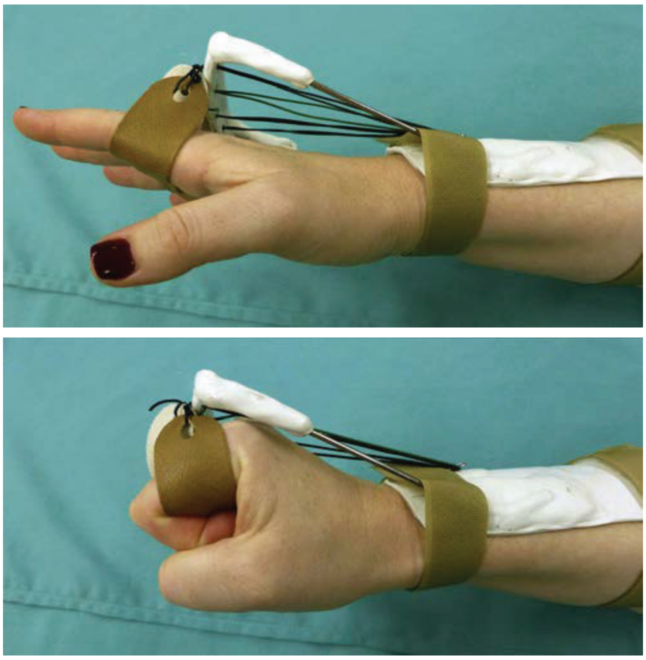

FIG. 26.3 Radial palsy splint with metacarpophalangeal joint extended and flexed.

Procedures

Local anesthetic blocks or injections of hydrocortisone can be used,[22] but are rarely necessary and have shown only temporary symptomatic relief. Lateral epicondylitis may mimic posterior interosseous nerve entrapment at the elbow. When lateral epicondylitis does not respond to conservative treatment, including injections of the lateral epicondyle, a diagnostic and therapeutic radial nerve injection at the elbow may be indicated.[23]

Technology

There is no specific technology for the treatment or rehabilitation of this condition.

Surgery

Surgical decompression may be required for patients who do not respond to conservative treatment or patients with severe nerve injury. Radial tunnel release has been utilized for compression neuropathies of the posterior interosseous nerve.[24] Surgical intervention for anastomosis may be indicated in cases of complete radial injury (neurotmesis). Tendon transfers may be considered in these instances if the surgery is not performed or is not successful.[25,26] Care must be exercised to avoid the radial sensory branch during operations involving the wrist.[22] Surgery has been noted to be less successful if there are coexisting additional nerve compressions or lateral epicondylitis or if the patient is receiving workers' compensation.[27]

Potential Disease Complications

Patients with incomplete recovery may suffer significant functional loss in the upper extremity. Like any patient with nerve injury, they are at risk for development of complex regional pain syndrome (reflex sympathetic dystrophy).[28] Contractures and chronic pain may develop as well.

Potential Treatment Complications

There are inherent risks with any surgery, including failure to correct the problem, infection, additional deformity, and death. Any injection or surgery involving the wrist should avoid the superficial radial sensory nerve, as this could cause additional paresthesias or dysesthesias.

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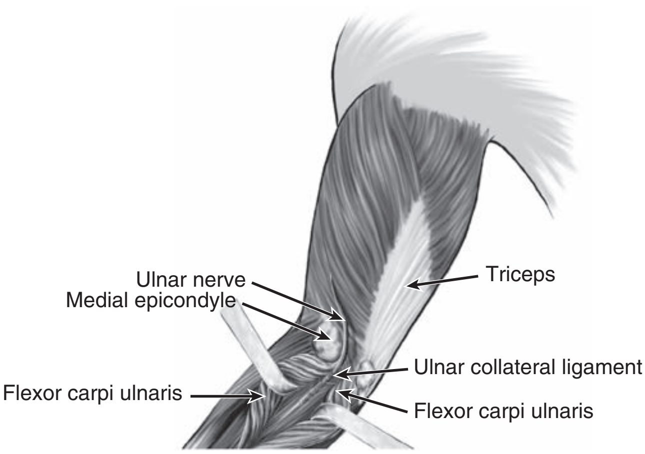

FIG. 27.1 The cubital tunnel. *(From Bernstein J, ed.* Musculoskeletal Medicine. *Rosemont, IL: American Academy of Orthopaedic Surgeons; 2003.)*

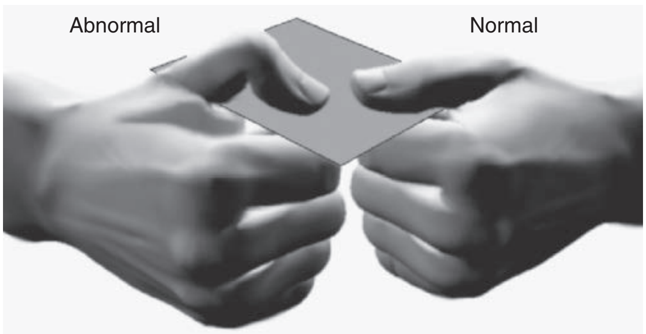

FIG. 27.2 Froment sign. Note prominent atrophy of the intrinsic muscles. *(From Weiss L, Silver J, Weiss J, eds.* Easy EMG. *New York: Butterworth-Heinemann; 2004.)*

patient should be tested for Froment sign. Here, a patient is asked to grasp a piece of paper between the thumb and radial side of the second digit. The examiner tries to pull the paper out of the patient's hand. If the patient has injury to the adductor pollicis muscle (ulnar innervated), the patient will try to compensate by using the median-innervated flexor pollicis longus muscle (see Fig. 27.2).

Functional Limitations

The patient with ulnar neuropathy at the elbow may have poor hand function and complain of dropping things or clumsiness. There may be difficulty with activities of daily living, such as dressing, holding a pen, or using keys.

Diagnostic Studies

Electrodiagnostic studies can help identify, localize, and gauge the severity of an ulnar nerve lesion at the elbow. The findings of abnormal spontaneous potentials (fibrillations and positive sharp waves) in ulnar innervated muscles on needle electromyographic study indicate axonal damage and portend a worse prognosis than with injury to the myelin only. Slowing of the ulnar nerve across the elbow or conduction block (a drop in compound motor action potential amplitude across the elbow) indicates myelin injury, which has a better prognosis.[7] These studies can also identify other areas of nerve compression that may accompany ulnar neuropathy at the elbow. Several studies using ultrasound have shown an increased cross-sectional area of the ulnar nerve in patients with ulnar neuropathy at the elbow.[8,9] The nerve may appear swollen proximal to areas of compression.[10]

Magnetic resonance neurography may play a role in the evaluation of ulnar neuropathy at the elbow.[11] Radiographs of the elbow with cubital tunnel views can be obtained if fractures, spurs, arthritis, and trauma are suspected. In rare cases, magnetic resonance imaging[12] with arthrography may be used to assess for tears in the ulnar collateral ligament or soft tissue disease.

Differential Diagnosis

Ulnar neuropathy at a location other than the elbow
C8-T1 radiculopathy
Brachial plexopathy (usually lower trunk)
Thoracic outlet syndrome
Elbow fracture
Elbow dislocation
Medial epicondylitis
Carpal tunnel syndrome
Ulnar collateral ligament injury
Soft tissue disorders at the elbow

Treatment

Initial

Treatment initially involves relative rest and protecting the elbow. Elbow pads or night splinting in mild flexion may be beneficial. Treatment should be directed at avoidance of aggravating biomechanical factors, such as leaning on the elbows, prolonged or repetitive elbow flexion, and repetitive valgus stress in throwing. Nonsteroidal anti-inflammatory drugs may also be prescribed.

Rehabilitation

Successful rehabilitation of ulnar neuropathy at the elbow includes identification and correction of biomechanical factors. This may include workstation modifications to decrease the amount of elbow flexion, substitution of headphones for telephone handsets, and use of forearm rests. Often, an elbow pad can be beneficial; the pad protects the ulnar nerve and keeps the elbow in relative extension. A rehabilitation program should include strengthening of forearm pronator and flexor muscles. Flexibility exercises should be instituted to maintain range of motion and to prevent soft tissue tightness. Advanced strengthening, including eccentric and dynamic joint stabilization exercises, can be added.[13]

Procedures

Procedures are not typically performed to treat ulnar neuropathy at the elbow.

Technology

There is no specific technology for the treatment or rehabilitation of this condition.

Surgery

If conservative management has failed or if significant damage to the ulnar nerve is evident, surgery may be considered.[14-16] The type of surgery depends on the area of ulnar nerve injury and may involve release of the cubital tunnel, ulnar nerve transposition,[17] decompression of the ulnar nerve (open or arthroscopic),[18,19] subtotal medial epicondylectomy,[20,21] or ulnar collateral ligament repair. Simple decompression and decompression with transposition have been shown to be equally effective in idiopathic ulnar neuropathy at the elbow,[22] although decompression with transposition has been associated with more wound infections.[23]

Potential Disease Complications

If ulnar neuropathy at the elbow is left untreated, complications may include hand weakness, poor coordination, intrinsic muscle atrophy, sensory loss, and pain. In addition, flexion contractures and valgus deformity may develop at the elbow.[13]

Potential Treatment Complications

The results of surgery depend on the extent of ulnar nerve compression, accuracy of identifying the site of compression, type of procedure, thoroughness of compression release, comorbid factors, degree of prior intrinsic muscle loss, and previous sensory loss.[13,24-27] Nonsteroidal anti-inflammatory drugs may cause gastric, hepatic, or renal complications.

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Functional Limitations

Functional limitations result from deficits in finger control, with power grasp affected if the ulnarly sided tendons are involved, and precision grasp affected if the radially sided tendons are involved. The patient may present with inability to button shirts, to pinch small objects, or to firmly grasp objects.

Diagnostic Studies

Obtain anteroposterior and lateral radiographs of the involved fingers when there is the possibility of bony fracture, dislocation, or foreign body retention in the soft tissues. Diagnostic imaging for direct tendon visualization can be performed with ultrasonography or magnetic resonance imaging (MRI). Ultrasonography is an inexpensive alternative to MRI and is particularly helpful for detection of foreign bodies, evaluation for partial tendon injuries, and dynamic evaluation of flexor tendon function.[8]

Differential Diagnosis

Partial tendon laceration
Anterior interosseous nerve injury
Trigger finger (stenosing tenosynovitis)
Median nerve injury

Treatment

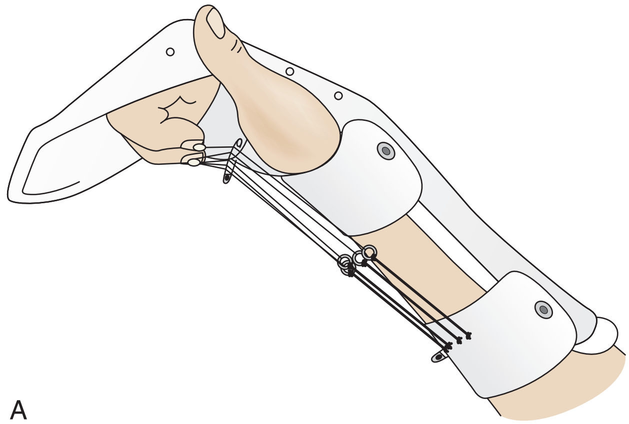
Initial

Surgical intervention is almost always required for flexor tendon injuries, irrespective if injury is open or closed.[1,4,5,7] Appropriate wound care should be provided with any open injury. If surgical referral is delayed, repair of superficial wounds and protection of the affected finger in a bulky dressing should be performed.

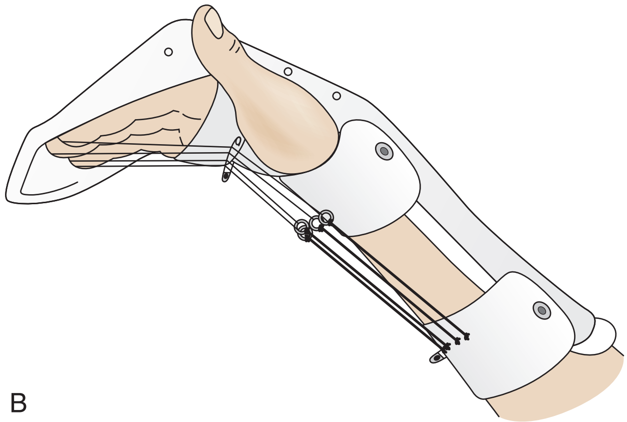
Emergent flexor tendon repair is indicated in the setting of coexisting neurovascular damage that requires microvascular repair or reconstruction. Otherwise, surgical correction of the flexor tendon injury should be performed within the first days of injury up to 6 weeks post injury if the vincular system remains intact to ensure continued nutrition to the tendon.[1] Delayed repair past this time period risks tendon retraction within the sheath, making surgical repair difficult.[1,7]

Rehabilitation

Advances in suture material and surgical technique have improved the strength of repaired flexor tendons, providing for earlier rehabilitation.[1,7] Historically, repaired fingers were placed in immobilization splints for up to 2 months. This led to adhesion formation with loss of motion and function.[9] The initiation of early protected-motion protocols in the past three decades has markedly changed the postoperative rehabilitation of repaired tendons.[9,10] Early motion protocols have been shown to limit development of postoperative adhesions and thus improve range of motion and overall outcomes compared to static splinting.[1,11]



A



B

FIG. 31.3 (A) Dorsal dynamic protection splint; fingers in resting position. (B) Dorsal dynamic protection splint; active extension exercises.

Early motion protocols for postoperative flexor tendon repair can be early passive mobilization or early active mobilization.[2,11] A combination of both can be used. Early passive mobilization can be manually performed, by therapist or patient, or via dynamic flexion traction device.[2] This can usually begin within 24 hours of surgical repair. Early active mobilization protocols employ active contraction of the involved flexor through a predicated flexion range of motion.[2] This may begin several days following repair, pending an appropriate repair technique was utilized and the surgeon is comfortable with the level of rehabilitation advancement.

Rehabilitation schemes for repaired flexor tendons are essentially the same for all zones. Immediately postoperatively, the hand is placed in a protective dorsal splint that allows dynamic passive flexion. The splint creates 20 to 30 degrees of wrist flexion, and 20 to 50 degrees of metacarpophalangeal (MCP) joint flexion, depending on zone of injury.[12] A dorsal hood extends to the fingertip level, allowing PIP and DIP joint extension to 0 degrees. All fingers are held in flexion by dynamic traction applied by rubber bands originating from the proximal forearm with a pulley at the palm and attachment to the fingernails (Fig. 31.3A).

The patient is typically seen by the therapist 24 to 48 hours after surgical repair. Dressings are changed, edema control measures are initiated, and rehabilitation goals are

CHAPTER 32

Hand and Wrist Ganglia

Michael F. Stretanski, DO, AME

Synonyms

Carpal cyst
Synovial cyst
Mucous cyst
Intraosseous cyst

ICD-10 Codes

M67.40	**Ganglion of joint, unspecified site**
M67.40	**Ganglion of tendon sheath**

Definition

Hand and wrist ganglia account for 50% to 70% of all hand masses. A ganglion is a benign, mucin-filled cyst found in relation to a joint, ligament, or tendon, first appreciated by Hippocrates. Hand and wrist ganglia are usually dorsal surface, asymptomatic masses, which often present with cosmetic rather than functional complaints. They are usually benign masses occurring in the second to fourth decades, although they may be atypical presentations of more ominous pathology, such as synovial sarcoma, extra-skeletal chondrosarcoma, avascular necrosis, or venous or arterial aneurysms of the wrist. They are typically filled from the joint through a tortuous duct or "stalk" that functions as a valve directing the flow of fluid. The mucin itself contains high concentrations of hyaluronic acid as well as glucosamine, albumin, and globulin.[1] When it is used to describe ganglia, the term *synovial cyst* is a misnomer because ganglion cysts do not contain synovial fluid and are not true cysts lined by epithelium, but rather by flat cells. The etiology of ganglia remains a mystery, although many think that ligamentous degeneration or trauma plays an important role.[1,2]

By far, the most common location for a ganglion is the dorsal wrist (Fig. 32.1), with the pedicle arising from the scapholunate ligament[3] in virtually all cases. Only 20% of ganglia are found on the volar wrist (Fig. 32.2), with the most common site at the volar wrist crease between the flexor carpi radialis and abductor pollicis longus at the scaphotrapezoid joint. Alternatively, ganglia can occur near the joints of the finger. One subtype of hand-wrist ganglia is the "occult" cyst, which is not palpable on physical examination.

Ganglion cysts occur more commonly in women, usually between the ages of 20 and 30 years. However, they can develop in either sex at any age. Ganglia of childhood usually resolve spontaneously without sequelae, although somewhat controversial surgical options have shown good long-term outcomes as well.[4] The most commonly seen ganglion of the elderly, the mucous cyst, arises from an arthritic distal interphalangeal joint (Fig. 32.3), which is most commonly associated with osteoarthritis of the joint.

Other common types of ganglia in the hand include the retinacular cyst (flexor tendon sheath ganglion; Fig. 32.4), proximal interphalangeal joint ganglion, and first extensor compartment cyst associated with de Quervain tenosynovitis. Less common ganglia include cysts within the extensor tendons or carpal bones (intraosseous) and those associated with a second or third carpometacarpal boss (arthritic spur). Rarely, ganglia within the carpal tunnel or Guyon canal can produce carpal tunnel syndrome or ulnar neuropathy, respectively, the complexity of which can be evaluated with noninvasive nerve conduction studies.

The direct cause of ganglion cyst formation remains unknown. However, an increased incidence in typists, musicians, surgeons, and draftsmen seems to suggest an association with repetitive activities. Interestingly, there is no increased risk in heavy laborers, who bear a greater load on their wrists. Wrist instability has also been discussed as both a possible cause and an effect of the disease. Overall, there is some history of trauma in 10% to 30% of people presenting with the disease,[2] but with the commonality of bumps and bangs to the hands we all get in daily life, it is hard to draw a direct causative correlation.

Symptoms

Patients with a wrist ganglion usually present with a painless wrist or hand mass of variable duration. The cyst may fluctuate in size or disappear altogether for a time. Pain and weakness of grip are occasional presenting symptoms; however, an underlying concern about the appearance or seriousness of the problem is usually the reason for seeking medical attention. The pain, when present, is most often described as aching and aggravated by certain motions. With dorsal wrist ganglia, patients often complain of discomfort

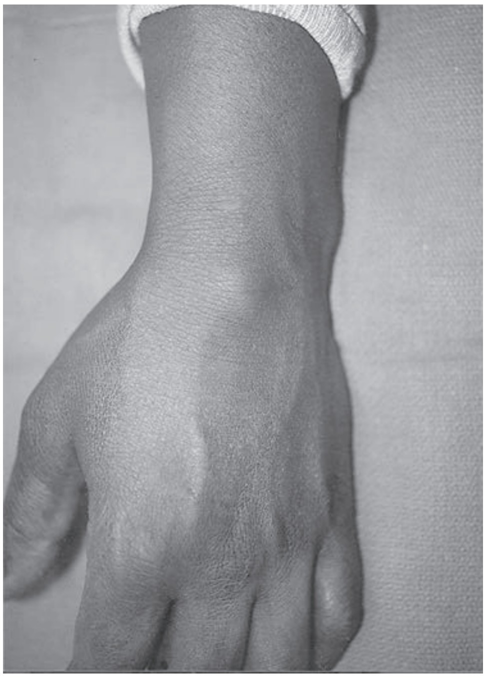


FIG. 32.1 Dorsal wrist ganglion. The mass is typically found overlying the scapholunate area in the center of the wrist.

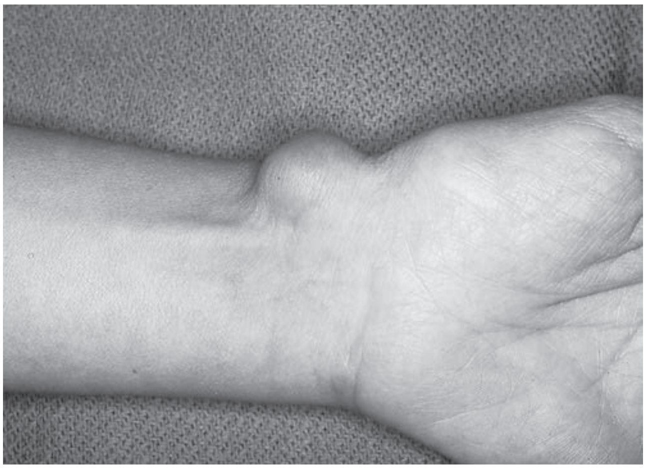


FIG. 32.2 Clinical appearance of a volar wrist ganglion.

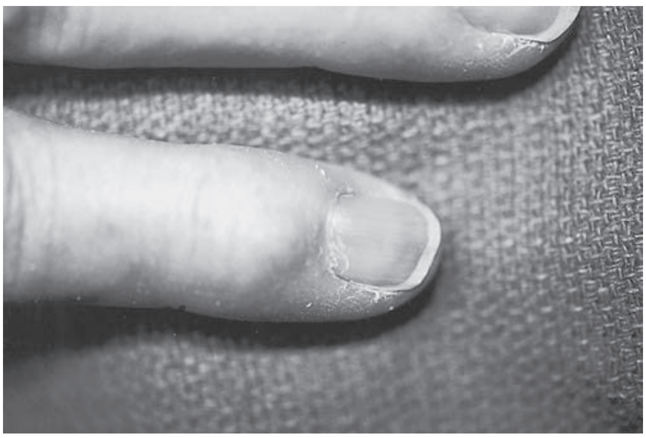


FIG. 32.3 Mucous cyst. This ganglion originates from the distal interphalangeal joint. Pressure on the nail matrix by the cyst may produce flattening of the nail plate, as is seen here.

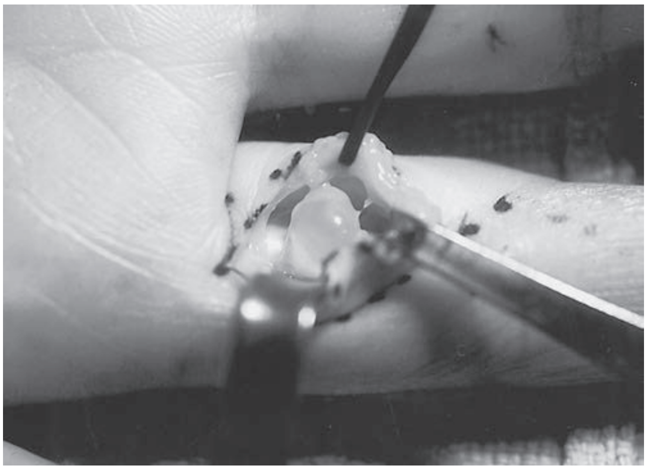


FIG. 32.4 Retinacular cyst. This ganglion originates from the flexor tendon sheath.

as the wrist is forcefully extended (e.g., when pushing up from a chair). Interestingly, dorsal wrist pain may be the principal complaint of patients with an occult dorsal wrist ganglion, which is not readily visible. The wrist pain usually subsides as the mass enlarges.

Retinacular cyst patients usually complain of slight discomfort when gripping, such as a racket handle or milk jug. Patients whose complaints of pain are primarily related to de Quervain tenosynovitis (see Chapter 28) may notice a bump over the radial styloid area and have the classic Finkelstein sign. Pain with grip may be a complaint of patients with a carpometacarpal boss. On occasion, the digital extensor tendons may jump over the cyst with radioulnar deviation. Mucous cysts can drain spontaneously and can also produce nail deformity, either of which may be a presenting complaint. Symptoms identical to those of carpal tunnel syndrome will be noted by patients with a carpal tunnel ganglion. A ganglion in the Guyon canal will produce hand weakness (due to loss of intrinsic function) and may produce numbness in the ring and small fingers.

Physical Examination

The importance of a complete hand and wrist orthopedic, vascular, and neurologic exam cannot be overstated. This includes observation for muscle wasting, neurosensory loss, and vascular compromise. Ganglia are typically solitary cysts, although they are often found to be multiloculated on surgical exploration. They are usually mobile a few millimeters in all directions on physical examination. The mass may be slightly tender. When the cyst is large, transillumination (placing a penlight directly onto the skin overlying the mass) will help differentiate it from a solid tumor.

The classic location for a dorsal wrist ganglion is ulnar to the extensor pollicis longus, between the third and fourth tendon compartments, or directly over the scapholunate ligament.[1] Bilateral or symmetric presentation should trigger considerations of more atypical histology from a migratory embryological origin.[5] However, ganglia may have a long pedicle that courses through various tendon compartments and exits at different locations on the dorsal wrist or even the volar wrist, and multiple review articles suggest that

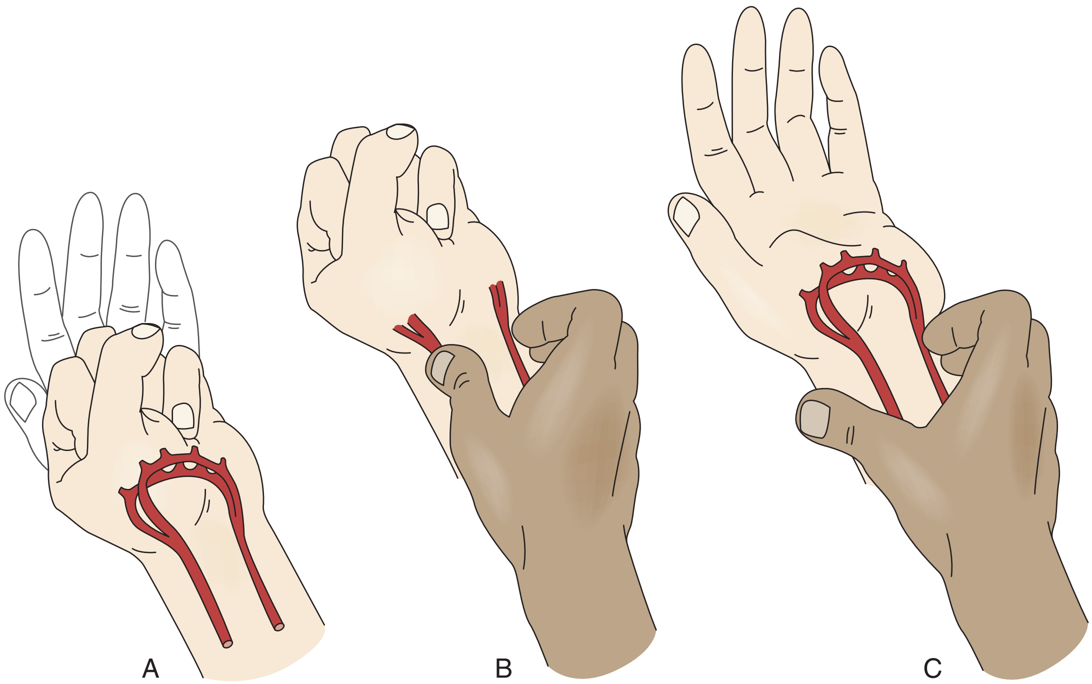


FIG. 32.5 The Allen test is performed by asking the patient to open and close the hand several times as quickly as possible and then to make a tight fist (A). The examiner then compresses the radial and ulnar arteries as the hand is opened (B). One artery is tested by releasing the pressure over the artery to see whether the hand flushes (C). The other artery is tested in a similar fashion, and the opposite hand is tested for comparison.

gross exam usually underestimates the size and complexity of the cyst. When the ganglion is small, it may be apparent only with wrist flexion. Wrist extension and grip strength may be slightly diminished. Dorsal wrist pain and tenderness with no obvious mass or instability should suggest an occult ganglion. Examination of the wrist should be carried out, considering non-ganglia associated conditions. Extensor retinacular tendonitis, extensor carpi ulnaris dislocation,[6] and pseudotumor tuberculosis of the wrist[7] have all been seen and documented initially as ganglia. Volar ganglia occur most commonly at the wrist flexion crease on the radial side of the flexor carpi radialis tendon but may extend into the palm or proximally, or dorsally into the carpal tunnel. They can involve the radial artery, complicating their surgical removal. They may seem to be pulsatile, although careful inspection will demonstrate that the radial artery is draped over the mass.

Retinacular cysts are usually not visible, but are palpable as pea-sized masses, typically located at the volar aspect of the digit at the palmar digital crease. They are adherent to the flexor tendon sheath and do not move with finger flexion. Alternatively, intratendinous ganglia are distinguished by the fact that they move with finger motion.

Mucous cysts are located over the distal interphalangeal joint, and the overlying skin may be quite thin and they may be mistaken for warts and overlap with Heberden nodules. Nail plate ridging deformity is an associated finding. Spontaneous drainage and with septic distal interphalangeal joint arthritis are not uncommon. Proximal interphalangeal joint ganglia are located on the dorsum of the digit, slightly off midline. Ganglia associated with carpometacarpal bosses produce tender prominences on the dorsum of the hand distal to the typical location for a wrist ganglion.

An important sign on physical examination, especially in planning for surgery, is compression of the median or ulnar nerve or of the radial artery. An Allen test should be performed before surgery to evaluate radial and ulnar artery patency, particularly in the case of a volar cyst (Fig. 32.5).

With any concern over compressive neuropathy, an electrodiagnostic medicine study (EDX) should be obtained. Simple nerve conduction studies should document the degree of median nerve involvement. The degree of axonal loss and the needle electromyography portion of the EDX should document presence or absence of active ongoing denervation and suggests the need for expeditious decompression of the median nerve. EDX may also reveal underlying generalized peripheral neuropathy that may possibly help explain a “blurred” inconclusive physical exam.

Functional Limitations

Physical limitations due to ganglion cysts are rare. With dorsal wrist ganglia, fatigue and weakness are occasional findings. Patients may have difficulty with weight bearing on the affected extremity with the wrist extended (e.g., when pushing up from a chair).

Diagnostic Studies

The diagnosis of a ganglion cyst is usually straightforward, and ancillary studies are often unnecessary. With wrist ganglia, plain radiographs of the wrist are usually obtained preoperatively to evaluate the carpal relationships and to exclude the possibility of an intraosseous ganglion. With a mucous cyst, radiographs of the affected digit will usually demonstrate a distal interphalangeal joint osteophyte. Ultrasonography or magnetic resonance imaging may be useful in identifying deep

Table 50.3 Comparison of Various Diagnostic Tests for Lumbar Spinal Stenosis

Imaging Method	Pertinent Findings	Advantages	Disadvantages	Accuracy
Plain radiography	Anteroposterior view: narrow interpedicular distance (normally 23–30 mm)[8] Lateral view: decreased canal width Ferguson view: far-out syndrome[18] Facet degeneration, cyst formation Ligamentum flavum ossification Intervertebral disc space narrowing Vertebral body end-plate osteophyte	Inexpensive Easy to obtain Can rule out gross bone disease	Poor soft tissue visualization	Sensitivity 66% and specificity 93% compared with plain CT as reference[18]
Plain myelography	Ventral extradural defects: caused by disc protrusions and vertebral end-plate osteophytes Lateral or posterior extradural defects: caused by facet osteophytes Hourglass constriction: indicates central stenosis	Shows sagittal plane	Invasive May need several dye injections for high-grade stenosis Limited view of foramen Contraindicated in patients with contrast allergy, alcoholism, seizures, phenothiazine intake[18]	71.8% correlation with surgical findings[16] Sensitivity 54%–100%; equivocal compared with CT or MRI Specificity slightly higher than that of CT or MRI
Plain computed tomography (CT)	Fat plane obliteration at exiting root Canal shape (trefoil vs. round or ovoid) Pedicle length—direct measurement	Relatively inexpensive Axial view Superior bone detail	Poor soft tissue visualization Higher radiation exposure vs. other imaging techniques	83% correlation with surgical findings[16] Sensitivity 74%–100%
Computed tomographic myelography	As above Useful in degenerative scoliosis or history of prior instrumentation	Visualization of central and lateral canals	Invasive Higher radiation exposure vs. other imaging techniques	Sensitivity 87%[17] Comparable to MRI
Magnetic resonance imaging (MRI)	Disc degeneration: dark on T2 Annular tears: bright on T2 Stenosis and herniations in central and foraminal zones well visualized Evaluation of spine and spinal cord tumors	Noninvasive Shows sagittal plane Good soft tissue visualization	Interference from ferromagnetic implants Limitations on patient's body size, need to lie still, claustrophobia Expensive and time-consuming	83% correlation with surgical findings[16] Sensitivity 77%–87% Three-dimensional magnetic resonance myelography sensitivity 100% As accurate as CT myelography[19]
Electrodiagnostics	Bilateral multilevel lumbosacral radiculopathy is most common diagnosis Paraspinal mapping electromyography score > 4[20] Tibial F wave and soleus H reflex latencies after exercise[21,22]	Can evaluate for peripheral neuropathy and entrapments as well as progression of neurologic impairment Can rule out other neuromuscular disease	Significant interpretation bias May be difficult to differentiate lumbar spinal stenosis from other multiroot diseases (e.g., arachnoiditis) Patient discomfort or pain Expensive and time-consuming	Abnormal study in 78%–97% of patients with stenosis Paraspinal mapping electromyography score > 4: specificity 100% and sensitivity 30%

for patients with lumbar spinal stenosis, modalities have no additional effect to exercise and that surgery leads to better long-term (2 years) outcomes for pain and disability, but not walking distance, than physical therapy.[22]

General recommendations include relative rest (avoidance of pain-exacerbating activities while staying active to minimize deconditioning) and a flexion-biased exercise program, including inclined treadmill and exercise bicycle. Flexion biasing increases the cross-sectional area of the spinal canal compared with exercises performed in neutral or extension, thereby maximizing activity tolerance.[23] One case series[24] reported on three patients who demonstrated significant improvements in pain and function at 18 months after undergoing specialized physical therapy programs that included spinal manipulation, flexion and rotation spine mobilization exercises, hip joint mobilization, hip flexor stretching, muscle retraining (lower abdominal, gluteal, and calf muscles), body weight-supported ambulation, and daily walking with properly prescribed orthotics. Physicians and therapists must be aware of any medical comorbidities, such as cardiovascular and pulmonary disease, osteoporosis, cognitive deficits, and other musculoskeletal or neuromuscular conditions, which may have an impact on therapy tolerance. A retrospective analysis of predictors of walking performance and walking capacity showed that body mass index, pain, female sex, and age predict walking performance and capacity in people with lumbar spinal stenosis, those with low back pain without lumbar spinal stenosis, and asymptomatic control subjects. The authors of this analysis concluded that obesity and pain are modifiable predictors of walking deficits that could be targets for future intervention studies aimed at increasing walking performance and capacity in both the low back pain and lumbar spinal stenosis populations.[25]

Body weight-supported ambulation acts to decrease the axial loading of the spine to increase the cross-sectional area of the neural foramina, and studies have provided some support of this strategy.[26] Based on a case series, a wheeled walker set to induce lumbosacral flexion was reported to improve walking and reduce pain in most patients (about 70%).[27]

Procedures

According to the North American Spine Society evidence-based recommendations, interlaminar epidural steroid injections may provide short-term (2 weeks to 6 months) symptom relief in patients with neurogenic claudication or radiculopathy due to degenerative lumbar spinal stenosis, but there is conflicting evidence for long-term (21.5 to 24 months) efficacy.[28]

One systemic review[29] concluded that for interlaminar, transforaminal, and caudal epidural steroid injections, there is strong evidence for short-term relief and limited to moderate evidence for long-term relief of lumbar radicular pain. A number of studies have cited short-term success rates of 71% to 80%[30,31] and long-term success rates of 32% to 75%.[30,32] Furthermore, symptomatic management with epidural steroid injections may delay surgery an average of 13 to 28 months.[33,34]

A randomized controlled trial revealed that patients with lumbar central spinal stenosis received significant pain relief and their Oswestry disability index scores improved after receiving lumbar interlaminar injections with or without steroids.[35] In another study evaluating the short-term effects of transforaminal epidural steroid injection for degenerative lumbar scoliosis combined with spinal stenosis, the researchers found that the Oswestry disability index showed a significantly greater improvement in the steroid group compared to the lidocaine group. The authors concluded that the findings suggest that fluoroscopic transforaminal epidural steroid injections may be an effective nonsurgical treatment option for patients with degenerative lumbar scoliosis combined with spinal stenosis and radicular pain.[36]

In a multicenter, double-blind study of fluoroscopically guided epidural injections for lumbar spinal stenosis, there were no significant differences between patients assigned to glucocorticoids plus lidocaine and those assigned to lidocaine alone with respect to pain-related functional disability or pain intensity at 6 weeks. Patients in both treatment groups had decreased pain and improved function. At 3 weeks, the glucocorticoid-lidocaine group had greater improvement than the lidocaine-alone group, but the differences were clinically insignificant.[37]

In general, epidural steroid injections can be considered a safe and reasonable therapeutic option for symptomatic management before surgical intervention is pursued.

Technology

Currently there is no specific technology for the treatment or rehabilitation of this condition.

Surgery

Lumbar spinal stenosis is the most common reason for spinal surgery in patients over 65 years.[38] Patients with persistent symptoms despite conservative measures may benefit from surgical treatment. Randomized trials comparing surgical decompression with nonsurgical management have suggested that surgery leads to more rapid resolution of symptoms, but the high rate of crossover has been a limitation.[39]

A key feature in the selection of patients is ensuring that the symptoms indeed arise from nerve root compression. Screening for depression also is important. A prospective clinical study of lumbar spinal stenosis patients who underwent surgery found at 2-year follow-up that patients with depressive symptoms had poorer surgical outcomes than those with normal mood.[40] Surgery generally consists of decompressive laminectomy with medial facetectomy. The decompressive laminectomy relieves central canal stenosis; the medial facetectomy and dissection along the lateral recesses decompress areas of foraminal stenosis.

The Maine Lumbar Spine Study prospectively examined the outcome of initial surgical versus nonsurgical treatment in 148 patients out to 10 years. The benefits from surgery diminished over time, although improvements in leg pain and back-related functional status were maintained. Rates of improvement in low back pain and leg pain at 1 year ranged from 77% to 79% in the surgery group and 42% to 45% in the nonsurgical group. At 8- to 10-year follow-up, the rates dropped to 53% to 67% in the surgery group and remained essentially stable at 41% to 50% in the nonsurgical group.[41]

The Spine Patient Outcomes Research Trial (SPORT) compared the 4-year and 8-year outcomes of patients who underwent surgical management of lumbar spinal stenosis with the outcomes of those who received nonoperative care.[42,43] In this prospective randomized trial with a concurrent observational cohort study, surgical candidates with at least 12 weeks of symptoms and confirmatory imaging were enrolled in a randomized cohort or observational cohort. At 2-year follow-up and at 4-year follow-up, intention-to-treat analysis showed significant improvement in SF-36 bodily pain and Oswestry disability index from baseline in the surgical group compared with the nonsurgical group. In addition, comparative effectiveness evidence for defined diagnostic groups from the SPORT showed good value for surgery compared with nonoperative care measured at 4 years. The study also found that patients with predominant leg pain improved significantly more with surgery than did patients with predominant low back pain. However, patients with predominant low back pain still improved significantly more with surgery than with nonoperative treatment.[42] Between 4 and 8 years, patients with symptomatic

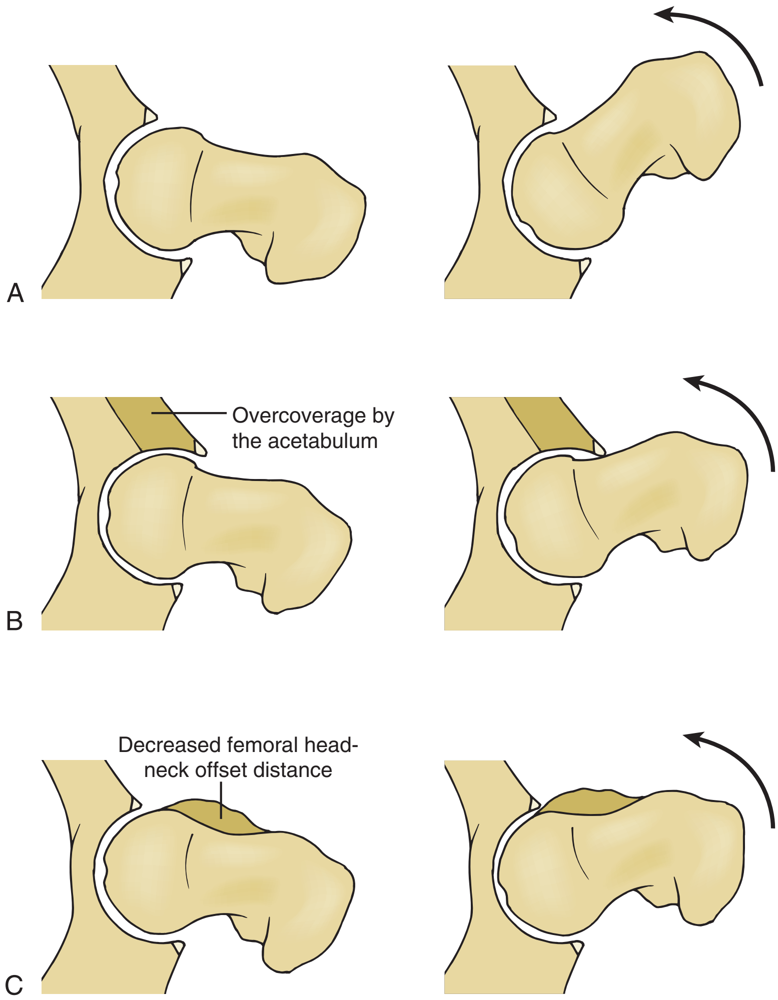


FIG. 56.1 (A) Normal hip joint, which allows unrestricted hip motion. (B) Pincer type femoroacetabular impingement due to excessive coverage of the femoral head by the acetabulum. (C) Cam type femoroacetabular impingement secondary to a decreased femoral head-neck offset distance.

internal rotation during the hip range of motion assessment. A neurologic examination of the lower extremities should be completed, including evaluation of strength, sensation, and reflexes. The neurologic examination findings are typically normal. The most reliable test for FAI and a labral tear is the anterior hip impingement test, which is done by flexing the hip beyond 90 degrees, then adducting and internally rotating the hip (Fig. 56.2). This test is considered positive if it elicits anterior groin pain.[11] This test demonstrates 94% to 99% sensitivity for FAI and labral tears. Flexion-internal rotation test, where the hip is flexed to 90 degrees and internally rotated, also demonstrates excellent sensitivity at 96%.[7] A hip scouring maneuver, in which the hip is taken from an abducted and externally rotated position, through a flexed and neutral rotation position, and finally into adduction and internal rotation, may produce pain and possibly a "click" if a labral tear is present. Passive hip extension and external rotation may cause pain if a posterior labral tear is present. This is commonly referred to as the posterior impingement test. Hip disease can also be provoked by placing the patient's leg in a figure of four position. This test is referred to as the Patrick test or FABER test since the hip is in a flexed, abducted, and externally rotated position. Intra-articular hip disease can also be elicited by a resisted straight-leg raise in

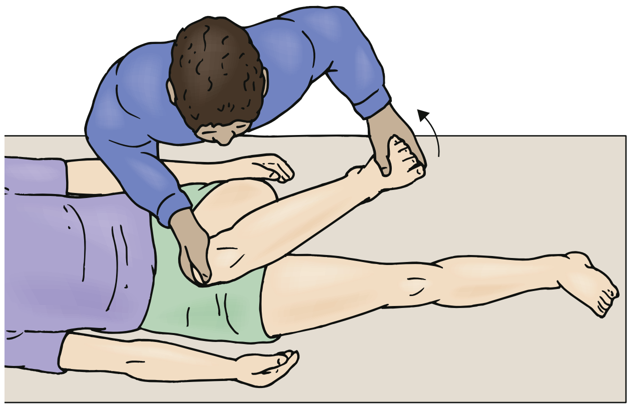


FIG. 56.2 The anterior impingement test involves flexing the patient's hip to 90 degrees, and adducting and internally rotating the hip. A positive test is characterized by hip pain.

the supine position. This is commonly referred to as Stinchfield's test. Although a detailed physical examination assists the clinician in determining that the patient's pain is coming from their hip joint, it is nonspecific and cannot differentiate between the various etiologies of hip pain.

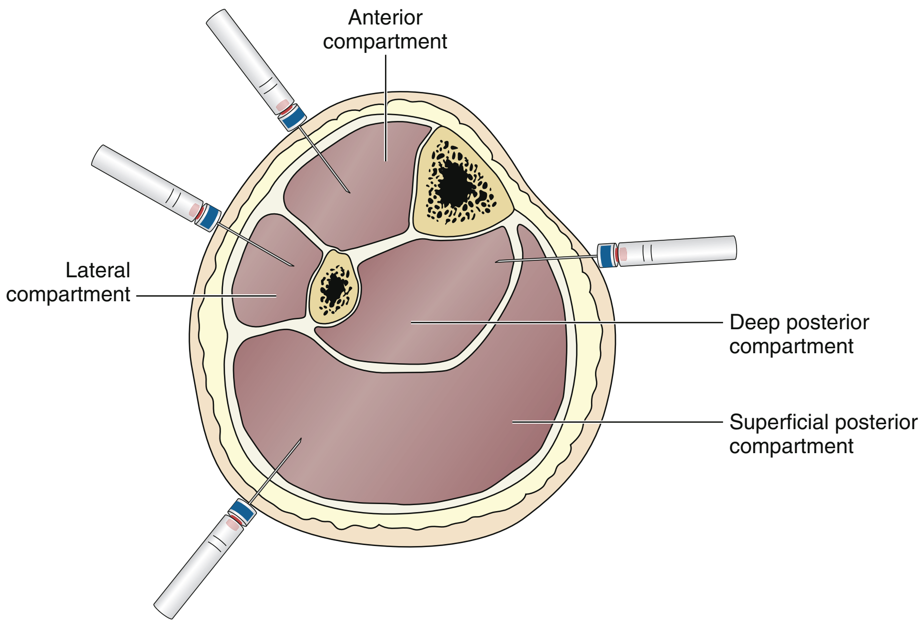


FIG. 67.2 Compartmental tissue pressure measurements for the diagnosis of compartment syndrome with the use of slit and wick catheters.

this can cause, including difficulties with stairs, sports participation, and activities of daily living. In addition, it can lead to muscle necrosis, thereby causing long-term disability.[15]

Chronic Exertional Compartment Syndrome

With CECS, functional limitations usually occur around the same point each time during exercise, at that individual's ischemic threshold. For example, symptoms may start to develop each time a runner reaches the half-mile mark or each time a cyclist climbs a large hill. This may significantly limit sports participation and occasionally even interferes with activities of daily living, such as prolonged walking.

Diagnostic Studies

Compartmental tissue pressure measurement is the "gold standard" for diagnosis. The devices most commonly used to measure intracompartmental pressures were traditionally the slit and wick catheters (Fig. 67.2).[7] Newer devices, such as the transducer-tipped probe (Fig. 67.3), are now gaining popularity.[16] Ultrasound-guided transducer placement is especially helpful in patients receiving anticoagulation, patients with a large body habitus, and patients with distorted anatomy, such as after surgery or trauma.[10]

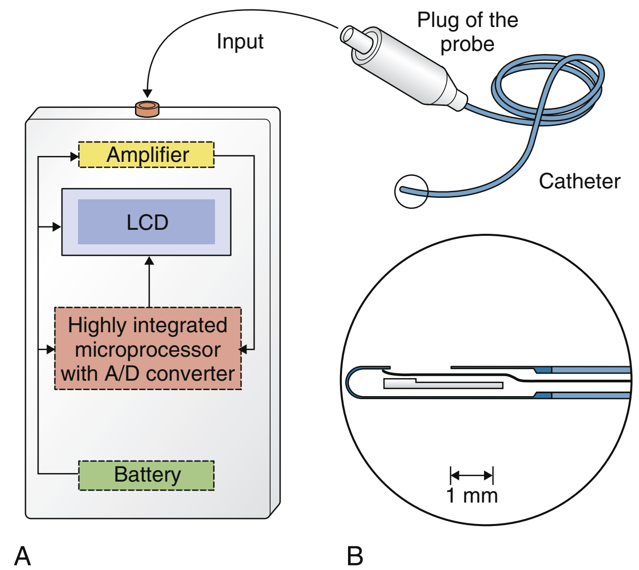


FIG. 67.3 Transducer-tipped probe. (A) Hand-held device. (B) Catheter tip with pressure-sensing mechanics. *(From Willy C, Gerngross H, Sterk J. Measurement of intracompartmental pressure with use of a new electronic transducer-tipped catheter system.* J Bone Joint Surg Am. *1999;81:158–168.)*

Acute Compartment Syndrome

Normal compartment pressure is less than 10 mm Hg. Traditionally, absolute tissue pressure above 30 mm Hg was considered the cutoff value for fasciotomy to be performed.[7,17] However, it is likely that many unnecessary fasciotomies were performed by use of this measure alone. Currently, continuous monitoring of compartment pressures is used in high-risk cases, such as leg trauma with tibial fractures. The differential pressure, calculated as the intramuscular pressure subtracted from the diastolic blood pressure, determines the treatment course. If the differential pressure is less than 30 mm Hg, fasciotomy is indicated.[17,18] Studies have shown that if this differential pressure remains consistently above 30 mm Hg, even with markedly elevated tissue pressures, patients have excellent clinical outcomes and fasciotomy is not necessary.[17,18]

Because of the invasive nature of compartmental pressure measurements, other diagnostic tools have been sought. Magnetic resonance imaging may be helpful in making the diagnosis. Magnetic resonance imaging findings may include loss of normal muscle architecture on T1-weighted images, edema within the compartment, and strong enhancement of the affected compartment with the contrast agent gadolinium-DTPA.[19]

Chronic Exertional Compartment Syndrome

For CECS, absolute pressure measurements obtained at rest, during exercise, and after exercise are used to make the diagnosis. Interestingly, there does not seem to be a particular threshold compartmental pressure at which symptoms occur, and patients with higher pressures do not necessarily have worse symptoms than those with lower pressures.[18] The average time from the onset of symptoms to the time that the diagnosis is made is 22 months.[7]

The following is one set of values[7,8] commonly used to diagnose *anterior compartment* syndrome:

- Pre-exercise pressure > 15 mm Hg
- 1 minute after exercise > 30 mm Hg
- 5 minutes after exercise > 20 mm Hg

A large systematic review found that pre-exercise, means values ranged from 7.4 to 50.7 mm Hg for CECS patients and 5.7 to 12 mm Hg for controls; 1-minute post exercise timing interval showed values ranging from 34 to 55.4 mm Hg and 9 to 19 mm Hg in CECS patients and controls, respectively. The authors concluded that levels above the highest reported value for controls (27.5 mm Hg) along with a good history should be regarded as highly suggestive of CECS.[8]

It is important that the patient's symptoms correlate with the compartment in which there is elevated pressure. Pressure should increase in the symptomatic compartment with exercise and remain elevated for an abnormal time.[8] Values for *posterior compartments* are more controversial. Normal resting pressures are less than 10 mm Hg, and values should return to resting levels after 1 to 2 minutes of exercise.[14]

Drawbacks to measurement of pressures include the following:

- They are invasive and can be complicated by bleeding or infection.
- Because of the anatomy, it is difficult to test the deep posterior compartment.
- Pressures are dependent on the position of the leg and the technique used, so strict standards should be followed.
- It is time-consuming because each compartment must be tested separately, and all compartments should be tested because multiple areas are often involved.
- It is often difficult for patients to exercise with the catheter in place.

Because of these drawbacks, alternative tests to confirm the diagnosis are sometimes used. Magnetic resonance imaging done before and after exercise can show increased signal intensity throughout the affected compartment in the T2-weighted images after exercise in patients with compartment syndrome.[20,21] Alternatively, near-infrared spectroscopy has been used as well. This method measures the hemoglobin saturation of tissues.[8] In cases of CECS, there is de-oxygenation of muscle during exercise and delayed re-oxygenation after exercise. Failure of the compartment to return to baseline within 25 minutes after exercise is diagnostic of CECS.[10] Although near-infrared spectroscopy seems to be helpful in patients with anterior compartment syndrome, light absorption may be altered in deeper compartments, and therefore it is more difficult to monitor the pressure in these deeper compartments.[8] Other methods include thallium stress testing and nuclear magnetic resonance spectroscopy, which may also be helpful in the diagnosis of CECS.[8] Triple-phase bone scan and single-photon emission computed tomography scans can be used to rule out other conditions in the differential diagnosis, such as medial tibial stress syndrome or stress fractures.[10]

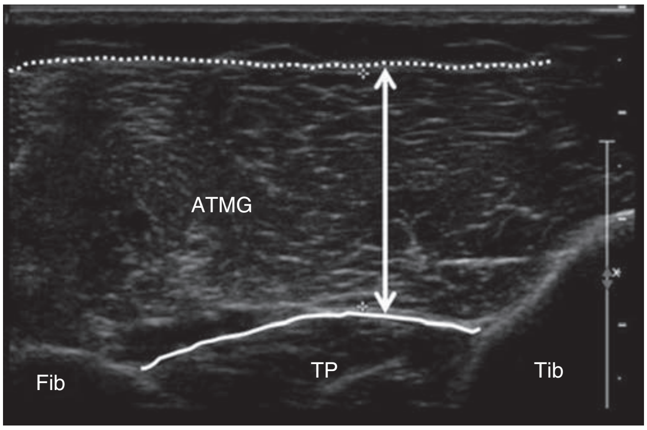


FIG. 67.4 Ultrasound marker placement for anterior compartment thickness measurement using ultrasound in patients at rest. *(From Rajasekaran S, Beavis C, Aly AR, Leswick D. The utility of ultrasound in detecting anterior compartment thickness change in chronic exertional compartment syndrome: a pilot study.* Clin J Sport Med. *2013;23(4):305–311.)*

More recently, ultrasound has been studied as a point of care diagnostic tool used to diagnose CECS. One pilot study showed that the mean anterior compartment thickness (ACT) in patients with CECS versus control subjects significantly increased after exertion at 0.5 minutes, 2.5 minutes, and 4.5 minutes.[22] The authors concluded that ultrasonography reveals a significant increase in ACT in patients with CECS of the anterior leg compartment (Fig 67.4).

Differential Diagnosis

ACUTE COMPARTMENT SYNDROME
Arterial occlusion
Severe muscle trauma
Neuropraxia of the common, deep, or superficial peroneal or tibial nerve
Deep venous thrombosis
Cellulitis
Fracture

CHRONIC EXERTIONAL COMPARTMENT SYNDROME
Tibial or fibular stress fractures
Medial tibial stress syndrome or shin splints
Atherosclerosis with vascular claudication
Popliteal artery compression from aberrant insertion of the medial gastrocnemius
Muscle hyper-development causing compression of the popliteal artery
Cystic adventitial disease[23]

Treatment

Initial

Acute Compartment Syndrome

If the differential pressure is less than 30 mm Hg, treatment of ACS is surgical fasciotomy.[17] In cases in which the

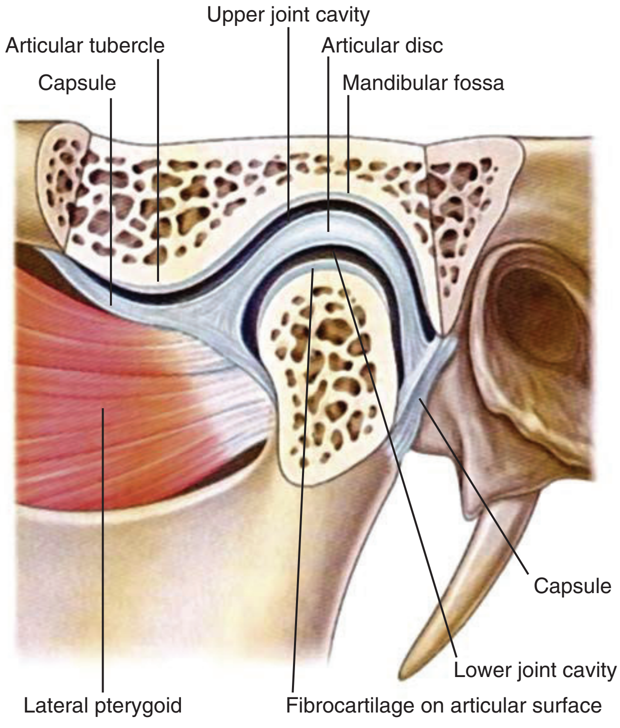


FIG. 114.1 Anatomy of the temporomandibular joint. *(Image from http://www.usa.gov.)*

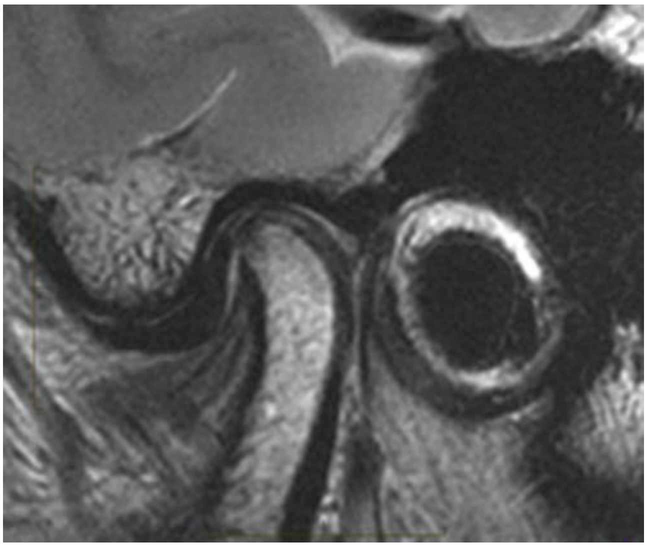


FIG. 114.2 Magnetic resonance image showing anterior displacement of the articular meniscus in relation to the mandibular condyle in closed-mouth position. (*Reprinted with permission from Maizlin ZV, Nutiu N, Dent PB, Vos PM, Fenton DM, Kirby JM, et al. Displacement of the temporomandibular joint disk: correlation between clinical findings and MRI characteristics.* J Can Dent Assoc. *2010;76:a3<http://www.jcda.ca/article/a3>.*)

noise with mandible range of motion; and decreased range of motion/function of mandible.[2] Pain is often the defining feature of this disease and can usually be exacerbated with palpation of the TMJ or the surrounding musculature. Patients may also present with having their jaw "lock" (abrupt decrease in jaw opening), commonly referred to as trismus, upon trying to open the jaw wider in addition to a clicking noise with jaw motion. Headaches are occasionally associated, which are often occipital in location but classified as tension or migraine in quality.[7] Other symptoms of TMJD can include tinnitus, dizziness, and dysphagia.

Physical Examination

A physical exam should start with inspection to assess for any gross abnormalities in size near the joint space, skin integrity, and should always be compared to the opposite side of the body. Next, the joint line and muscles of mastication should be carefully palpated to evaluate for any tender or painful areas or masses. Range of motion, including end-range, and any deviation of the jaw upon opening should also be assessed.[1] When the patient is suffering anterior displacement with reduction, there is a clicking noise that is appreciated upon opening and closing the jaw. As the pathology worsens, movement of the intra-articular disc will not be as free and, as a result, reduction will not occur (Fig. 114.2). This will result in the jaw being locked upon maximal opening. It is also important to assess for any crepitus with opening, as there may be a sign of arthritic change. If there is any question of head or neck trauma, a thorough head and cervical examination should be completed as well.

Functional Limitations

Based on the natural progression of internal derangement (most likely anterior disc displacement), functional limitations may take several years to decades to develop. Once pain is present, decreased range of motion of the jaw (trismus) can be seen.[7] Pain with mastication and/or speaking may be seen. If headaches are a component of the syndrome, sleep may be affected. Because of the chronic nature of pain seen in this condition, there may be other psychosocial components that affect sleep and mood.[8]

Diagnostic Studies

The most common imaging modality used for initial evaluation of orofacial pain is a panoramic x-ray. This modality is useful for evaluating for fracture or arthritic changes, but fails to assess the soft tissue and ligamentous structures often affected in TMJD.[9] Cone-beam computed tomography (CBCT) is an increasingly used imaging study that has the advantage of visualizing the TMJ from sagittal, coronal, and axial planes. This technique is especially useful in determining alterations in bony tissue, including fracture, erosion, osteophytes, and tumor growth. However, CBCT is not able to evaluate soft tissues or the TMJ disc. One study evaluating the incidence of osteoarthritis in TMJD patients found that approximately 70% of joints show some form of degeneration, with the vast majority of those changes occurring in the condyle.[10]

In order to visualize soft tissue structures, magnetic resonance imaging (MRI) is the gold standard for assessing TMJD. MRI has the advantage of three-dimensional analyses of the TMJ disc and retrodiscal tissues in addition to all the surrounding musculature and ligamentous structures (Fig. 114.3). MRI should be considered with clinical symptoms

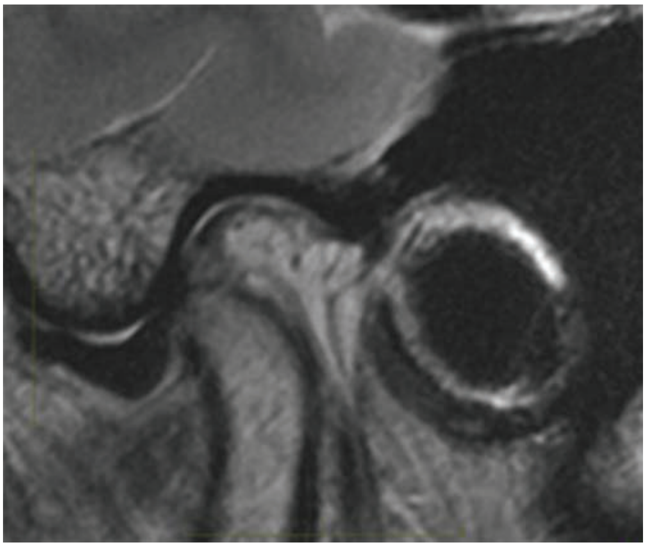


FIG. 114.3 Magnetic resonance image showing the displaced disk does not reduce when the mouth is opened. The opening of the mouth was restricted in this patient. (*Reprinted with permission from Maizlin ZV, Nutiu N, Dent PB, Vos PM, Fenton DM, Kirby JM, et al. Displacement of the temporomandibular joint disk: correlation between clinical findings and MRI characteristics.* J Can Dent Assoc. *2010;76:a3*<*http://www.jcda.ca/article/a3*>.)

Differential Diagnosis

Infection
Neoplasia
Dislocation
Fracture
Headache
Temporal arteritis
Gout/Pseudogout
Myopathy
Tick-borne disease
Trigeminal neuralgia

including pain and noise with jaw range of motion or if a neoplasm is suspected.[9] MRI is 95% sensitive and 88% specific when compared to arthroscopy for evaluating TMJD.[11]

Treatment

Initial

Conservative management is the mainstay of TMJD treatment, and up to 40% of patients will have a spontaneous resolution of their symptoms, and of those who need to be treated, up to 90% will do well with conservative management alone.[12,13] Patient education, behavior modification, and a short 10- to 14-day trial of nonsteroidal anti-inflammatory drugs is typically first-line treatment. During this initial phase, care should be taken to appropriately address any concomitant sleep, headache, or muscular issues.

Rehabilitation

Within the field of rehabilitation, there are many different types of therapies and modalities that may be used. Studies in the past have focused primarily on manual therapy, active exercise, acupuncture, and biofeedback. The most commonly prescribed of these therapies is a mixture of manual manipulation and active exercise. One particular technique of manual manipulation is muscle energy. This relies on the patient to perform an isometric contraction against an equal force performed by a clinician once they have been placed into their end range of motion. This contraction is held for 3 to 5 seconds. This is followed by a rest period, in which the patient is then brought to a new end range of motion followed by another isometric contraction. The goal is to increase range of motion to the affected joint. Muscle energy for the TMJ requires movement of the mandible in several planes of motion, including jaw opening and deviation toward either side to improve range of motion.[14] In fact, a recent meta-analysis showed a positive and significant effect for increasing jaw opening and reduction of pain when compared to more conservative treatments for TMJD.[15] Exercise therapy tends to give similar results, but studies that examined this modality often included some form of manipulation or mobility training as well, so it is unclear as to how effective exercise therapy is alone.[16]

Procedures

There is a wide range of procedural options to treat TMJD based on the etiology and symptomatology of the disease. For example, if hypertonic musculature is the problem, trigger point injections may prove to be beneficial. These injections can also be of diagnostic benefit; if pain persists after this treatment, then a further workup may be considered.[5] For pathology that primarily affects the joint space, intra-articular injection is effective. The question then falls on where to inject these medications based on the double joint space that is formed by the intra-articular disc. A recent study examined this question and found that it is more beneficial to inject both spaces as opposed to the previously commonly accepted practice of injecting only the superior joint space.[17]

When TMJD becomes so severe that patients start to have headaches, botulinum injections may become a viable treatment option. One recent study evaluated injecting botulinum toxin into the masseter and results showed decreased pain and decreased use of analgesic medication.[18]

Acupuncture is another modality used to treat pain. Within the spectrum of TMJD, acupuncture has been shown to have a limited effect on pain when compared to sham treatment based on a recent systematic review and meta-analysis.[19]

Technology

Part of first-line treatment for this disease is the use of occlusal splints, which are usually made of resin and are removable. Its relative ease of use and low cost make it a smart choice for first-line therapy. They are proven to increase maximal jaw opening, decrease the amount of painful clicking episodes, and decrease pain overall.[20] More recently, there has been an emergence of gene-directed therapy and stem cell implementation for various pathologies. This trend

TABLE 137.2 International Myelodysplasia Study Group: Criteria for Assigning Motor Levels

Motor Level	Criteria for Assigning Motor Levels	Functional Movement Noted
T10 or above	Determined by sensory level and/or palpation of abdominal muscles	
T11	Trunk extensors (thoracic and lumbar)	
T12	Abdominal and paraspinal muscles provide some pelvic control. Hip hiking from the quadratus lumborum may also be present	Hip hike
L1	Grade 2 weak iliopsoas, quadratus lumborum	Hip hike
L1-L2	Exceeds criteria for L1 but does not meet L2 criteria	
L2	Meets or exceeds criteria for L1 + Grade 3 iliopsoas Grade 3 sartorius Grade 3 hip adductors (adductor magnus, brevis, longus; pectineus; gracilis)	Hip flexion Hip adduction
L3	Meets or exceeds the criteria for L2 + Grade 3 quadriceps	Knee extension
L3-L4	Exceeds criteria for L3 but does not meet L4 criteria	
L4	Meets or exceeds the criteria for L3 + Grade 3 medial hamstrings Grade 3 tibialis anterior A weak peroneus tertius may also be seen	Medial knee flexion Ankle dorsiflexion
L4-L5	Exceeds criteria for L4 but does not meet L5 criteria	
L5	Meets or exceeds the criteria for L4 + Grade 2 gluteus medius Grade 3 lateral hamstring (biceps femoris) Grade 3 tibialis posterior	Hip abduction Knee flexion Ankle inversion
L5-S1	Exceeds criteria L5 but does not meet S1 criteria	
S1	Meets or exceeds the criteria of L5 + Grade 3 gluteus medius Grade 2 gastrocnemius/soleus complex	Hip abduction Ankle plantarflexion
S1-S2	Exceeds criteria for S1 but does not meet S2 criteria	
S2	Meets or exceeds the criteria for S1 + Grade 4 gluteus maximus Grade 3 gastrocnemius/soleus complex	Hip extension Ankle plantarflexion
S2-S3	All of the lower limb muscle groups are of normal strength (grade 4 in 1–2 muscle groups) Also includes normal-appearing infants who are too young to be bowel and bladder trained	
"No loss"	Meets all of the criteria for S2-S3 + has no bowel or bladder dysfunction	

From McDonald CM, Jaffe KM, Shurtleff DB, et al. Modifications to the traditional description of neurosegmental innervation in myelomeningocele. *Dev Med Child Neurol*. 1991;33(6):473–481.

ambulation.[21] The strength of the iliopsoas, quadriceps, gluteus medius, gluteus maximus, and anterior tibialis are important predictors of ambulatory potential.[10] Generally, patients with thoracic lesions are wheelchair dependent. Teaching early wheelchair mobility (around 2 years of life but as young as 18 months, depending on cognitive function and emotional maturity) is essential to allow toddlers to keep up with their peers and prevent learned helplessness, as wheelchair mobility is often a more functional means of independent mobility. Patients with upper lumbar lesions usually ambulate household distances with variable wheelchair dependency. Patients who have lower lumbar and sacral lesions ambulate longer community distances. These patients may still require some bracing and/or assistive devices and may have gait deviations and decreased endurance.[22]

Other factors, such as presence of hydrocephalus, seizures, muscle tone, contractures, fractures, weight, cognition, and motivation, among others, have been implicated with ambulation and mobility. Positive predictors for ambulation include: lower MMC levels with presence of knee extensor musculature, strong family support, and lack of secondary impairments such as contractures, cognitive issues, and shunt dysfunction.[23]

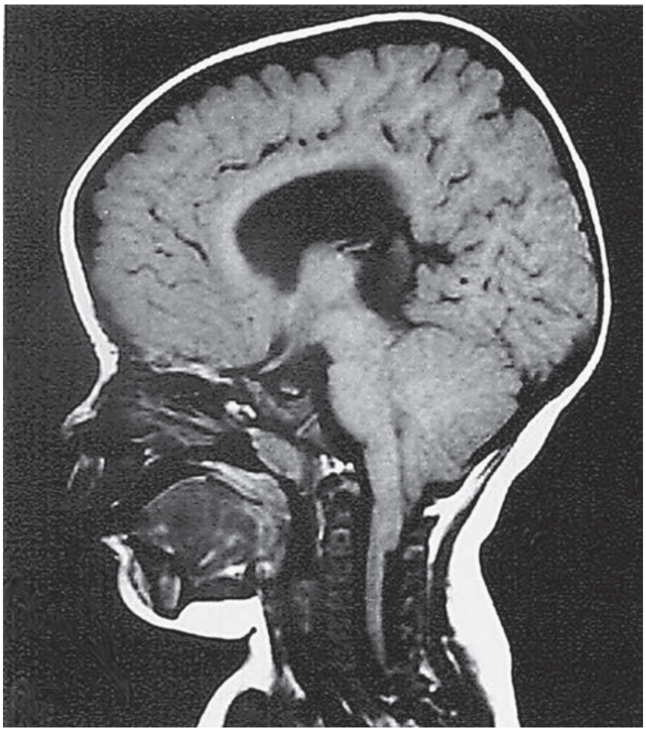


FIG. 137.1 T1-weighted magnetic resonance image of the brain: Arnold-Chiari type II malformation with tonsillar displacement, of more than 3 mm, through the foramen magnum (hindbrain herniation) resulting in obstructive hydrocephalus.

Differential Diagnosis

Alternating hemiplegia of childhood
Arthrogryposis
Cerebral palsy
Charcot Marie Tooth disease
Congenital hypothyroidism
Congenital myopathies
Congenital neuropathies
Currarino syndrome
Hereditary spastic paraparesis
Inborn error of metabolism
Intracranial tumor
Ischemic or hemorrhagic stroke
Multiple vertebral segmentation defects
Myotonia congenital
OEIS complex
Poliomyelitis
Sacrococcygeal teratoma
Spinal epidural abscess
Spinal cord hemorrhage
Spinal cord infarct
Spinal cord injury
Spinal muscle atrophy
Spinal tumor
VACTERL association

Diagnostic Studies

Prenatal

Elevated serum levels of alpha fetoprotein detected in the second trimester suggest a NTD. Fetal ultrasonographic imaging and MRI can aid in determining prognosis and parental decision-making.[24] Amniocentesis may show elevated levels of alpha fetoprotein and acetylcholinesterase, also consistent with NTD.

Postnatal

At birth, the determination of neurologic status is based on clinical observations. A head ultrasound is performed to assess for ventricular size. An echocardiogram can be performed prior to surgical repair to rule out congenital heart disease. Cranial or spinal ultrasound can be used for noninvasive assessment of neurologic status in an infant. Beyond infancy, MRI is performed to assess the spinal cord, and CT is used to assess for hydrocephalus. A shunt series can be performed if there is concern for shunt malfunction. A VPS tap may be performed to rule out infection. Plain film x-ray or CT is used when there is concern for a joint or spine deformity or pathologic fracture.

Urologic studies include renal and bladder ultrasound, voiding cystourethrogram, urodynamic studies, a DMSA nuclear scan, and post void residuals (PVR) measurement.[25] Cystoscopy is performed for bladder cancer surveillance, especially if the patient has undergone bladder augmentation procedures or uses a chronic indwelling bladder catheter.[26] Routine blood work may include a complete blood count, comprehensive metabolic profile, lipid panel,[27] 25(OH) vitamin D level, and cystatin C level.[25] Depending on the clinical presentation, other diagnostic studies may include: endocrine hormone levels, prealbumin level, electroencephalogram, DXA scan, polysomnography, and a latex allergy assay. Throughout childhood and adulthood, healthcare providers should continue to perform routine health maintenance. Neuropsychological testing should be performed prior to entrance into school in preparation for creating a school plan.

Patients with MMC may have abnormal biochemical markers for cardiovascular disease, insulin resistance, and altered bone metabolism.[17]

Treatment

Initial

Surgical closure of MMC within 48 hours is customary.[28] Fetal surgery can be performed around 26 weeks' gestational age.[29] Lower incidences of hindbrain abnormalities, shunt dependent hydrocephalus, as well as improved motor outcomes have been reported for in utero compared to postnatal surgical closure.[30,31] Fetal surgery has not been shown to improve lower urinary tract function or cognitive scores.[32] Moreover, it has resulted in increased incidence of spontaneous rupture of membranes, oligohydramnios, preterm delivery, uterine dehiscence, and fetal or neonatal death.[33] Because of the potential negative consequences, fetal surgery is not the standard of care.

First-line management of hydrocephalus is placement of a VPS. Endoscopic third ventriculostomy (ETV) combined with choroid plexus cauterization represents an alternative technique by addressing both the communicating and non-communicating mechanisms of hydrocephalus without introducing shunt dependency.[10]