common MSK disorders which can be diagnosed with US that can mimic entrapment neuropathy.

Target Audience:
• Neurologists, physical medicine and rehabilitation and other physicians interested in neuromuscular and electrodiagnostic medicine
• Health care professionals involved in the management of patients with neuromuscular diseases
• Researchers who are actively involved in the neuromuscular and/or electrodiagnostic research

Accreditation Statement - The AANEM is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education (CME) for physicians.

CME Credit - The AANEM designates this live activity for a maximum of 3.25 AMA PRA Category 1 Credits™. If purchased, the AANEM designates this enduring material for a maximum of 5.75 AMA PRA Category 1 Credits™. This educational event is approved as an Accredited Group Learning Activity under Section 1 of the Framework of Continuing Professional Development (CPD) options for the Maintenance of Certification Program of the Royal College of Physicians and Surgeons of Canada. Physicians should claim only the credit commensurate with the extent of their participation in the activity. CME for this course is available 10/2012 - 10/2015.

CEUs Credit - The AANEM has designated this live activity for a maximum of 3.25 AANEM CEUs. If purchased, the AANEM designates this enduring material for a maximum of 5.75 CEUs.

2011-2012 Course Committee

Shawn J. Bird, MD, Chair
Philadelphia, PA

Shashi B. Kumar, MD
Tacoma, WA

Marcy C. Schlinger, DO
Bath, MI

Lawrence W. Frank, MD
Elmhurst, IL

A. Arturo Leis, MD
Jackson, MS

Nizar Souayah, MD
Westfield, NJ

Taylor B. Harrison, MD
Atlanta, GA

Benjamin S. Warfel, II, MD
Lancaster, PA

2011-2012 AANEM President

John C. Kincaid, MD
Indianapolis, IN
Ultrasound

Faculty

Dr. Jeremy D. P. Bland, MB ChB
Department of Neurology
East Kent Hospitals University NHS Trust (Canterbury)
Kings College Hospital (London)
United Kingdom

Dr. Bland is a practicing clinical neurophysiologist at both Kings College Hospital in London and The Kent and Canterbury Hospital in Canterbury, UK. He has written on all aspects of the diagnosis and management of carpal tunnel syndrome (CTS) and is developing an interest in peripheral nerve ultrasound imaging. Dr. Bland shares his CTS work on his website: http://www.carpal-tunnel.net, which draws on information from more than 3500 CTS articles and work with more than 30,000 patients. His medical degree was earned at the University of Manchester in the United Kingdom.

Michael S. Cartwright, MD, MS
Associate Professor of Neurology
Department of Neurology
Wake Forest School of Medicine
Winston-Salem, NC

Dr. Cartwright is an Associate Professor in the Department of Neurology at Wake Forest University School of Medicine (WFUSM). He is an author or co-author of more than 40 publications, many of which focus on neuromuscular ultrasound. He is also the co-editor of the textbook Neuromuscular Ultrasound. Dr. Cartwright is active in clinical practice as well as research and has particular interest in projects that advance the clinical utility of neuromuscular ultrasound. His medical degree, internal medicine internship, and neurology residency were completed at WFUSM.

Craig Mitchell Zaidman, MD
Department of Neurology
Washington University St. Louis
St. Louis, Missouri

Dr. Zaidman was a Fulbright Postgraduate Research Fellow and completed residencies in pediatrics and neurology at Brown University and a residency in child neurology and a fellowship in clinical neurophysiology at Washington University (WU). He serves now as an assistant professor in WU’s Neuromuscular and Pediatric Neurology Divisions. Dr. Zaidman is a member of the American Academy of Neurology and the American Institute of Ultrasound in Medicine. His research interests include developing new approaches in using ultrasound to evaluate diseases of the nerves and muscles.

Victor H. Flores, MD
Private Practice
Physical Medicine Associates
Fort Worth, Texas

Dr. Flores is in private practice in Fort Worth, TX. He received his medical degree from Centro de Estudios Tecnicos in Santa Domingo, Dominican Republic. His fellowship was completed at the University of Texas, Southwestern Medical School in Dallas, Texas. Dr. Flores is a diplomate of the American Board of Physical Medicine & Rehabilitation and the American Board of Electrodiagnostic Medicine. His interests include musculoskeletal medicine, especially musculoskeletal ultrasound.
INTRODUCTION: WHY WORRY ABOUT PROGNOSIS?

If medicine is the art and science of improving health, then the most important resource for physicians is knowledge of which actions will produce results that are beneficial or detrimental for the patient who is seeking advice. The accumulation and refinement of this knowledge is the aim of evidence-based medicine and all of the studies of disease are ultimately in the service of answering the patient’s question: “What should I do in order to get better?” In electrodiagnosis this is often forgotten, with the perception that the focus should be defined by the word “diagnosis,” embodied in the term itself, and that the primary aim is to to put a name to the patient’s problem. Diagnosis, however, is to some extent a side issue. Putting a name to a patient’s disorder is often a useful stepping stone towards choosing a course of action but is rarely an end in itself. The aim here is to review the knowledge which will help to answer the patient’s question in the context of peripheral nerve disease and with particular reference to whether the new techniques of nerve imaging adds to the ability to predict the future.

Diagnosis may not be the ultimate aim but it does provide a means of structuring the discussion. Peripheral nerve disease can be divided into two groups:

- Multifocal or diffuse nerve pathology (polyneuropathy or mononeuritis multiplex)
- Localized pathology of a single nerve (entrapment, mononeuritis, or trauma)

By a large margin, the most common single nerve condition is carpal tunnel syndrome (CTS). There is a wealth of literature on both the effectiveness of different treatments and on the pretreatment factors which influence the success of individual treatments. Therefore, the focus here will be to discuss the prognosis in CTS. The hope is that principles established from the study of CTS will prove more widely applicable in other disorders.

Only three interventions for CTS are conclusively known to have a significant therapeutic effect greater than placebo: neutral angle splinting, steroids, and surgery. The patterns of response to these interventions are different in time course and in the types of symptoms which occur. Each intervention also has to be viewed in terms of short- and longterm outcome.

EVIDENCE-BASED TREATMENT OPTIONS FOR CARPAL TUNNEL SYNDROME

Surgery

Surgery is the definitive treatment for CTS. The outcome for the patient results from the effects of two different anatomical changes: relief of pressure on the median nerve and the division of the transverse carpal ligament (which has a role both in stabilizing the structure of the carpus and in the mechanical action of the long flexor tendons). In the short term, the immediate discomfort of surgery may be worse than the CTS symptoms. In the long term, recurrence of median nerve compression, and thus clinical CTS, after successful surgery is very rare, but long-term complaints of pain and weakness of grip are relatively common. The outcome therefore depends both
on the ability of the median nerve to recover from the compressive insult and how resilient the patient is in coping with the effects of section of the transverse carpal ligament. Surgical success rates in different studies vary from 23% to 100%, with a mean of about 75%.

Steroids

Steroids do not produce a major anatomical change in the carpal tunnel; indeed the mechanism of action is somewhat obscure. Many studies have demonstrated that there is a marked short-term benefit and an equally marked long-term tendency to recurrence of CTS symptoms. The response to steroids is therefore composed of the initial symptomatic response and subsequently, in those who respond positively, the length of remission. The patient, however, does not have to contend with the adverse effects of the destruction of the transverse carpal ligament. It should also be noted that, although they are often grouped together as “failure of injection treatment,” there is a marked difference between those patients who have no response to injection and those who respond well in the beginning and then relapse. Initial response rates to injection are generally about 70-80%, with up to 92% relapsed by 2 years postinjection.

Splinting

There are few studies on the results of splints. Splints share with steroid injection the lack of permanent anatomic change and thus the lack of major adverse consequences from the treatment. A study of 83 patients found that 26 (31%) who had been treated with splinting alone required no further treatment after 1 year.

It should be remembered that CTS is not always progressive; one-fifth of patients improved without specific treatment in one study.

PROGNOSIS FOR CARPAL TUNNEL SYNDROME

The questions physicians can try to answer for the patient include:

- Will splinting help the symptoms?
- Will injection improve symptoms in the short term?
- If symptoms do respond to injection, how long will it last?
- Will surgery improve CTS symptoms both in the short and long term?
- What other consequences are there likely to be from surgery?

In each case, the physician should try to tailor the answer to the individual circumstances of that patient. Physicians need to make use of general information such as age, gender, other disease present, and so forth. Physicians also need to use the results of laboratory measurements of the median nerve, both anatomical (cross sectional area [CSA] on ultrasound [US]) and physiological (nerve conduction study [NCS] severity expressed as Canterbury severity grade), and also the pattern and severity of their CTS symptoms. Subjective severity of hand symptoms can be evaluated both before and after treatment using the Boston/Levine Questionnaire Symptom Severity Score (SSS) and Functional Status Score (FSS) subscales.

Many of the numerical results listed below are drawn from a patient database of 33,586 individuals referred to the electrophysiology laboratory in Canterbury, United Kingdom, with a suspected diagnosis of CTS between 1991 and 2012. Most of these data are unpublished and all figures have been recalculated here using the most recently available data. All such figures are identified as using the Canterbury data.

Prognosis for Splinting

There are few studies of splinting used in isolation as a treatment for CTS. Splints are mostly used in combination with other “treatments”—nonsteroidal anti-inflammatory drugs (NSAIDs), rest, activity modification, etc.—even though there is little or no evidence from good-quality randomized controlled trials to show that these other interventions are any better than placebo. In a typical example, 331 hands in 229 patients were treated with some combination of wrist splint, NSAIDs, steroid injection (16.4%), and oral steroids (26.8%). A good clinical result (no need for surgery) was associated with age < 50 years, duration of symptoms < 10 months, intermittent rather than constant parasthesias, a negative Phalen’s test (30 s), and the absence of “stenosing flexor tenosynovitis” (though the way in which this last diagnosis was made is not clear). In this study, it is impossible to disentangle the effect of splinting from the other interventions. Analysis of the splint subgroup in a Dutch study of splinting versus surgery suggested that a short duration of history at presentation and less severe nocturnal parasthesias were predictors of success.

Nerve Conduction Studies

Patients with electrophysiologically more severe CTS are less likely to report benefit from use of a night splint. Of 3,473 patients who had already tried splinting when first demonstrated to have confirmed CTS on NCS, 1,756 (51%) reported obtaining some relief of symptoms. The response rates by neurophysiological grade (1 = mildest to 6 = most severe CTS) are shown in Table 1 (Canterbury data).
In 155 patients who had already tried splinting when first confirmed to have CTS on NCS, the mean combined CSAs of the two median nerves were 23.7 mm$^2$ in those who reported a response to splinting and 28.1 mm$^2$ in those who reported no response ($p = 0.004$) (Canterbury data).

**Specific Symptoms**

Of the 3,473 patients with confirmed CTS who had already tried splinting, those who report night-time waking with CTS symptoms were more likely to report a response to splinting (1,538/3,473 or 52%) than those who did not have night-time waking (218/542 or 41%) ($p < 0.0001$). Similarly, patients who reported intermittent symptoms (598/1,073 or 56%) were more likely to report benefit from splinting than those who reported constant symptoms (621/1,386 or 45%) ($p < 0.0001$) (Canterbury data).

**Prognosis for Injection**

**Initial Response**

Not all patients report a response to steroid injection when it is first performed. Some patients report no benefit.

**Symptom Severity**

Symptom severity may predict response. In a study of 89 symptomatic hands, in 58 patients who were followed up for 3 months of a conservative treatment regimen, a lower SSS was found to be predictive of successful conservative treatment. In this group, 14/58 or 24% of patients did not require surgery and 12 of these had steroid injection as part of their treatment regimen. This study likely reflects largely the outcome of steroid treatment. Thirty-three percent of patients with SSS < 2.5 required surgery compared to 89% of those with SSS > 2.5.$^9$

At 6 weeks after an initial injection in 1,612 patients, 40 patients reported symptoms to be worse, 109 unchanged, 230 somewhat improved, 743 much improved, and 490 completely symptom free. Taking the last two categories as indicating successful injection, the success rate is 76% with a further 14% indicating some improvement. The mean preinjection SSS for the successful injections was 2.70 compared to 2.91 for the unsuccessful ones ($p < 0.0001$). The mean preinjection FSS for the successful injections was 2.03 compared to 2.27 for the unsuccessful ones ($p < 0.00001$) (Canterbury data).

**Electrophysiology**

A good initial response to steroid injection is less likely with electrophysiological grade 5 CTS (grade 5 versus grades 2-4, $p < 0.01$) and with grade 1 CTS (grade 1 versus grades 2-4, $p < 0.05$) (see Fig. 1) (Canterbury data).

**Ultrasound**

In a study in the Netherlands, successful injection treatment was associated with a CSA of 10.9 mm$^2$ compared to 12.48 mm$^2$ for those patients who had to resort to surgery.$^{10}$ A small Korean study reported a weak ($r = 0.52$) but significant correlation between clinical response to injection and the median nerve CSA 2 cm proximal to the point of maximum enlargement.$^{11}$ Patient outcomes reported in Kent 6 weeks after injection are shown in Table 2. The difference between the 6-9 mm$^2$ (normal) group and the > 15 mm$^2$ group is significant ($p < 0.05$) (Canterbury data).

**Time to Relapse after Successful Injection**

Electrophysiology may be of some help in predicting length of remission. A Dutch study of 273 patients who were injected with 40 mg methylprednisolone found median times to relapse of 15 months in mild cases, 5 months in moderate cases, and 4.5 months in severe cases, as judged by electrophysiology.$^{12}$ This result cannot be duplicated in data from Canterbury. Mean times to presentation for further treatment are shown in Table 3 for 655 patients who initially reported benefit from their first steroid injection at 6 weeks.

**Symptom Severity**

Length of remission after steroid injection is likely to be longer in patients with milder initial subjective symptoms. Mean relapse times related to the Boston/Levine SSS and FSS scores are shown in Table 4 and show a significant trend to longer remissions in pa-
Table 3. Mean time to relapse after successful first injection in 655 patients
Mean relapse times are given in days.
Canterbury NCS grade for CTS: 0 = normal to 5 = very severe

<table>
<thead>
<tr>
<th>Canterbury NCS grade</th>
<th>Patients</th>
<th>Mean relapse time</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>6</td>
<td>414</td>
</tr>
<tr>
<td>1</td>
<td>103</td>
<td>439</td>
</tr>
<tr>
<td>2</td>
<td>216</td>
<td>377</td>
</tr>
<tr>
<td>3</td>
<td>240</td>
<td>370</td>
</tr>
<tr>
<td>4</td>
<td>57</td>
<td>411</td>
</tr>
<tr>
<td>5</td>
<td>33</td>
<td>428</td>
</tr>
</tbody>
</table>

NCS = nerve conduction study

Table 4. Mean time to relapse after successful first injection versus subjective severity
SSS are grouped into bands.
Mean times to re-presentation for further treatment in days.

<table>
<thead>
<tr>
<th>Preinjection SSS/FSS</th>
<th>Patients</th>
<th>Mean relapse time (SSS)</th>
<th>Mean relapse time (FSS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 to 1.99</td>
<td>113</td>
<td>493</td>
<td>397</td>
</tr>
<tr>
<td>2 to 2.99</td>
<td>288</td>
<td>384</td>
<td>388</td>
</tr>
<tr>
<td>3 to 3.99</td>
<td>220</td>
<td>365</td>
<td>361</td>
</tr>
<tr>
<td>4 to 5</td>
<td>33</td>
<td>273</td>
<td>457</td>
</tr>
</tbody>
</table>

p = 0.0004 p = 0.23

FSS = functional status score, SSS = symptom severity score

In contrast to the outlook for injection, in surgical cases the SSS shows no definite predictive value for surgical outcome (p = 0.5, r = −0.14) but impaired function, as indicated by the FSS scale, is clearly, though weakly, correlated with poorer outcomes (p = 0.00006, r = −0.163) (Canterbury data).

Ultrasound

Studies regarding US have produced mixed results. One study of 112 operations found that patients with larger nerves had better outcomes. The maximal mean CSA before successful operations in 71 patients was 15.8 mm² while the same measure in 41 unsuccessful operations was 13.4 mm² (p = 0.017). In a second study of 88 operations, no correlation could be demonstrated between preoperative CSA and outcome.

In Kent, the presurgical CSA, though weakly correlated with the electrophysiological severity, is not significantly correlated with surgical outcome. In 101 patients to date, the surgical success rates at 3 months after surgery are 12/20 (60%) in hands with normal CSA (< 10 mm²), 30/55 (55%) in those with CSA of 10-15 mm², and 15/26 (58%) in those with CSA > 15 mm². Analyzing these data in the same way as the study by Naranjo provides the following CSA data: successful group 13.3 mm² and unsuccessful group 12.8 mm² (p = 0.33, 101 cases.) (Canterbury data).

In a multivariate analysis of the outcomes of 146 operations, no single factor was independently predictive of outcome. A combined model incorporating age, body mass index, US CSA, SSS, FSS electrophysiological grade, and duration of symptoms was, however, able to predict the outcome of surgery with moderate accuracy (area under a receiver operating curve 0.84). This result requires verification using a prospective data set and further study with greater numbers (Canterbury data).

Nerve Conduction Studies

The literature offers widely varying opinions on the predictive value of NCS abnormalities but data from Canterbury strongly support a complex relationship between electrophysiological grade and surgical outcome. The data show the best outcomes occur in patients with middle-grade abnormalities (Figure 2).
variability in outcome remains unexplained, especially in relation to surgical carpal tunnel decompression. It is likely that factors acting after the physician meets the patient and chooses a treatment, such as the expertise of the surgeon, play an important role.

**SPECIAL CIRCUMSTANCES IN CARPAL TUNNEL SYNDROME**

The widespread use of US imaging in CTS has revealed several interesting observations. First, although occasionally commented on in the surgical literature, high branching of the median nerve, proximal to the carpal tunnel, is observed on US. In Canterbury images, 161 of 2,336 nerves showed two branches at the wrist crease (6.9%) and 11 showed three branches (0.5%). In many cases, it is likely that the imaging is showing two major nerve divisions which are in fact still enclosed within a common epineurium and which would appear to the eye as a single nerve at surgery. Whether this finding is of relevance to either injection therapy or surgery, especially endoscopic surgery, remains to be explored. Much has been made in the US literature of the discovery of anatomical abnormalities in CTS cases by imaging before surgery, but there is little evidence that knowledge of, for example, a ganglion within the carpal tunnel or a persistent median artery makes any difference to the surgical approach unless endoscopic decompression is being considered and such findings are much rarer than bifid median nerves.

Secondly, failed carpal tunnel decompression is often a reason for referral for postoperative NCSs. The surgical outcomes of repeat carpal tunnel decompressions are considerably worse than those of primary operations (48% success versus 73% success overall, p < 0.000005) (Canterbury data). It is important to establish whether a complete section of the transverse carpal ligament has been achieved as this is a relatively common surgical error (54% of all failed operations in one series) and is amenable to repeat surgery. Incomplete decompression can be strongly suspected when NCSs have either deteriorated or remained unchanged shortly after surgery but US imaging can add further support to this assessment, often showing a sharply defined indentation of the nerve on longitudinal imaging or the persistence of a continuous band of hyperechoic ligament across the nerve in transverse images.

**PROGNOSIS IN OTHER ENTRAPMENT NEUROPATHIES**

Currently, the only other entrapment neuropathy to have provided enough cases for analysis of the surgical prognosis is ulnar neuropathy at the elbow. Beekman and colleagues were able to follow 46 conservatively treated and 28 surgically treated cases. With both treatment options, subjects with good outcomes had smaller ulnar nerve diameters at the elbow than those whose condition remained unchanged or deteriorated (2.8 versus 3.5 mm for conservative treatment, 3.0 versus 3.5 mm for surgical treatment). In a multivariate analysis, ulnar nerve diameter, conduction block across the elbow, and motor conduction slowing were significant predictors of outcome.

**OTHER SINGLE Nerve LESIONS**

There are obvious applications for US imaging in trauma to peripheral nerve as the imaging can demonstrate structural continuity of the nerve at the site of injury even when there is complete axonotmesis or conduction block and before needle electromyography (EMG) changes of denervation occur. Ultrasound imaging has been found to correlate well with the findings at surgical exploration. There are however no published studies demonstrating the prognostic value of such information in significant numbers of patients.

**POLYNEUROPATHIES**

Both acquired and inherited polyneuropathies have been shown to demonstrate either diffuse or focal enlargements of nerve on US imaging. Beekman and colleagues found focal enlargements in the majority of 21 patients with multifocal motor neuropathy, sometimes unrelated to the occurrence of focal NCS abnormalities at the same sites. Such imaging changes have not as yet been related to either the natural progression of disease or response to treatment.

**CONCLUSION**

Prediction of the outcome of medical treatment will always be somewhat uncertain and is difficult to study. However, it is clear that laboratory measures, including NCSs and US imaging do have some predictive power for some interventions, at least in CTS. Making use of this information for individual patients requires an understanding both of the complex relationships between individual measurements and prognosis and of the considerable uncertainty which remains even when every currently known prognostic variable is known.

**REFERENCES**

Neuromuscular Ultrasound Outside the Wrist: Other Entrapment Neuropathies

Michael S. Cartwright, MD, MS
Associate Professor of Neurology
Department of Neurology
Wake Forest School of Medicine
Winston-Salem, North Carolina

INTRODUCTION

Ultrasound (US) of diseased muscle was first described in the early 1980s in pediatric patients with muscular dystrophy and spinal muscular atrophy. In 1988, Formage provided the first description of high-resolution US for the assessment of peripheral nerves, and in 1991 Buchberger and colleagues first described the ultrasonographic findings in entrapment neuropathy (carpal tunnel syndrome [CTS]). Since then, techniques have been refined, knowledge has been gained, and many neuromuscular diseases have been studied with US, and neuromuscular US (NM US) has matured into a discipline that can be used in clinical and research settings to improve the assessment and treatment of a variety of conditions. Ultrasound is a promising technique because it is readily available, inexpensive, radiation-free, painless, portable, and it provides very high-resolution images of superficial structures (0.3 mm lateral resolution for US compared to 1 mm for magnetic resonance imaging and computed tomography). The area in which the most effort has occurred with NM US is in the evaluation of focal neuropathies caused by entrapment, and hundreds of articles have been written about NM US for CTS alone. The ultrasonographic findings in entrapment are relatively consistent across all the different entrapment syndromes, and they include enlargement of the nerve just proximal to the site of entrapment, changes within the nerve that result in a more hypoechoic (darker) appearance to the nerve, and increased vascularity within the nerve. Of these changes, nerve enlargement is likely the most accurate for diagnosis, so efforts have been made to establish reference values for nerve cross-sectional area using US (Table).

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Site</th>
<th>Upper limit of normal (mm²)</th>
<th>Side-to-side upper limit difference (mm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median</td>
<td>Pronator teres</td>
<td>11</td>
<td>2.8</td>
</tr>
<tr>
<td>Ulnar</td>
<td>Wrist</td>
<td>8.1</td>
<td>2.6</td>
</tr>
<tr>
<td></td>
<td>Forearm</td>
<td>8.3</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Distal elbow</td>
<td>8.6</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Elbow</td>
<td>8.8</td>
<td>2.2</td>
</tr>
<tr>
<td></td>
<td>Proximal elbow</td>
<td>9.3</td>
<td>1.8</td>
</tr>
<tr>
<td>Radial</td>
<td>Spiral groove</td>
<td>13.3</td>
<td>4.5</td>
</tr>
<tr>
<td>Tibial</td>
<td>Popliteal fossa</td>
<td>20.9</td>
<td>9.5</td>
</tr>
<tr>
<td></td>
<td>Fibular head</td>
<td>17.8</td>
<td>4.9</td>
</tr>
<tr>
<td></td>
<td>Ankle</td>
<td>22.3</td>
<td>5.7</td>
</tr>
</tbody>
</table>

Sources:
This discussion will describe the ultrasonographic changes seen in the common entrapment syndromes, except for CTS (which is described elsewhere in this booklet), with a practical emphasis on the technical aspects of performing these studies.

ULNAR NERVE

The ulnar nerve is most commonly entrapped at the elbow, with focal neuropathy potentially occurring in the cubital tunnel, at the medial epicondyle, and in the supracondylar region. The other site at which the ulnar nerve can become entrapped is at the wrist, as the nerve passes through Guyon’s canal.

At the Elbow

Ulnar neuropathy at the elbow (UNE) is the second most common site of focal neuropathy from entrapment, after the median nerve at the wrist (CTS). While electrodiagnostic (EDX) studies are helpful for the diagnosis of UNE, they are not as sensitive as they are for CTS because sensory studies across the elbow are challenging and there are no convenient ulnar-median comparison studies at the elbow. For these reasons, US is an attractive tool for evaluating UNE.

When evaluating potential UNE with US, a cross-sectional view is helpful (Figure 1). The ulnar nerve should be imaged in cross section from the mid forearm (distal to the cubital tunnel), across the medial epicondyle, and up into the mid arm. After examining this section of the ulnar nerve with US, the point of maximal enlargement should be identified and a cross-sectional area measurement made (taking care to ensure the transducer is perpendicular to the nerve so the smallest cross-sectional measurement is obtained). Then, distal and proximal sites along the ulnar nerve should be identified and measured, to provide internal reference values. An absolute cross-sectional area of the ulnar nerve greater than 10 mm² (or a ratio of the site of maximal enlargement compared to a distal or proximal uninvolved segment of greater than 1.5:1) should be used to diagnose focal UNE. The sensitivity of US for the diagnosis of UNE is 80% and specificity is 91%, and adding NM US to nerve conduction studies (NCSs) increases the sensitivity for diagnosis from 78% (NCSs alone) to 98%. It is often helpful to surgical colleagues to inform them of the exact site of ulnar nerve enlargement, as it may alter their surgical approach.

In addition to focal enlargement, US can also identify changes in ulnar nerve echogenicity and mobility in those with focal neuropathy. While the UNE tends to be more hypoechoic than other peripheral nerves, the author’s personal experience indicates that focal neuropathy at the elbow is often accompanied by a very hypoechoic, almost anechoic, ulnar nerve (Figure 1). Ultrasound can also be used to accurately identify ulnar nerve subluxation with flexion of the elbow, and a study of 212 elbows in healthy volunteers identified subluxation in 23.1% of elbows (with full dislocation in 8.5% of elbows). While it has not been proven that ulnar nerve subluxation increases the risk of focal neuropathy, it is suspected and it is worthwhile assessing for subluxation or complete dislocation when diagnosing UNE with US. Identification of a subluxed or dislocated ulnar nerve with US can also help trace the exact course of the ulnar nerve, which leads to a more precise measurement and NCS result. In a study of 78 elbows, ulnar nerve displacement during elbow flexion resulted in a falsely-increased calculated nerve conduction velocity by an average of 5.33 ms (SD 2.29 ms), which lead to false negative results and may explain the relatively decreased sensitivity of NCSs for the diagnosis of UNE.

At the Wrist

Ulnar neuropathy at the wrist is not nearly as common as it is at the elbow. However, it does occur and because it may be associated with anatomic abnormalities in Guyon’s canal it is informative to investigate it with NM US. To visualize the ulnar nerve at the wrist the transducer is placed to obtain a cross-sectional view. The ulnar artery is typically easily identified and the nerve lies adjacent and medial to the artery. Once the nerve is identified it should be followed distally as it enters Guyon’s canal. It runs lateral to the pisiform bone. The point of maximal cross-sectional area should be noted and measured, with a normal area less than 8 mm². It is within Guyon’s canal that particular attention should be paid to possible anatomic causes of entrapment and focal neuropathy. Case reports and series have described ganglion cysts, anomalous muscles, and thrombosed ulnar arteries within the canal compressing either the entire ulnar nerve or just the motor branch to the palm. In some cases, ulnar neuropathy at the wrist secondary to a ganglion cyst has presented with acute symptoms, which may raise the possibility of an anatomic compressive etiology.
Ganglion cysts have a typical appearance with US; they are round, anechoic, minimally compressible, and have no flow with Doppler imaging (Figure 2).

**RADIAL NERVE**

The radial nerve lies next to the humerus, in the spiral groove, and when compressed against the humerus a focal neuropathy and wrist drop can occur. This is sometimes termed a “Saturday night palsy” because an individual that falls asleep with an arm draped over a chair can suffer this type of injury. More distally, the radial nerve splits at the antecubital fossa into the superficial radial nerve and the deep posterior interosseous nerve. The posterior interosseous nerve can be entrapped by the supinator muscle.

**At the Spiral Groove**

To image the radial nerve it works best to position the patient supine and place the transducer on the lateral portion of the antecubital fossa to obtain a cross-sectional view. In this position, the radial nerve often has two prominent fascicles, and when they are traced distally they become the superficial radial and posterior interosseous nerves. When traced proximally the two fascicles converge and become a single round structure that lies adjacent to the humerus in the spiral groove (Figure 3). The small profundus artery can often be identified running with the radial nerve at this level. In cases of “Saturday night palsy” the radial nerve typically has a relatively normal appearance, although it may be slightly enlarged and hypoechoic. Importantly, US and dynamic imaging with flexion and extension of the elbow can be used to demonstrate that the radial nerve is anatomically intact in severe cases.

**Posterior Interosseous**

As described previously, the radial nerve is easily identified because of the two prominent fascicles at the level of the lateral antecubital fossa. When followed distally the radial nerve splits and the deep branch (posterior interosseous nerve) runs deep to the supinator muscle. As it does so, it normally flattens but the over cross-sectional area is no different than distal and proximal portions of the posterior interosseous nerve, so this flattening should not be mistaken for entrapment. When entrapment of the posterior interosseous nerve by the supinator muscle does occur, the nerve enlarges and becomes hypoechoic.

**Median Nerve**

Carpal tunnel syndrome, which results from median mononeuropathy at the wrist, is the most common median nerve entrapment syndrome, but the median nerve can be entrapped at the elbow. Entrapment of the median nerve has been described at several sites around the elbow, but the most common site is as the nerve passes between the humeral and ulnar heads of the pronator teres muscle, and this site can be evaluated with US.

**At the Pronator Teres**

To visualize the median nerve at the elbow, the nerve can first be identified in cross-section at the wrist and then traced to the elbow. Alternatively, with practice, the median nerve can be initially identified at the elbow, as it lies medial to the biceps tendon and the brachial artery. The nerve can be seen passing between the two heads of the pronator muscle, and when the nerve is pinched abnormalities can be detected with imaging. As with other focal neuropathies, US shows nerve enlargement and loss of normal echotexture.

**FIBULAR NERVE**

After CTS and UNE, fibular (previously called peroneal) neuropathy at the fibular head is the third most common entrapment syndrome.

**At the Fibular Head**

Obtaining a clear cross-sectional view of the fibular nerve at the fibular head with US is the key to evaluating this neuropathy. One method is to position the patient prone and start with a cross-sectional view of the distal sciatic nerve just proximal to the popliteal fossa, and then trace the fibular nerve distally as the sciatic nerve branches. The sciatic nerve is readily identified at this location because it lies just superficial to the popliteal artery. The other approach is to identify the head of the fibular bone, as it is hyperechoic and superficial. This can be performed with the patient prone (the author’s preference), lateral decubitus position, or supine. Once the fibular head is identified, the transducer is moved superiorly and medially until the fibular nerve is identified. With either technique, once the fibular nerve has been identified it should be traced from the fibular head through the popliteal fossa and to the point where it joins with the tibial nerve to form the sciatic nerve. The site of maximal enlargement should be identified and a cross-sectional area measured, with a normal area of the fibular nerve being less than 20 mm².

The use of NM US over the past 10 years has revealed an interesting potential finding in fibular neuropathy at the fibular head. Visser examined 28 individuals with isolated fibular mononeuropathy...
view. In addition to the tibial nerve, the tibialis posterior tendon, flexor digitorum longus tendon, flexor hallucis longus tendon, and posterior tibial artery pass through the medial ankle compartment and can be identified with US. In addition, several posterior tibial veins are also seen in this region. With EDX-confirmed TTS, enlargement of the tibial nerve can be identified. Nagaoka and Matsuzaki used US to assess 17 ankles with TTS prior to surgery and identified ganglion cysts in 13 and abnormal varicose veins in three as the cause of the tibial mononeuropathy.

**CONCLUSIONS**

Neuromuscular ultrasound is an excellent complement to EDX studies for the evaluation of suspected focal neuropathies. It allows quick, painless, and radiation-free identification of nerve enlargement and change in echotexture, and it can identify anatomic abnormalities intrinsic and extrinsic to the nerve that cannot be detected with electrodiagnosis alone. Padua and colleagues prospectively studied NM US in individuals with focal neuropathies and found that it altered the therapeutic options in 42.3% of cases in their electromyography (EMG) laboratory. While further investigation is needed, particularly for focal neuropathies other than CTS, the US technique is based on a large body of literature and is currently available to assist in the diagnosis of focal entrapment neuropathies.

**REFERENCES**


---

**TIBIAL NERVE**

Focal mononeuropathy of the tibial nerve as it passes posterior to the medial malleolus at the ankle has been termed tarsal tunnel syndrome (TTS). Unlike CTS, TTS is a controversial condition because it is often difficult to confirm the presence of a tibial mononeuropathy with EDX studies in suspected cases. Some believe it is overdiagnosed and others believe it is an underdiagnosed condition. Either way, the tibial nerve at the ankle can be easily imaged with NM US, and imaging can assist in establishing the diagnosis and possibly the etiology.

**At the Ankle**

With the patient supine and leg externally rotated, the medial ankle is imaged with the transducer placed to obtain a cross-sectional view.
Clinical Uses of Muscle Ultrasound

Craig Zaidman, MD
Assistant Professor
Neuromuscular Division, Division of Child Neurology
Department of Neurology
Washington University in St. Louis
St. Louis, Missouri

INTRODUCTION

Ultrasound (US) is a painless, noninvasive technique to identify skeletal muscle and nerve pathology. Its use was pioneered by Heckmatt and Dubowitz in the early 1980s in the evaluation of Duchenne muscular dystrophy (DMD). Since this time, studies of US imaging of skeletal muscle and nerve has expanded to include neuromuscular disorders of various etiologies. Ultrasound provides a practical and effective supplement to the physical examination in the evaluation of neuromuscular pathology. It can be performed at the bedside and allows for examination of multiple muscles quickly and without reliance on patient participation. This allows for a directed electrophysiologic examination and can be of assistance when selecting sites for biopsy. These qualities make US a practical and informative tool in the evaluation of the infant or child with a suspected neuromuscular disorder.

NORMAL MUSCLE

Normal muscle shows low echogenicity (mostly dark) on US. Interspersed within this low signal are multiple, homogenously distributed, well-defined brighter punctate or curvilinear bright areas. These represent the fibroadipose septa and tendinous fibrils interspersed among the muscle fibers. The myofascial fibrils coalesce near the myotendinous junction. At these areas, the echo intensity is increased, with higher anisotropy. For diagnostic purposes, it is best to avoid these areas and focus on the bulk of the muscle belly. The fascia around the muscle belly is brighter and thicker than the fibrous tissue in the muscle belly. At high magnification, multiple low-signal, dark honeycombed structures surrounded by a thin, medium intensity ribbon can be seen. These structures appear like individual muscle fibers; however, US does not have sufficient resolution to visualize individual muscle fibers. Rather, these areas are likely bundles of muscle fibers surrounded by brighter fibro-adipose tissue. Bone is very bright (highly echogenic) with a well-defined, crisp edge and casts a shadow on US deep the this boney reflection. Subcutaneous fat has similar echo intensity to muscle and is interspersed with poorly organized threads of brighter connection tissue.

The appearance of skeletal muscle on US changes with age. In infants, muscle is more echo-dark than in older children. At this age, there are few myofascial planes in the muscle parenchyma. By age 2-3 years, there are more myofascial planes than in an infant. By age 5, myofascial planes are seen in a homogenous pattern typical of the adult. After age 5, muscle echogenicity increases more gradually, if at all, through most of early adulthood. In later life, at about age 60 years, muscle echogenicity increases more rapidly with advancing age. These changes vary with muscle group and are most pronounced in the biceps brachii and quadriceps muscles, particularly in males. There is no difference in echogenicity between males and females until the teenage years. From this age forward, muscle in males is slightly darker on US than females.

Muscle bulk also changes throughout the lifespan and varies with age, gender, and muscle group. In the first 20 years of life, muscle thickness increases. As with muscle signal intensity, differences in gender begin in the early teenage years, with males having larger muscles than females. Until age 40 years, muscle thickness remains relatively stable in both genders. In older adults, muscle thickness in some muscles declines considerably.
thickness decreases in the quadriceps by 30% in women and 50% in men between ages 40-90. In the biceps brachii, similar but less severe declines (20-30%) are seen. In contrast, muscle thickness in adults in the sternocleidomastoid, tibialis anterior, and, in women, the forearm flexors remains more stable through the lifespan.

Muscle atrophy can also be assessed qualitatively by comparison of the thickness of muscle to subcutaneous fat; the typical ratio is approximately 2:1.11,12 While this method is helpful as a quick screening tool, the muscle to subcutaneous fat ratio can be misleading in obese patients and in infants as the thickness of the subcutaneous fat changes rapidly through the first year of life.9,13 Thus, assessment of muscle thickness must be interpreted using norms adjusted for patient characteristics, including age and body habitus.

NEUROMUSCULAR ULTRASOUND AS A DIAGNOSTIC TOOL

Myogenic weakness typically appears as homogenous increase in echo signal intensity with relatively preserved muscle bulk. In contrast, US in a patient with neurogenic weakness shows reduced muscle size with an increase in the subcutaneous fat to muscle ratio, and a pattern of streaky, increase echoes scattered heterogeneously within the muscle. Central hypotonia does not cause substantial alterations in the muscle signal intensity on US, although in the author’s experience disease can reduce muscle size. Using these criteria, US can play a role as a well-tolerated screening test and can be performed at the bedside without need for sedation or patient discomfort.

The sensitivity and specificity of diagnostic US for neuromuscular disorders has been studied in children and depends on the type and severity of disorder (reviewed by Pillen and colleagues14). In the evaluation of infants, in whom electrophysiologic evaluation can be technically challenging, US has similar sensitivities and specificities for identifying and characterizing neuromuscular pathologies to needle electromyography (EMG). In a study of 41 hypotonic infants aged 2-24 months, both qualitative US was highly concordant with needle EMG.12 Ultrasound and needle EMG both showed abnormalities consistent with myopathy in 6/6 infants with myogenic hypotonia and both showed neurogenic abnormalities in 16/16 infants with neurogenic hypotonia. In 17 infants with central hypotonia, both US and needle EMG were normal. Needle EMG and US results conflicted in only two infants; the muscle and nerve biopsy ultimately did not reveal a diagnosis.

In older children, qualitative US is also sensitive and specific for detecting neuromuscular disease. In a study of 134 patients with suspected neuromuscular disorders, qualitative US showed sensitivities of 81% and specificities of 96% in the assessment of any neuromuscular disorder.15 Ultrasound was less sensitive (71%) in identifying abnormalities in children with neuromuscular disorders under age 3.

In a study of 100 children with suspected neuromuscular disease, US was 78% sensitive and 91% specific for identifying any neuromuscular disease, was more reliable in children over age 3 years, and was least reliable in those under 1 year of age.16 Sensitivity and specificity varied with the degree of US abnormality. A mildly abnormal US is neither sensitive nor specific for a neuromuscular disorder. Only 7/13 children with a mildly abnormal US scan had a neuromuscular disorder. In contrast, all of the children with moderately or severely abnormal USs (Heckmatt grade III or IV) had a neuromuscular disorder. Similarly, nearly all (62/69) children with a normal US (Heckmatt Grade I) did not have a neuromuscular disorder. Quantitative US results in similar sensitivities for detecting neuromuscular disorders. A prospective study of quantitative grey-scale US analysis of 150 children referred for evaluation for neuromuscular disorders was 71% sensitive and 91% specific for identifying neuromuscular disorders.17 Again, the sensitivity of US in children younger than 3 years old was lower than in older children; however, specificity was 100%, with no false-positives in the younger age group.

Ultrasound has higher specificity than sensitivity in differentiating myopathic and neuropathic changes.15,17,18 Thus, abnormalities that distinguish the two pathologies such as muscle size and patterns of homogeneity are helpful when present but are less useful as a screening tool. Ultrasound was more specific than sensitive for detecting myogenic (92 versus 67%) and neurogenic (98 versus 77%) changes in 134 children studied by Brockmann and colleagues. The pattern of muscle involvement can also be helpful in identifying neuropathies, which affect the distal more than proximal muscles of the legs. A quantitative US study of 31 children with myopathic and 27 with neuromuscular disorders, brighter echoes and more atrophy in the legs than arms was 67% sensitive and 94% specific for identifying neurogenic disease.17 This type of analysis did not distinguish myopathic disease from non-neuromuscular conditions.17 In adults, a study comparing 145 healthy control subjects, 17 myopathic patients, and 15 neuropathic patients, brighter signal in the biceps brachii (increased grey-scale values) was 94% sensitive and 93% specific for myopathy while increased signal inhomogeneity was 100% sensitive and 93% specific for neuropathy.18 However, in children, this same quantitative approach did not distinguish between myopathic and neuropathic disease.19

The relationship between strength, function, and the degree of image abnormality may vary with differences in the underlying pathologies. In patients with muscular dystrophies, subclinical abnormalities and changes with disease progression have been detected on magnetic resonance imaging (MRI) and US, suggesting that imaging can be used to evaluate disease severity and progression.20-25 In contrast, the severity of imaging findings in children with mitochondrial myopathies26 or congenital muscular dystrophies3 may not correlate with disease severity of function. Additional studies of the neuromuscular pathologies are needed to compare patient strength, function, and prognosis with the degree of image abnormality.

ULTRASOUND ABNORMALITIES IN INHERITED MYOPATHIES

Hereditary myopathic disorders are a diverse group of pathologies that include the muscular dystrophies and the metabolic, mitochondrial, and congenital myopathies. They generally present with progressive, symmetric weakness more than atrophy of the proximal arms and legs and are classified by the pattern of inheritance, clinical findings, histopathology, and genetic abnormality. Muscular dystrophies are the most common type of myopathy and are characterized by findings on muscle biopsy of early and extensive
ULTRASOUND OF MUSCULAR DYSTROPHIES

Abnormal ultrasonography in neuromuscular disease was first described in males with DMD, an X-linked muscular dystrophy caused by mutations of the dystrophin gene. Much of the ongoing work in US of myopathies continues to be performed in patients with DMD and BMD, a less severe form of the disease. Ultrasound in DMD/BMD, as well as other muscular dystrophies characterized by increased connective tissue and fatty replacement of muscle, shows a diffuse and often marked increase in muscle echogenicity. The muscle shows a grainy, ground-glass like appearance, typically with preserved muscle bulk. In more advanced pathology, the muscle echoes are very bright and the attenuation greatly increased, resulting in a relatively darker appearance in the deep than superficial portion of the muscle and reduced or absent bone echoes.

The sensitivity of US to pathology associated with muscular dystrophy increases with age and disease severity. In DMD, US is often abnormal in children by the time they are toddlers. In one study, qualitative US was abnormal in nearly all (21/22) boys with DMD aged 3-7 years but in none of the seven boys aged 2-30 months.11 In a study of quantitative US, abnormalities were detected in 32/38 boys aged 1-11 years; all six normal USs were in children under age 6 years. In another quantitative US study, grey-scale levels were abnormally high in 10/11 boys with DMD ages 3-9 years but in only 1/2 boys aged 3 weeks and 7 months.17

In DMD and BMD, US measurement of muscle pathology varies with the severity of muscle pathology. Both US and strength abnormalities are more severe in the quadriceps than the biceps brachii.11,27 Ultrasound backscatter is higher and increases twice as much with age in patients with DMD than those less severely affected with BMD.28 Ultrasound signal abnormalities also increase with worsening strength and function in DMD29,30 and are more or similarly sensitive to changes in pathology over time as compared to functional measures of disease progression.31 Additional studies in the muscular dystrophies are needed to determine the sensitivity of US to detect effects of treatment.

Calf enlargement is a common clinical finding in patients with muscular dystrophy and other neuromuscular disorders. Calf enlargement can be either associated with normal or increased echogenicity and is a common and nonspecific finding in neuromuscular disorders.30 Interestingly, when there is severe fatty infiltration in the calf muscle, the US actually appears dark, similar to subcutaneous fat. As muscle pathology from increased fat typically results in brighter US echoes, the US appearance of severe pathology in the calf may be misleading.

ULTRASOUND OF MITOCHONDRIAL MYOPATHIES

Mitochondrial myopathies are a heterogeneous group of neuromuscular disorders that can affect multiple organ systems. Symptoms are related to dysfunction of energy metabolism and include weakness and exercise intolerance. Skeletal muscle US in mitochondrial myopathies can be abnormal but is less sensitive than in other myopathies. In 14 children with mitochondrial myopathies, US assessment was concordant with histologic findings in only eight patients, including one myogenic, two neurogenic, four nonspecific, and one normal pattern on both US and histology.15 In a prospective study of quantitative US in 53 children with suspected mitochondrial disorders, only 7/28 children with definite or probable mitochondrial disorders had abnormal US echogenicity.26 An additional six children had only borderline US abnormalities. Echo intensity in the 28 subjects with mitochondrial disorders did not correlate with strength or the percentage of intramuscular fat or connective tissue, but did increase with age. Six of the eight children with abnormal USs were over 5 years old. Ultrasound is thus not sufficiently sensitive to use as a screening test for mitochondrial disorders but does detect pathology that is independent, and complimentary, to functional and histologic results.

OTHER CONGENITAL AND HEREDITARY MYOPATHIES

The congenital and hereditary myopathies are comprised of a large group of heterogeneous disorders and phenotypes. Radiologic studies of specific congenital or hereditary myopathies are often limited to small case series. Although relatively few radiologic studies of specific congenital or hereditary myopathies have been reported, US and MRI can detect the presence and pattern of skeletal muscle pathology in patients with these disorders (reviewed by Pillen and colleagues19 and Mercuri and colleagues8). Certain patterns of muscle involvement can direct focused genetic testing or guide selection of a muscle for biopsy. However, as with many neuromuscular disorders, the patterns and degree of muscle involvement on US varies within a genotype and with disease severity. The heterogeneity in phenotype and the small number of reported cases make it difficult to determine the specificity of a pattern of muscle involvement on US or MRI. For instance, selective involvement of the medial gastrocnemius, with sparing of the lateral gastrocnemius, has been described in several inherited myopathies, including EDMD/LGMD-1B (Lamin A/C), LGMD 1C (Caveolin), LGMD-2C (Dysferlin), LGMD-2L (ANOS5), myotonic dystrophy (DM1), and central nuclear (dynamin 2 centronuclear myopathy [DNM2]).

Nonetheless, a few case reports in hereditary myopathy report unique, specific radiologic patterns. Imaging of Bethlem and the more severe Ullrich congenital muscular dystrophies, both collagen type VI disorders characterized by proximal weakness and contractures, shows a unique “outside-in” pattern of muscle involvement.32-34 These studies show relative sparing of the central portion of the muscle belly with involvement of the outer rim of muscle in a concentric pattern. This pattern is best described in the rectus femoris of patients with Bethlem myopathy, and it is termed the “central shadow” sign. This “shadow” seen on US refers not to darkening but rather increased echogenicity and thickening.
along the central fascia (a normally thin, bright, band that vertically divides the rectus femoris from the superficial fascia to the middle of the muscle belly). Similar but more severe findings are seen in Ullrich myopathy. In a study of nine patients with Ullrich myopathy,34 the involved concentric rim with central sparing was best seen in the vastus lateralis and was present but less distinct in the rectus femoris. Increased signal is also seen in the connective tissues between the soleus and gastrocnemius in both Bethlem and Ullrich myopathies, but was more distinct in Bethlem myopathy. In contrast, EDMD, which shares some clinical features with collagen VI disorders, shows more diffuse thigh and selective medial gastrocnemius involvement15 and does not show the “outside-in” pattern or central shadow sign.

Another unique pattern of pathology on US has been reported in six patients with hereditary inclusion body myositis with homozygous GNE mutations.36 These patients showed selective involvement of the rectus femoris with relatively spared vastus medialis, lateralis, and intermedialis muscles. Additionally, these patients showed areas of increased echogenicity within the central portion of the hamstring muscles, producing a target-like brightness inside a rim of relatively spared hypoechoic muscle. This study also showed more severe atrophy of the anterior foreleg compared to the calf and more severe involvement of the hamstring muscles compared to the anterior thigh.

Patients with acid-maltase deficiency (Pompe disease) have a specific pattern of muscle involvement that can be detected using US. This pattern includes sparing of the superficial but not deep portion of the biceps brachii, differential involvement of the biceps brachii but sparing of the triceps brachii, and differential involvement of the vastus intermedius more than the rectus femoris.37 The degree of increased echogenicity in the muscle of a patient with acid-maltase deficiency correlated with strength. One distinguishing feature of this study in patients with acid-maltase deficiency was that findings were compared to patients with other myopathies. This demonstrates the potential for imaging studies in neuromuscular disease to identify patterns of muscle involvement that are specific and could direct further testing. Additional radiologic imaging studies are needed to describe more myopathies with specific patterns of muscle involvement.

**ULTRASOUND, FASCICULATIONS, AND MOTOR NEURON DISEASE**

Ultrasound is particularly useful in the identification of fasciculations, which are more frequent and widespread in patients with neuromuscular disorders.38,39 Ultrasound has been shown to detect fasciculations with higher sensitivity than the clinical examination or needle EMG.38,40,41 Fasciculations are rapid (0.2-0.5 s) contractions of focal areas of muscle that deform the surrounding areas and occur at random intervals and areas. Occasional fasciculations (≤ 2 per 10 s observation) are detectable on US in as many as 8-43% of healthy subjects, are more common in older patients, but rarely occur in muscles proximal to the knee or in the arm.38,42 In contrast, fasciculations were very common (85/92 [92%]) in subjects with spinal muscular atrophy (SMA), Charcot-Marie-Tooth, or motor lumbar radiculopathies43 and in 24/25 (96%) of subjects with early amyotrophic lateral sclerosis (ALS).39 Most patients with ALS in this study had on average more than four fasciculations/10 s. The presence of fasciculations can facilitate the diagnosis of ALS and is augmented by incorporating US into the evaluation.43,44 In practice, one method is to screen for fasciculations for 10 s in each imaged muscle and comment on their presence if they are frequent (> 2/10 s) in multiple muscles (especially in muscles above the knee as this is uncommon in normal subjects) or if they occur in the presence of other abnormalities.

Two studies using US detected fibrillations as small amplitude muscle movements.45,46 Fibrillations appear as small amplitude, irregularly recurring movements within the muscle without other movement of the surrounding tissues or muscle.46 Detection of fibrillations requires a high frame rate. This can be easily achieved by using the zoom function. Detection of fibrillations using US did not reach high levels of sensitivity or specificity.47 This is likely due to several pitfalls that can mimic the small amplitude movements seen from fibrillations. These include movement of tissue due to nearby arterial blood or artifact usually encountered along the lateral edge of the image or at a tissue-to-bone interface. Thus, the appearance of small amplitude movements on US should be interpreted in the appropriate clinical and electrodiagnostic context.

Other abnormalities that can be detected in motor neuron disease using US include those in muscle echogenicity and size. In motor neuron disease, muscle bulk is decreased and muscle echogenicity increases in some areas of the muscle while other areas appear normal.48,49 In ALS, the rate of increase in muscle echogenicity over time is an independent predictor of survival.50 In children with SMA, muscle echogenicity is highest in children with the most severe weakness.51 These studies demonstrate the potential for quantitative muscle measurements using US to inform prognosis and assess in disease progression and severity.

**REFERENCES**


INTRODUCTION

High frequency ultrasound is an emergent imaging musculoskeletal modality. It facilitates the performance of diagnostic evaluations as well as interventional procedures as reported in the literature from multiple specialties. This is a growing field that allows physicians to deliver patient care with demonstrated benefits, including safety, availability and cost, among others.\textsuperscript{1,2}

High frequency ultrasound’s ability to depict neuromuscular structures and provide guidance for interventional procedures with high resolution and accuracy enables physicians to perform procedures that previously were performed blindly or in some cases utilizing imaging modalities such as computed tomography (CT) with its inherent limitations. The recent advances in high frequency ultrasound technology allow physicians to diagnose and treat patients with a greater degree of confidence.\textsuperscript{3}

The literature shows that specialties such as radiology, rheumatology, physical medicine, neurology, anesthesiology, sports medicine, podiatry, and others utilize high frequency ultrasound as a diagnostic and interventional tool.\textsuperscript{4a,4b} The number of articles, websites, and other public internet based media about this technology continues to grow.

One of the most common procedures performed is peri- and intra-articular aspiration and injection. Table 1 shows the success rate when ultrasound is used to guide the aspiration of different joints. This shows a 68% failure rate with blinded aspirations.

<table>
<thead>
<tr>
<th>Aspiration site</th>
<th>Conventional</th>
<th>US guided</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shoulder</td>
<td>4 (1)</td>
<td>2 (2)</td>
</tr>
<tr>
<td>Elbow</td>
<td>8 (3)</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Wrist</td>
<td>4 (1)</td>
<td>—</td>
</tr>
<tr>
<td>Hip</td>
<td>—</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Knee</td>
<td>10 (4)</td>
<td>19 (18)</td>
</tr>
<tr>
<td>Ankle</td>
<td>5 (1)</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Small joints (CMC, MTP, PIP)</td>
<td>1 (0)</td>
<td>4 (4)</td>
</tr>
<tr>
<td>Soft tissue (bursa, tendon, sheath, cyst, wound)</td>
<td>—</td>
<td>4 (4)</td>
</tr>
<tr>
<td>Total</td>
<td>32 (10)</td>
<td>32 (31)</td>
</tr>
</tbody>
</table>

CMC = carpometacarpal, MTP = metatarsophalangeal, PIP = proximal interphalangeal, US = ultrasound

From Balint and colleagues.\textsuperscript{19}

Joints are being referred to here, given the frequency of procedures performed and size of the target to evaluate the improvement in accuracy of ultrasound-guided procedures such as aspiration and injection and at least the short-term benefits obtained.

---

Musculoskeletal Ultrasound Guided Procedures

Victor H. Flores, MD
Private Practice
Physical Medicine Associates,
Fort Worth, Texas

Due to unforeseen circumstances, the manuscript printed here is from a prior talk by Dr. Flores. It will cover a portion of his presentation and will provide additional information.
Smaller structures such as peripheral nerves in most cases are more difficult to identify by palpation or localization of musculoskeletal landmarks such as bone, tendon, or muscle. The use of high frequency ultrasound is becoming a more frequently used tool for evaluation, temporary anesthetic regional blocks, and in some cases the injection of steroids as in the treatment of painful neuromas.9

Current literature includes studies that have evaluated the accuracy and compared the effectiveness when interventional perineural-guided ultrasound is used, as in the case of the lateral femoral cutaneous nerve.10 These studies support ultrasound guidance as an effective modality, although the literature is more difficult to evaluate given the multiple variables including anatomical areas, dose and type of anesthetic, electrical stimulation versus palpation, lasting effect, and motor versus sensory response, among others.

One particular study concluded that ultrasound improves efficacy of peripheral nerve block compared with techniques that utilize a peripheral nerve stimulator (PNS) for nerve localization. Larger studies are needed to determine whether or not the use of ultrasound can decrease the number of complications such as nerve injury or systemic local anesthetic toxicity.11 The trend seems to indicate that high frequency ultrasound has the potential to become the standard of care for peripheral nerve block procedures.

Ultrasound guided muscle biopsy has been reported in the literature as a useful procedure, facilitating diagnosis in acute muscular disease. It provides results comparable with those of open surgical biopsy in acute muscular disease and it also may be of help in chronic disease.12 This particular study makes a cost comparison of open biopsy, being approximately three times more expensive than percutaneous ultrasound guided. It also considered the time of the procedure being reduced from 2-3 hours in the case of an open biopsy to 15-20 minutes when ultrasound guided was performed. The use of ultrasound guidance for botulin injections has also found to be of significant value in clinical practice.13 Dry needling of trigger points in the cervicothoracic musculature may reduce the potential of complications such as a pneumothorax.14

**GENERAL PRINCIPLES**

These are some of most important factors to consider when guided ultrasound guided procedures are performed.

Knowledge of the basic ultrasound physics, equipment, settings, and techniques are required to obtain the best possible image of the target structure and needle.

Documentation by static images and video clips is essential for patient followup (medicolegal as well as billing). Digital formats such as JPEG or DICOM are options that can facilitate archiving these images. Printed static images can also be used but their size and lifetime may present some limitations.

It is imperative to have a detailed knowledge of anatomy and sonoanatomy of the area to be treated. Of no less importance is to perform a complete diagnostic study so that a clear indication for the procedure to be performed is established. Other occult pathologies need to be considered and ruled out so that the diagnosis is as accurate as possible, decreasing the potential of unexpected findings. Awareness of normal anatomical variants is also important.

Neurovascular structures and tendons should be identified to prevent iatrogenic lesions. The use of Doppler can be helpful not only for the identification of vascular structures but also in some cases to visualize the needle.

It necessary to evaluate the potential contraindications such as anticoagulation, bleeding disorder, skin infection, etc. Informed consent should be obtained and the benefits and potential risks discussed.15 Practitioners should recognize their skills and limitations in performing these procedures as well as those that the equipment could present. For instance, it would not be advisable to perform procedures on targets such as small peripheral nerves with less than equipment of 12 MHz or use a large footprint probe for an anatomical area in which its positioning would limit the field of view.

It is recommended to plan the procedure including all the steps already mentioned. In addition to the previously listed recommendations, the material to be utilized—the needle length and gauge, anesthetic and steroid to be used, etc.—should be determined in advance. If a biopsy is planned, it is important to become familiar with the type of needle to be used and how the TRU-CUT is operated. Whether this material comes in a tray or as individual items, they should be ready and within easy reach, particularly if these procedures are performed without the help of an assistant.

Following principles of aseptic technique including the use of a sterile envelope or sleeve is advised by most of the experts in this field. This includes the use of sterile gel at the area of needle insertion. Compliance with the standards of practice of the institution or office policy manual where the procedure is being performed is recommended. See Figure 1.
The patient should be preferably in a supine and comfortable position in case a vasovagal reaction occurs. It is also important for the physician performing the procedure to adopt an ergonomic position which includes optimal location of the ultrasound machine and direct view of the monitor.

With all the above in mind and after the diagnosis is confirmed, the path approach to the already-identified target becomes of great importance. The frequency of the transducer and its footprint size needs to be selected as well as the frequency that would better fit the anatomical area where the procedure is to be performed, so that adjustments to the transducer position will provide optimal visualization of the needle at all times during the intervention within an optimal field of view.

**TECHNIQUE**

The visualization of the needle and target structure is the basic goal of ultrasound-guided procedures so that the needle can be advanced following the shortest possible and safest path to the target. The intervention can be achieved by indirect and direct techniques.

**Indirect**

Indirect ultrasound is utilized to identify the target structure and approach considering the surrounding structures and depth. The procedure in itself is not real-time guided but based on measurements and markings on the skin of the optimal site for needle insertion. See Figure 2. For confirmatory and documentation purposes an image can be obtained after the needle is in the expected position. See Figure 3.

![Figure 2](image2.png) **Figure 2** Markings on the skin of the optimal site for needle insertion for indirect ultrasound.

![Figure 3](image3.png) **Figure 3** Needle visualized after insertion using indirect ultrasound.
Direct

This technique uses real-time ultrasound that allows the physician to visualize the needle as it moves towards the target. The ability to depict the needle will depend on the angle of the needle in relation to the ultrasound beam. The long axis or longitudinal approach is in general recommended over the short axis or transverse approach. Ideally, the needle should be parallel to the transducer so the ultrasound view will be depicted as a straight hyperechoic line with the characteristic artifact called a comet tail. The bevel can be identified and should be maintained in the field of view at all times during the advancement of the needle. See Figure 4, top.

There are available needle guides that adapt to the transducer to facilitate the procedure but there appears to be no literature available to evaluate the benefit of their use with the exception of breast biopsy. The described “free-hand” technique basically is the procedure in which the transducer is held with the nondominant hand to allow the handling of the needle with the dominant hand, maintaining a sterile technique.

The target must be identified and then the skin marked at both ends of the transducer so that after the skin is disinfected one can easily find the target. See Figure 5. The insertion point of the needle will depend on the depth of the target. Deeper targets will require an entry site located farther away from the transducer to prevent a steeper angle in relation to the ultrasound beam that would cause limitations visualizing the needle.

After using local anesthesia penetrate the skin approximately 1 cm and localize the needle, advance under real time guidance. Injecting an anesthetic or normal saline can help by performing hydrodissection as the needle is advanced always under real time visualization. If the tip of the needle cannot be seen, DO NOT advance the needle. Sometimes it will be necessary to withdraw and redirect the needle or adjust the position of the transducer.

The short axis or transverse approach will place the needle perpendicular to the transducer and it will appear as a hyperechoic dot. This is not a recommended approach, although it may be the only option in some areas, such as superficial joints. When using this approach moving the tip in and out may be needed to find the angle that will allow reaching the target. As in the long axis approach the bevel should be in the field of view at all times. See Figure 4, bottom.

The so-called “heel-toe” maneuver can be used to position the transducer parallel to the needle. Also, the oblique stand-off technique can be used by applying a thicker layer of gel under the transducer at the needle entrance, filling the gap between the skin and the end of the transducer. New software is also available that allows depiction a needle at 45 degree angle in relation to the ultrasound beam. See Figure 6.
Adjustment in the positioning of the transducer or needle may be needed for the visualization of the needle. It MUST be remembered that the tip of the needle should be visualized at all times and that one should move or adjust the positioning of either the transducer or needle but NEVER move both at the same time.

Using beam steering or Doppler also can help localize the needle without moving it. The larger needles are more reflective and better visualized. Jiggle, rotating the bevel, and a stylet can improve the visualization of the needle. There are echogenic needles available but for the most part a regular needle should be visualized following proper technique.

Figure 7, top and middle, shows an injection of the median nerve at the carpal tunnel. The bottom of Figure 7 shows 15 days post-injection with 30 mg of triamcinolone. Most of these injections are performed blindly. The advantage of performing this procedure under ultrasound guidance is in case of normal variants, such a bifid median nerve or after carpal tunnel release, because the location of the nerve cannot be anticipated. The ulnar side approach and its advantage has been described.

COMPLICATIONS

The most frequent complications to consider are bleeding, hematomas, and infection. Observing general precautions should minimize the potential risks.

BILLING AND CODING

Documentation is a must when performing these procedures. A report should be generated that includes the diagnosis and the indication for ultrasound guidance. Description of the procedure itself should be included. Permanent pictures and video, if possible, also is necessary. The procedure code to be utilized is 76942-ultrasound guidance.

SUMMARY

Ultrasound allows physicians to perform guided neuromuscular interventions in a safe and efficient way. A diagnostic evalu-
tion MUST be performed prior to a guided procedure. Planning the approach and anticipating the material to be utilized will increase the success of the procedure. Experience in neuromuscular ultrasound is essential and cannot be emphasized enough prior to adopting this new tool for interventional purposes.

If the target structure and the instrument is available for a particular percutaneous procedure can be depicted by ultrasound in real time, does not increase significantly or even reduces the cost of the procedure, has demonstrated different benefits, and is likely to improve overall patient care, why not use it? Perhaps the time for physicians to develop or improve their skills utilizing this new imaging tool has arrived. Albeit, further studies are needed to evaluate long-term outcomes.

REFERENCES

Ultrasound CME Questions

1. The following pre-operative findings are associated with better outcomes after carpal tunnel decompression:
   A. Longer life.
   B. Smoking cessation.
   C. Normal power in abductor pollicis brevis.
   D. Longer duration of symptoms.

2. The following pre-operative measures of severity are linearly correlated with outcomes after carpal tunnel surgery:
   A. Distal median motor latency to abductor pollicis brevis.
   B. Functional impairment assessed using a subjective scale for activities of daily living.
   C. Median nerve conduction velocity between palm and wrist.
   D. Severity of symptoms assessed using a subjective scale.

3. Ultrasound imaging of peripheral nerve:
   A. Can demonstrate continuity of axons across a nerve injury site.
   B. Shows greater enlargement of the ulnar nerve at the elbow in patients who do less well with treatment.
   C. Shows greater enlargement of the median nerve at the wrist in carpal tunnel syndrome patients who respond well to local steroid injection.
   D. Shows greater enlargement of the median nerve at the wrist in carpal tunnel syndrome patients who report no benefit from wrist splinting.

4. The duration of remission of symptoms after steroid injection for carpal tunnel syndrome:
   A. Is significantly longer in patients with milder subjective symptoms.
   B. Is significantly longer in patients with more severe electrophysiological abnormalities.
   C. Is always less than 1 year.
   D. Is significantly longer in those who report complete resolution of symptoms shortly after injection.

5. In trials of treatment for carpal tunnel syndrome:
   A. A single local steroid injection has a higher long-term success rate than surgery.
   B. Acupuncture has been shown to be effective at relieving paraesthesiae in a meta-analysis.
   C. Splinting is more effective than local steroid injection.
   D. Non-steroidal anti-inflammatory drugs are no better than placebo.

6. Which of the following statements is correct regarding movement of the ulnar nerve during elbow flexion?
   A. Dislocation of the ulnar nerve can be detected with ultrasound, but subluxation cannot.
   B. Subluxation of the ulnar nerve is only seen in individuals with ulnar neuropathy at the elbow.
   C. Dislocation of the ulnar nerve is more common than subluxation.
   D. Subluxation and dislocation of the ulnar nerve occur in both healthy individuals and those with ulnar neuropathy at the elbow.

7. In a radial mononeuropathy at the spiral groove (Saturday night palsy), which of the following ultrasonographic features is most often present?
   A. Radial nerve transection.
   B. Normal radial nerve area.
   C. Hypervascularity within the radial nerve.
   D. An intraneural ganglion cyst.

8. Intraneural ganglion cysts are found in what percentage of cases of isolated fibular neuropathy at the fibular head?
   A. 0%.
   B. 5%.
   C. 18%.
   D. 52%.

9. All of the following have been noted during neuromuscular ultrasound for the evaluation of tarsal tunnel syndrome EXCEPT:
   A. Hypervascularity within the tibial nerve.
   B. Tibial nerve enlargement.
   C. Extraneural ganglion cysts.
   D. Tibial vein varicosities.

10. Failure to detect ulnar nerve subluxation with elbow flexion can result in which of the following:
    A. Falsely elevated motor amplitude.
    B. Falsely elevated motor conduction velocity across the elbow.
    C. Falsely elevated sensory amplitude.
    D. False positive nerve conduction studies for the diagnosis of ulnar neuropathy at the elbow.
11. The “central shadow” sign:
   A. Refers to darkening of the ultrasound signal in the center of the muscle.
   B. Is seen in myopathies from Collagen VI disorders (Bethlem/Ulrich).
   C. Is caused by pathology in the superficial rim of muscle.
   D. B and C.

12. Ultrasound echogenicity in normal skeletal muscle
   A. Increases linearly throughout the lifespan.
   B. Increases most rapidly in both men and women after age 60.
   C. Is different in prepubertal males and females.
   D. Changes with age at the same rate in all muscles.

13. Skeletal muscle ultrasound in children
   A. Has higher sensitivity than specificity in differentiating myopathic and neuropathic changes.
   B. Is more sensitive in children under age three than older children for detecting neuromuscular disorders.
   C. Is more sensitive for detecting mitochondrial disorders than muscular dystrophies.
   D. Yields similar results to EMG in evaluating hypotonic children ages 2-24 months.

14. Ultrasound of skeletal muscle in boys with Duchenne muscular dystrophy
   A. Can be normal in children under age 3.
   B. Shows more severe pathologies with increasing age.
   C. Shows brighter signal in children with weaker strength and function.
   D. All of the above.

15. Fasciculations are
   A. Detected with high sensitivity using ultrasound than electromyography.
   B. Are often seen in patients with motor neuron disease.
   C. Can be present in normals but are infrequent and often limited to muscles distal to the knee.
   D. All of the Above.