1917 2017 100

RHODE ISLAND MEDICAL JOURNAL

SPECIAL SECTION

PEDIATRIC REHABILITATION MEDICINE (PRM)

GUEST EDITOR: JON A. MUKAND, MD, PhD

NOVEMBER 2017 VOLUME 100 • NUMBER 11 ISSN 2327-2228

RHODE ISLAND
MEDICAL JOURNAL


PHOTOS COURTESY OF THE SARGENT REHABILITATION CENTER

Students Boris Franz (cover photo) and Billy Naughton (above) are shown participating in the pediatric physical therapy program, one of the many therapeutic, diagnostic and evaluation services offered at the Sargent Rehabilitation Center (sargentcenter.org) in Warwick, RI.

RHODE ISLAND
MEDICAL JOURNAL

RHODE ISLAND
MEDICAL JOURNAL

IN THE NEWS

PEOPLE/PLACES

NOVEMBER 2017
VOLUME 100 • NUMBER 11

RHODE ISLAND MEDICAL JOURNAL

RHODE ISLAND MEDICAL JOURNAL (USPS 464-820), a monthly publication, is owned and published by the Rhode Island Medical Society, 405 Promenade Street, Suite A, Providence RI 02908, 401-331-3207. All rights reserved. ISSN 2327-2228. Published articles represent opinions of the authors and do not necessarily reflect the official policy of the Rhode Island Medical Society, unless clearly specified. Advertisements do not imply sponsorship or endorsement by the Rhode Island Medical Society.

CONTRIBUTION

CASE REPORTS

PUBLIC HEALTH

Your records are secure.

Until they're not.

Data theft can happen to anyone, anytime. A misplaced mobile device can compromise your personal or patient records. RIMS IBC can get you the cyber liability insurance you need to protect yourself and your patients. Call us.

401-272-1050

RIMS IBC

IN COOPERATION WITH

BMI BUTLER & MESSIER, INC. INSURANCE

RIMS INSURANCE BROKERAGE CORPORATION 405 PROMENADE STREET, SUITE B, PROVIDENCE RI 02908-4811

MEDICALPROFESSIONAL/CYBER LIABILITY PROPERTY/CASUALTY LIFE/HEALTH/DISABILITY

Talkative patients

JOSEPH H. FRIEDMAN, MD
joseph_friedman@brown.edu

I HATE TO RUN LATE. I can't say it is an obsession, but I become uncomfortable when I make patients wait. Most of my patients appreciate this because I am, evidently, an uncommon physician, in that I run on time. When I run late, it is usually because a patient arrived late, for a variety of reasons, almost always unavoidable, and not indicative of moral turpitude on their part. And, of course, some people have difficult problems that take longer than scheduled. However, there is a small number of patients who are simply so talkative that it's hard to run on time. They drive me nuts.

As background for this essay it is important that we understand that M.D. stands for medical doctor and not medical deity. In a famous study (*Ann Int Med* 1984;101(5):692-6) 74 office visits were reviewed, in which only 23% of patients were able to complete their initial statements of what their problems were, and the average amount of time before they were interrupted by the doctor was 18 seconds. Unfortunately, these findings have been confirmed in other reports, indicating that too often the medical deity has taken charge. A physician needs to be sensitive to the needs of the patient, just as the patient needs to be sensitive to the fact that there is usually another patient waiting to be seen.

Recently I saw a patient whose pressured speech was clearly part of the syndrome for which I was being consulted. It reminded me of a similar patient decades ago who was the first I directly confronted by telling her that her non-stop talking was, in fact, part of her problem, and that she needed to stop for a while so that I could compete my evaluation. I was able to get her admitted to a psychiatric hospital and then visited her to find out how she was doing. When I met her on the ward, I didn't think she'd remember me because she had only met me the one time and she was "hyper," to say the least. "Of course I remember you. You're the doctor who told me to shut up," she said with a broad smile.

Handling talkative patients is probably an art. I found some articles on this, but, quite frankly, they were not informative. Perhaps because it was a distillation of suggestions from "good" doctors, discussing how they handle patients in a primary care setting who are excessively talkative. A common suggestion was to tell the patient there was only a limited amount of time, and if they didn't cover everything they'd have to come back next week. Obviously this was in Europe, not the U.S., where a "visit next week," is not an option.

There are several reasons for patients to talk "too much." Mania or extreme anxiety produce non-stop talking, which is clearly part of the problem itself, and becomes part of the examination findings. The more common causes for talkativeness are conceit or self-absorption, and presumed concern that an important aspect of the history will be overlooked. This tends to occur in the highly educated, often with scientific backgrounds. "I just thought that the fact that the taco had hot sauce on it followed by a cola might explain why I was feeling the pain, whereas the week before I didn't get it when I hadn't had the cola, although I had the first for lunch and the second for supper." And, of course, some people are simply non-stop talkers. Every history involves the friends, in-laws, time of day, and God-knows what else.

First of all, I always give people some time to think. I always have the 18-second and 23% completion-of-complaint rate firmly in my consciousness as a measure of bad medicine. Yet, to be honest, histories are, in fact, not that terribly useful in my line of work. The exam is generally much more important, and usually trumps the history if the two are not in alignment. Nevertheless I always take a detailed history. How do I do this in this setting? Generally I apologize for interrupting. "I'm sorry, but I'm having trouble keeping

track of all that information. It will be much better, since I'm taking notes, for me to ask you some questions in a particular order, or I'll get lost. If I don't cover something important at the end you should tell me about it." This approach, I've found, coupled with my age, allows me to take charge, and in a way that saves face. This doctor is limited in his attention and needs to focus. Sometimes I say that I have to behave like a lawyer to make sure I "get it right." That means that I need to ask the questions to get information to line up in a logical sequence. I need to not be distracted. This approach, I think covers all rational people. Even when the talkative ones start getting carried away I can use the same approach to apologize for my own needs, my own peculiar way of getting information that I can use. Unlike the recommendations of the European primary care providers, I don't say that we have limited time. I don't say that there are others waiting. Patients do not want to hear that their doctor is too busy for them, especially if they may have waited two or three months for the appointment.

Some of my patients bring lists of their problems. I encourage this as it makes it less likely that something will be forgotten, but I like to take the list and review it myself. This keeps the list from turning into a collection of bullet points for a lecture that could go on forever.

Patients like to be heard. Their problems need to be considered and validated. I believe that as long as this happens, time constraints should be implemented politely, which will neither demean nor affront the patient.

Easier said than done, unfortunately. ❖

Author

Joseph H. Friedman, MD, is Editor-in-chief of the *Rhode Island Medical Journal,* Professor and the Chief of the Division of Movement Disorders, Department of Neurology at the Alpert Medical School of Brown University, chief of Butler Hospital's Movement Disorders Program and first recipient of the Stanley Aronson Chair in Neurodegenerative Disorders.

Disclosures on website

Insurance News that's Beneficial for Medical Professionals

Medical professionals now save on their business and personal insurance through the Rhode Island Medical Society's exclusive partnership with Butler & Messier.

Contact Robert A. Anderson, AAI at 401.272.1050 – randerson@rimsibc.com

Home & Autos | Boats | Umbrella Liability | Flood | Business Insurance

1401 Newport Avenue, Pawtucket | 1085 Park Avenue, Cranston
405 Promenade Street, Suite B, Providence

butlerandmessier.com

The persistence of racially-based health care inequities

HERBERT RAKATANSKY, MD

There is a statue of Dr. Marion Sims (1813–1883) at 103rd street and 5th Avenue, across from the NY Academy of Medicine. Other statues of Dr. Sims are in Alabama, South Carolina and France. Dr. Sims is remembered for devising an operation that cured vesico-vaginal fistulas, a condition resulting from injury during birth resulting in continuous and uncontrollable urine dripping from the vagina. Dr. Sims also later established the first hospital devoted to treating women's diseases.

The experiments started in 1845, a year before anesthesia was discovered and were performed on slaves sent to Dr. Sims with the hope that a cure would enable them to return to work. Although anesthesia was widely adopted starting in late1846, the AMA did not approve it until 1849 by which time Dr. Sims had finished his experiments without using anesthesia. But even then, Dr. Sims did not use anesthesia on either white or black women. This seems incredulous today, but some belief patterns are difficult to break. For example, some doctors and some members of the clergy felt that anesthesia should not be used during childbirth. Dr. Charles Meigs of Philadelphia stated that painful contractions were "natural and physiological forces that the Divinity has ordered us to enjoy or suffer." That attitude lost traction after Queen Victoria used chloroform for her 8th delivery in 1853.

As with other statues and memorials of well-known military and political persons who endorsed racist beliefs, many people believe that the statue of Dr. Sims should be removed.

Much of the Sims' experimentation was on three slaves (Anarcha, Betsy, and Lucy). Dr. Sims wrote that his success was due "to the indomitable courage of these long suffering women, more than to any other one single circumstance."

But were the women really volunteers? They were restrained forcibly during the operations and likely had little choice. Informed consent, as we know it today, did not exist, though Sims stated that the women consented. But whatever consent was obtained would have been from the slave owner. The women themselves had no legal power to consent or refuse.

One defense of Dr. Sims by those wishing to preserve the statue is that he did what was right in the time and society in which he lived. But that argument could also be used to excuse the Nazi doctors who are remembered today only for their horrific experiments. There are, of course, no statues of them.

Was Dr. Sims a racist? He owned slaves but set them free in 1853, ten years before the Emancipation Proclamation. In 1876, Dr. Sims was president of the AMA. In his presidential address he welcomed the first woman delegate (Sarah Stevenson). Sims also welcomed "any colored man [who] should rise to the dignity of representing a…Medical Society." In spite of this personal welcome, the AMA refused to eliminate race as a criterion for membership.

Membership in the AMA (formed in 1847) was obtained (until modern times) only through membership in a state or county medical society and most of these refused to admit black doctors. A very few black doctors became AMA members in about 1880 but the first black delegate was not admitted until 1950. The RI Medical Society admitted Dr. James A. Gilbert in 1895, who thus became an early AMA member (though not a delegate to the AMA annual meeting). Even the Flexner report in 1910 endorsed segregated and less rigorous medical education for blacks.

In 1963, the RI Medical Society AMA delegation proposed excluding discriminatory state and county societies from AMA membership. The AMA rejected this proposal. Explicit racial discrimination at the AMA ended only with the civil rights law in 1964.

In 2008, the AMA formally apologized for its previous racist policies.

It is well known, but sad to remember, that biases have not been limited to skin color. In 1950 (as reported in the Providence Journal) a black doctor

in RI applied for an internship and was told, "I can't accept you, (but) I'd rather have you than a Jew." One black doctor reported he could not get on the staff of any Providence hospital while two others were accepted.

Disparities in health care derive from many factors, including but not limited to insurance status, income, access to health care facilities, lack of coverage by some health care plans, lack of minority representation on governance committees of the health care system, and racial biases in the health care system itself.

But do doctors themselves have racial biases about patients? Most of us professionals (doctors, nurses, etc., including me) believe that once patients are actually in the "system" they all will receive equal treatment.

But this may not be the reality everywhere. There is evidence that some persons believe that blacks have specific biologic characteristics and these beliefs influence their treatment. Black patients may receive lower doses of analgesics than whites. Black children with appendicitis were less likely to receive any medication and when they did it was less likely to be an opioid. In patients with cancer pain 35% of "racial minority" patients received World Health Organization suggested doses of pain medication while 50% of "non-minority" patients received the suggested dose. While pain in black patients may be undertreated because doctors hold false beliefs about blacks, the doctors who undertreated the black children did not espouse other racist opinions. They believed the pain in blacks was less severe.

In a study reported in 2015, 223 medical students and residents endorsed, on average, 11.5% of a list of 11 false beliefs about race.

50% of these medical students and residents stated that at least one of the false beliefs was at least possibly true, compared to 73% of a control group of non-medical persons. Omitting the opinions of first-year medical students (with less training) did not change the results. Treatment recommendations demonstrated that black patients would be under-treated by those endorsing false beliefs.

Among the 11 false beliefs were:

"Whites on the average have larger brains than blacks."

"Black people's nerve endings are less sensitive than White people's nerve endings."

Thus getting into "the medical system" may not guarantee equal treatment for black patients, though I have not personally observed this. Correcting socio-economic conditions is a public responsibility that needs urgent attention. But the medical profession itself needs to be diligent in addressing its own occult racism.

Getting back to the statue; if it is removed we will cease to honor Dr. Sims publicly. Women will, of course, continue to benefit from the procedures he invented.

Replacing the statue with one of Anarcha, Betsy, and Lucy would honor them and remind us of the racial biases and inequities suffered by black (and other minority) patients.

With or without a statue of them, however, we honor these women best by studying the causes of racially-based health care inequities at all levels and working to eliminate them, thus improving "the system" so that every patient is treated equally, regardless of skin color. ❖

Author

Herbert Rakatansky, MD, FACP, FACG, is Clinical Professor of Medicine Emeritus,The Warren Alpert Medical School of Brown University.

We are read everywhere

DUBLIN, IRELAND

Michael E. Migliori, MD, accessed the October issue at the Royal College of Surgeons in Ireland (RCSI), founded in 1784.

The building was used as a command post by the Irish Citizens Army under Michael Malin and Constance Markievicz in the Easter Rising of 1916, and theirs was the final detachment to surrender to the British.

Bullet holes from that conflict are still evident on the columns and pediment. Malin was executed but Markievicz was spared due to her gender, and went on to become the first woman elected as MP at Westminster in London.

Statues on the pediment include Aesclepius in the center, Hygeia on the right, and Athena on the left.

RIMJ reaches a worldwide audience. In recent months readers from 69 countries accessed our articles from their computers and mobile devices. Wherever you may be, or wherever your travels take you, be sure to check the journal on your mobile device and send us a photo: **mkorr@rimed.org.**

Pediatric Rehabilitation Medicine (PRM): An Integrative Approach to Identifying and Treating Congenital and Childhood Disabilities

JON A. MUKAND, MD, PhD
GUEST EDITOR

Pediatric Rehabilitation Medicine (PRM) is a challenging and rewarding sub-specialty of Rehabilitation Medicine. The American Board of Physical Medicine & Rehabilitation defines it as "the subspecialty that uses an interdisciplinary approach to address the prevention, diagnosis, treatment, and management of congenital and childhood-onset physical impairments including related or secondary medical, physical, functional, psychosocial, cognitive, and vocational limitations or conditions, with an understanding of the life course of disability."[1]

Children are vulnerable to disabling conditions starting before birth (e.g. toxoplasmosis), at birth (e.g. hypoxic-ischemic encephalopathy), and through the adolescent phase (e.g. injuries). CDC data (2015) on injury deaths are a useful index for disabilities among the survivors of those injuries.[2] For the two decades between ages 5–24, motor vehicle collisions were the most common cause of death. Suicide by firearms was the third (n = 139) most common cause for ages 10–14 and the fourth (n = 2,461) for ages 15–24. Even for young people, there is a risk of homicide by firearms: it was the second most common cause of death (n = 4,140) for the age group of 15–24. Some of the injuries that afflict children every day could be prevented by education from medical practitioners, for instance, advising parents to secure guns in safes and to ensure safe seating in cars.

This issue of the *Rhode Island Medical Journal* features the intersection of orthopedic surgery and rehabilitation for the pediatric population. Pediatric surgery requires a clear understanding of the natural course of pathologic conditions, the nature of pediatric tissue healing, the developmental process, and psychosocial issues.

Brachial plexus injuries at birth occur in up to fifty children in Rhode Island annually. They require the surgeon to be patient and wait for the natural recovery process, to enlist occupational therapists for rehabilitation, and to carefully select a small percentage of infants for surgery. At a later stage of life, children may have to contend with adolescent idiopathic scoliosis. Fortunately, only 10% of these children require surgical intervention. Sports injuries are relatively common in America. About half of injuries that eventually lead to surgery involve the knee, and in 25% of these cases the anterior cruciate ligament is injured. The most vulnerable athletes are 16-year-old females and 17-year-old males. All three of these conditions are discussed in this special issue, in articles that are authored by orthopedic surgery residents and co-authored by their attending physicians at Rhode Island Hospital. As a rehabilitation physician, I appreciated these articles for their accessibility and usefulness for general medical practitioners, and I hope that the readership of the journal will also benefit from them.

References

1. https://www.abpmr.org/Subspecialties/PRM, Accessed 10/7/17
2. https://www.cdc.gov/injury/images/lc-charts/leading_causes_of_injury_deaths_unintentional_injury_2015_1050w760h.gif, Accessed 10/7/17

Guest Editor

Jon A. Mukand, MD, PhD, is Consulting Medical Director, Southern New England Rehabilitation Center; Medical Director, Sargent Rehabilitation Center; Clinical Assistant Professor, Rehabilitation Medicine, Brown University, Tufts University.

A Review of Brachial Plexus Birth Palsy: Injury and Rehabilitation

JEREMY E. RADUCHA, MD; BRIAN COHEN, MD; TRAVIS BLOOD, MD; JULIA KATARINCIC, MD

ABSTRACT

Brachial plexus injuries during the birthing process can leave infants with upper extremity deficits corresponding to the location of the lesion within the complex plexus anatomy. Manifestations can range from mild injuries with complete resolution to severe and permanent disability. Overall, patients have a high rate of spontaneous recovery (66–92%).[1,2] Initially, all lesions are managed with passive range motion and observation. Prevention and/or correction of contractures with occupational therapy and serial splinting/casting along with encouraging normal development are the main goals of non-operative treatment. Surgical intervention may be warranted, depending on functional recovery.

KEYWORDS: Brachial plexus, Erb's palsy, Klumpke's palsy, serial splinting

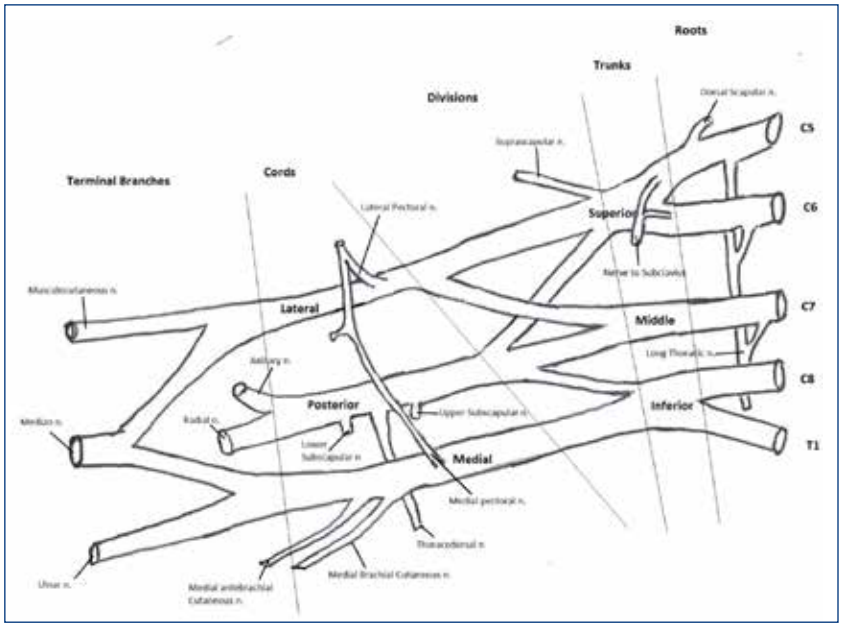
INTRODUCTION

Brachial plexus birth palsy (BPBP) involves injury to any nerve of the brachial plexus during birth. It occurs in 0.42 to 4.6 cases per 1,000 births, which translates to approximately 5 to 50 cases per year in Rhode Island, with varying degrees of severity.[1–3] The most common presentation is Erb's Palsy (50-60%), followed by the more severe upper plexus and pan-plexus variants.[1,4] Klumpke's lower plexus palsy is rare, and occurs in 0.6% of all patients.[5] Maternal risk factors include gestational diabetes, multi-parity and having a previous child with a brachial plexus injury. Maternal factors can cause fetal macrosomia and/or shoulder dystocia, increasing the risk of forceps or suction-assisted deliveries and traction nerve injury.[6] Since the majority of fetuses present in the left occiput anterior position, with the right shoulder under the maternal pelvis, the right upper extremity is most commonly involved.[7] However, only about half of patients have these risk factors, demonstrating our lack of true understanding of the etiology.[8] This article will review the pathology, diagnosis, treatment, rehabilitation and outcomes of BPBP.

ANATOMY

The brachial plexus is derived from the fifth cervical (C5) to the first thoracic (T1) nerve roots. It undergoes a complex pattern of branching and convergence before terminating as peripheral nerves that provide motor and sensory innervation to the upper extremity. (**Figure 1, Table 1**) The plexus can be divided into supraclavicular (roots and trunks) and sub-clavicular (cords and terminal branches) for prognostic purposes, with supraclavicular injuries having worse outcomes.[9]

Figure 1. Brachial Plexus Anatomy



Pathophysiology

The majority of BPBPs are traction injuries, as with shoulder dystocia when traction on the infant's neck leads to an increased neck shoulder angle.[1] Very rarely, compression injuries from fractured clavicles, hematomas, and pseudoaneurysm can occur.[7] Lesions can be divided into symptomatic categories using multiple systems. The simplest approach is to classify lesions as pre-ganglionic or post-ganglionic, distal to the dorsal root ganglion. Pre-ganglionic lesions, with the nerve injured proximally, e.g., root avulsions, are more difficult to heal/repair and have worse outcomes than post-ganglionic lesions. It is only possible to determine this

Table 1. Brachial Plexus Functions

Branching Location	Nerve	Root	Innvervation	Muscle action
Roots	Dorsal Scapular n.	C5	M: Rhomboid mm. and Levator Scapulae m.	Rhom: scapular retraction, Levator=scapular elevation
	Long Thoracic n.	C5,C6 & C7	M: Serratus anterior m.	Scapular protraction
	First intercostal n.	T1	M: intercostal m.	n/a
Trunks	Suprascapular n.	C5, C6	M: Supraspinatus m, Infraspinatus m. S: Shoulder joint capsule	Supra= Arm abduction. Infra= Arm external rotation
	Nerve to Subclavius	C5, C6	M: Subclavius m.	n/a
Divisions	none			
Cords				
Posterior	Upper Subscapular n	C5-T1	*Motor*: Upper subscapularis m.	Arm internal rotation
	Lower Subscapular n	C5-T1	*Motor*: Lower Subscapularis m., Teres Major m.	LS= Arm internal rotation
	Thoracodorsal n.	C5-T1	*Motor*: Latissimus dorsi m.	Arm adduction
Lateral	Lateral Pectoral n.	C5-C7	*Motor*: Pectoralis Major m.	Arm Adduction
Medial	Medial Pectoral n.	C8-T1	*Motor*: Pectoralis Major m., Pectoralis Minor m.	Arm Addution
	Medial Brachial cutaneous n.	C8-T1	*Sensory*: medial arm	n/a
	Medial Antebrachial cutaneous n.	C8-T1	Sensory: medial forearm	n/a
Terminal Nerves	Radial n.	C5-T1	*Motor*: Triceps mm, brachioradialis m., ECRL, ECRB, ECU, EDC, EIP, EDM, EPL, EPB, APL, Supinator m., Finger extensors *Sensory*: posterior brachial cutaneous, inferior lateral brachial cutaneous, posterior antebrachial cutaneous, superficial radial (post. radial hand)	Elbow extension, Wrist extension, Finger Extension, Thumb extension, thumb abduction, Forearm supination; Brachioradialis=elbow flexion
	Axillary n.	C5-T1	*Motor:* Deltoid m., Teres Minor m., Sensory: Lateral proximal arm	Delt= Arm abduction, Teres= Arm external rotation
	Musculocutaneous n.	C5-C7	*Motor*: Biceps brachii m., Brachialis m, corocobrachialis m. *Sensory:* Lateral cutaneous n. of the forearm	Elbow Flexion, Forearm supination S: lateral forearm
	Median n.	C5-T1	*Motor*: FCR, Palmaris longus m., FDS, radial 1/2 FDP, Pronator teres m. FPL, Pronator quadratus m., FPB (superficial head), Opponens pollicis, APB, 1st-2nd lumbricals *Sensory*: Radial 3 1/2 fingers, palmar cutaneous branch	Wrist flexion, Forearm pronation, thumb flexion/abduction/opposition, finger PIP flexion, IF/MF MCP and DIP flexion
	Ulnar n.	C8-T1	*Motor*: FCU, ulnar 1/2 FDP, Flexor DM, Abductor DM, Opponens DM, Adductor pollicis, FPB (deep head), Palmaris brevis m. Dorsal interossei mm. Palmar interossei mm., 3rd-4th Lumbricals *Sensory*: Dorsal ulnar cutaneous n., Palmar ulnar cutaneous n.	Wrist flexion, Thumb adduction/flexion, SF flexion/abduction/opposition, finger adduction/abduction, 4th and 5th finger DIP/MCP flexion

classification after advanced imaging. The Sunderland classification (**Table 2**) categorizes nerve injuries based on the nerve structures damaged, ranging in severity from neuropraxia to neurotmesis.[10] As expected, patients with less severe damage, e.g., neuropraxia, have a better chance at recovery.

The most common way to describe BPBPs is based on the nerve roots involved, which can be detected by physical examination. Upper trunk (Erb-Duchenne) palsies involve only the disruption of input from the C5 and C6 nerve roots. Upper plexus palsies involve roots C5, C6 and C7, with the addition of more distal deficits. Lower plexus (Klumpke's) palsies involve the C8 and T1 nerve roots and can also affect the sympathetic chain with pre-ganglionic injuries. The most severe is the all-encompassing pan-plexus injury involving nerve roots C5-T1, with disruption to all functions of the upper extremity.

Table 2. Sunderland Classification

	Type of Nerve Injury	Prognosis
Neuropraxia	Stretch injury with intact nerve continuity	Spontaneous recovery likely
Axonotmesis	Axonal injury with intact nerve sheath	Variable recovery
Neurotmesis	Complete nerve rupture; neither axon nor sheath intact	Poor prognosis for spontaneous recovery

Sunderland SS. The anatomy and physiology of nerve injury. *Muscle Nerve*. 1990;13(9):771-784. doi:10.1002/mus.880130903

DIAGNOSIS

Maternal history, physical examination and diagnostic imaging can provide a wealth of information to make the proper diagnosis and injury classification. The patient's mother should be interviewed for the BPBP risk factors mentioned previously. Abnormal primitive reflexes, e.g., Moro reflex and tonic neck reflex, are often the first clues in the newborn examination. It is also important to palpate the infant's clavicle and humerus, as fractures can affect upper extremity movements and be confused with brachial plexus palsy. A septic shoulder and isolated radial nerve palsy should also be in the differential diagnosis, but they are less common and are associated with different physical and laboratory findings.

Depending where the lesion is located, the patient's affected extremity will present in different positions. With Erb's palsy (C5-6), the arm is adducted and internally rotated at the shoulder and extended at the elbow, due to weakness in the deltoid, supero-posterior rotator cuff and biceps. A patient with upper plexus palsy (C5-7) has the above posture as well as wrist and fingers flexion due to radial nerve involvement and wrist/finger extensor weakness. Pan-plexus injuries (C5-T1) typically present with a flaccid extremity. Pre-ganglionic injuries, which carry a worse prognosis,, may lead to head tilting to the opposite side (denervation of paraspinal musculature), medial winging of the scapula, diaphragm dysfunction, and Horner's syndrome.

As children age, their disabilities become more apparent. Scoring systems such as the Toronto Test Score, Active Movement Scale, and Modified Mallet system have been developed to grade and track upper extremity function.[11] The Modified Mallet score is the most commonly used when evaluating older children (≥3 years old). It uses five categories to assess shoulder function, with a 0–5 grading for each category. Higher scores correlate to higher function, but the examination requires patient participation and is heavily weighted toward shoulder external rotation.

Imaging can help clarify the diagnosis and classification. Initially, radiographs of the upper extremity should be obtained to rule out fractures, which could be confused with or occur concomitantly with brachial plexus palsy. MRI and CT myelography can be used to detect root avulsions. Electromyography has been suggested if there is no nerve recovery by 6 months of age, in order to detect a pre-ganglionic injury, which is potentially amenable to operative intervention. Other evidence shows, however, that electromyography can be discordant from clinical bicep function at 3 months of age and the test may not be a reliable indicator for surgery.[1]

TREATMENT AND OUTCOMES

Treatment for a suspected brachial plexus palsy should begin immediately with frequent, passive range of motion of the affected upper extremity. Parents should be instructed to range both arms at every diaper change to make it a daily routine and encourage compliance. Some authors recommend a two-week period of immobilization to promote healing and decrease pain,[9] but others find little evidence that immobilization has any benefit.[1] Contractures can begin as early as 2-3 weeks after birth, with the glenohumeral joint most commonly affected. Without early treatment, the contractures can progress rapidly and cause posterior subluxation/dislocation of the humeral head.[1,9,12]

After an initial observation period, children can be categorized as having either partial or total paralysis. Patients with total paralysis should be referred to a tertiary center for early surgical evaluation, as they have a very low likelihood of spontaneous recovery. Patients with partial paralysis have a higher chance of recovery, and there is complete recovery by 3 months of age with as many as 92% of these patients.[9,13] Other evidence suggests complete recovery rates may not be as high as originally thought, with as many as 20–30% of patients having a long-term defecit.[1,2] Patients who do not have complete recovery by 1 month of age should be evaluated by a pediatric therapist for continued monitoring and rehabilitation. Many physicians use the lack of antigravity biceps function return by 3 months as an indication for nerve surgery since it is a poor prognostic indicator for complete spontaneous recovery;[1,4] however, this does not preclude good functional recovery. Recovery of wrist extension is also a positive prognostic sign. Other physicians advocate continued rehabilitation until at least 6 months of age before considering surgery. Some evidence has shown that children who recovered antigravity biceps function between the 3rd and 6th months of life always had an incomplete recovery compared to those who regained function prior to 3 months.[9]

If children undergo surgery, it is typically performed between 3 and 8 months of age; earlier surgery (at 3 months) is indicated in children with pan-plexus palsies and Horner's syndrome. The main goals of surgery, in order of importance, are to restore elbow flexion, shoulder abduction, shoulder external rotation, wrist extension and hand function.[7] The options for early surgical intervention include direct nerve repair with resection and grafting, or nerve transfers from surrounding motor nerves. In spite of surgery, many patients still suffer some degree of long-term sequalae.[2] In patients who develop contracture or have persistent weakness, later surgery can be beneficial. Lysis of contractures, osteotomies and local tendon transfers can help return functional motion and correct deformities.[12,14]

Children who recover meaningful biceps function by 6 months of age are typically treated non-operatively with rehabilitation and monitoring. There is little high grade research discussing non-operative management techniques and protocols for brachial plexus birth palsies. All non-operative treatment involves a multidisciplinary team approach, with occupational therapy and splinting to prevent or correct contractures. The goals of treatment prior to muscle function recovery are to prevent contracture, strengthen recovering muscles, stimulate sensory nerves, and encourage the achievement of normal developmental milestones. As the

child grows, passive range of motion should be transitioned to participation in age-appropriate activities for rehabilitation with regular follow-up to assess functional scores, arm growth and joint integrity. Elbow flexion contractures are a fairly common occurrence, even with triceps sparing palsies. For children who develop contractures, stretching and serial night splinting can be used for contractures less than 20 degrees. Treating deformities in this range will prevent progression and help cosmetic appearance, as the elbow's functional range of motion is between 30–130 degrees.[15] Serial casting and splinting of elbow flexion contractures can yield good results, but this approach can be complicated by radial head dislocation, bony ingrowth at the joint, and loss of elbow flexion while gaining extension. Much of the research is focused on elbow flexion contractures but contractures preventing forearm supination and shoulder external rotation are also commonly present. A pilot study has shown improvement in Toronto and Active Movement Scales of supination and shoulder external rotation with a Supination-External rotation orthosis worn 22 hours per day with reprieves for therapy twice per day.[16] Botulinum toxin injections with serial casting have also shown promise in patients who failed serial casting alone.[17] The toxin relaxes the antagonist muscle at the contracted joint, particularly in cases of co-contraction, a common long-term complication.

Despite the lack of consensus regarding surgical indications and rehabilitation protocols, patients do have good long-term outcomes. Most studies show the majority of patients are independent in activities of daily living, even with persistent functional deficits.[1,2,7,8,13,19] In a subjective study of adolescents, all patients reported a 'really good' quality of life, but they were also all dissatisfied with their current condition and hoped for continued improvement.[20]

CONCLUSION

Brachial plexus birth palsies can be stressful and challenging for parents and children. Despite a better understanding of the pathology and treatment options, injury incidence has remained unchanged.[1–3] In general, upper plexus palsies recover better than lower plexus and pan-plexus palsies; neuropraxia does better than neurotmesis; and post-ganglionic lesions recover better than pre-ganglionic lesions. Care should continue to focus on early identification and therapy to minimize complications. Early referral to tertiary centers is crucial, as a multi-disciplinary approach can help promote recovery and prevent complication. Fortunately there is a high rate of spontaneous recovery, but for patients who don't recover spontaneously there are non-surgical and surgical options to improve functional outcomes and prevent devastating contractures. With an ever-expanding body of research geared towards improving care and knowledge of the injury, the future should show improved long-term outcomes for these patients.

References

1. Malessy MJA, Pondaag W. Obstetric Brachial Plexus Injuries. *Neurosurg Clin N Am*. 2009;20(1):1-14. doi:10.1016/j.nec.2008.07.024.
2. Hoeksma AF, Wolf H, Oei SL. Obstetrical brachial plexus injuries: incidence, natural course and shoulder contracture. *Clin Rehabil*. 2000;14(5):523-526. doi:10.1191/0269215500cr341oa.
3. Evans-Jones G, Kay SPJ, Weindling AM, et al. Congenital brachial palsy: incidence, causes, and outcome in the United Kingdom and Republic of Ireland. *Arch Dis Child Fetal Neonatal Ed*. 2003;88(3):F185-9. http://www.ncbi.nlm.nih.gov/pubmed/12719390. Accessed May 15, 2017.
4. Kozin SH. Brachial Plexus Microsurgical Indications. *J Pediatr Orthop*. 2010;30:S49-S52. doi:10.1097/BPO.0b013e3181cd9ed2.
5. al-Qattan MM, Clarke HM, Curtis CG. Klumpke's birth palsy. Does it really exist? *J Hand Surg Br*. 1995;20(1):19-23. http://www.ncbi.nlm.nih.gov/pubmed/7759926. Accessed May 15, 2017.
6. Gilbert WM, Nesbitt TS, Danielsen B. Associated factors in 1611 cases of brachial plexus injury. *Obstet Gynecol*. 1999;93(4):536-540. http://www.ncbi.nlm.nih.gov/pubmed/10214829. Accessed May 15, 2017.
7. Shenaq SM, Bullocks JM, Dhillon G, Lee RT, Laurent JP. Management of infant brachial plexus injuries. *Clin Plast Surg*. 2005;32(1):79-98. doi:10.1016/j.cps.2004.09.001.
8. Hale HB, Bae DS, Waters PM. Current concepts in the management of brachial plexus birth palsy. *J Hand Surg Am*. 2010;35(2):322-331. doi:10.1016/j.jhsa.2009.11.026.
9. Abid A. Brachial plexus birth palsy: Management during the first year of life. *Orthop Traumatol Surg Res*. 2016;102(1 Suppl):S125-32. doi:10.1016/j.otsr.2015.05.008.
10. Sunderland SS. The anatomy and physiology of nerve injury. *Muscle Nerve*. 1990;13(9):771-784. doi:10.1002/mus.880130903.
11. Duff S, DeMatteo C. Clinical Assessment of the Infant and Child Following Perinatal Brachial Plexus Injury. *J Hand Ther*. 2015;28(2):126-134. doi:10.1016/j.jht.2015.01.001.
12. Abid A, Accadbled F, Louis D, et al. Arthroscopic release for shoulder internal rotation contracture secondary to brachial plexus birth palsy: clinical and magnetic resonance imaging results on glenohumeral dysplasia. *J Pediatr Orthop B*. 2012;21(4):305-309. doi:10.1097/BPB.0b013e328353688e.
13. Laurent JP, Lee R, Shenaq S, Parke JT, Solis IS, Kowalik L. Neurosurgical correction of upper brachial plexus birth injuries. *J Neurosurg*. 1993;79(2):197-203. doi:10.3171/jns.1993.79.2.0197.
14. Abzug JM, Kozin SH. Evaluation and Management of Brachial Plexus Birth Palsy. *Orthop Clin North Am*. 2014;45(2):225-232. doi:10.1016/j.ocl.2013.12.004.
15. Ho ES, Roy T, Stephens D, Clarke HM. Serial casting and splinting of elbow contractures in children with obstetric brachial plexus palsy. *J Hand Surg Am*. 2010;35(1):84-91. doi:10.1016/j.jhsa.2009.09.014.
16. Durlacher KM, Bellows D, Verchere C. Sup-ER orthosis: an innovative treatment for infants with birth related brachial plexus injury. *J Hand Ther*. 2014;27(4):335-39; quiz 340. doi:10.1016/j.jht.2014.06.001.
17. Basciani M, Intiso D. Botulinum toxin type-A and plaster cast treatment in children with upper brachial plexus palsy. *Pediatr Rehabil*. 2006;9(2):165-170. doi:10.1080/13693780500402229.
18. Kerr C, McDowell B, Cosgrove A, Walsh D, Bradbury I, McDonough S. Electrical stimulation in cerebral palsy: a randomized controlled trial. *Dev Med Child Neurol*. 2006;48(11):870-876. doi:10.1017/S0012162206001915.
19. DiTaranto P, Campagna L, Price AE, Grossman JAI. Outcome following nonoperative treatment of brachial plexus birth injuries. *J Child Neurol*. 2004;19(2):87-90. doi:10.1177/08830738040190020101.
20. Squitieri L, Larson BP, Chang KWC, Yang LJS, Chung KC. Understanding quality of life and patient expectations among adolescents with neonatal brachial plexus palsy: a qualitative and quantitative pilot study. *J Hand Surg Am*. 2013;38(12):2387-2397.e2. http://www.ncbi.nlm.nih.gov/pubmed/24416766. Accessed May 8, 2017.

Authors

Jeremy E. Raducha, MD; Department of Orthopaedic Surgery, Warren Alpert Medical School of Brown University, Providence, RI.

Brian Cohen, MD; Department of Orthopaedic Surgery, Warren Alpert Medical School of Brown University, Providence, RI.

Travis Blood, MD; Department of Orthopaedic Surgery, Warren Alpert Medical School of Brown University, Providence, RI.

Julia Katarincic, MD; Department of Orthopaedic Surgery, Warren Alpert Medical School of Brown University, Providence, RI.

Correspondence

Jeremy E. Raducha, MD
Department of Orthopaedic Surgery
Rhode Island Hospital
593 Eddy Street
Providence, RI 02903
401-444-4030
Fax 401-444-6182
Jeremy.raducha@gmail.com

The Role of Rehabilitation in the Management of Adolescent Idiopathic Scoliosis

JOSE M. RAMIREZ, MD; CRAIG P. EBERSON, MD

ABSTRACT

Adolescent idiopathic scoliosis (AIS) is a common clinical entity that affects approximately 2–3% of children and adolescents. AIS is defined as a curvature of the spine > 10 degrees and it usually presents as a right thoracic curve. Only a small fraction of patients with AIS go on to surgical intervention. This article will review the role of rehabilitation in the management of adolescent idiopathic scoliosis, specifically as related to the preoperative, perioperative and postoperative care of patients with AIS.

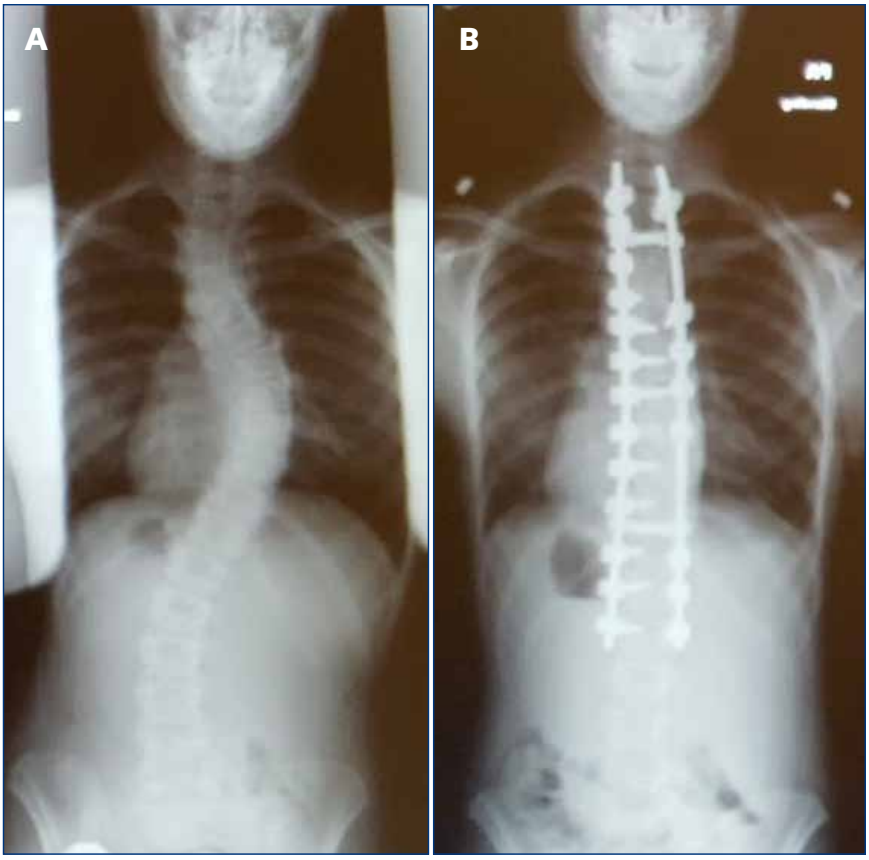
INTRODUCTION

Adolescent idiopathic scoliosis (AIS) is a common clinical entity that affects approximately 2–3% of children and adolescents. AIS is defined as a curvature of the spine > 10 degrees and it usually presents as a right thoracic curve. Only 10% of patients with AIS go on to surgical intervention.[14,1]

The exact pathogenesis of AIS is not yet known. Multiple genetic and environmental factors have been implicated and continue to be the subject of intense research. Lifestyle factors have been implicated in the progression of AIS, but a recent, large cross-sectional study of over 2,700 female high school students failed to show any factors that were significantly related to AIS.[13]

The management of AIS is guided by curve magnitude and remaining skeletal growth. Rehabilitation plays an important and complementary role in the management of patients with AIS across the entire continuum of care. AIS patients with curves less than 25 degrees are typically observed and may be prescribed physical therapy in the form of scoliosis-specific exercises (SSE). Patients with curves between 25–45 degrees are often braced, with consideration of SSE as an additional treatment option. The aim of these interventions is to slow or reverse deformity in the spine. Posterior spinal fusion (PSF) (**Figure 1**) is often indicated for patients who fail these conservative measures and progress to curves greater than 45 degrees with substantial skeletal growth remaining, as well as for patients with curves greater than 50 degrees.

Figure 1. Thirteen-year-old patient initially treated with bracing and SSE for idiopathic scoliosis. She was poorly compliant and required surgery. Postoperatively, she returned to competitive swimming after 6 months, **A**. Preoperative anteroposterior image, **B**. Postoperative image.



Preoperative Considerations

Exercise therapy may improve an imbalance of peri-spinal musculature that is seen in AIS patients with small curves (< 20 degrees).[15,16,18] This therapy may also be used a complement to bracing therapy in patients with larger curves. A study by Ko et al[15] recently analyzed the effects of a 12-week

core stabilization program on patients with AIS and curves of 10–20 degrees; there was a significant decrease in lumbar curve magnitudes in the treated groups. Gür et al[17] similarly showed significant improvement in lumbar curve levels in patients undergoing a core stabilization exercise program. Moreover, a 2015 meta-analysis[19] found moderate-quality evidence supporting the role of exercise therapy in modifying curve values, thoracic kyphosis trunk rotation, and quality of life in patients with AIS.

The increasingly popular Schroth rehabilitation program utilizes specific postures to correct scoliosis. A randomized clinical trial showed significantly improved curve magnitude as measured by Cobb angle and trunk rotation.[23] Similarly, active self-correction and task-oriented exercises have also been shown to be effective in achieving Cobb angle improvement (greater than 5 degrees) in a randomized clinical trial.[24] While by no means a panacea to AIS, postural rehabilitation and active self-correction rehabilitation programs should be considered for the management of AIS. Patient compliance and follow-through with the home portion of the program is imperative for success. Longer-term studies to evaluate these exercise interventions are ongoing and will determine the usefulness of these interventions.

Perioperative Considerations

For patients with progressive deformity, surgery heralds the beginning of an intensive rehabilitation period. The primary goal of inpatient rehabilitation after PSF is to ensure a safe home discharge. Doing so requires the coordinated effort of medical, nursing, and rehabilitation providers.

Medical priorities in the postoperative period include: monitoring the patient's hemodynamic status; achieving adequate pain control; managing the surgical site and various lines/drains (i.e., intravenous, bladder catheter); and ensuring normal bodily functions such as voiding.

Physical and occupational therapy goals include mobilizing the patient out of bed, ambulating on flat ground, navigating stairs, and performing activities of daily living in a safe manner. A survey of Shriners Hospital surgeons found the following physical therapy goals: Sitting on day 1, standing on day 2 and walking on day 2 or 3.[12]

Recently, care pathways have been developed to standardize and improve the postoperative rehabilitation of patients undergoing PSF for AIS. These pathways are designed to achieve a safe and efficient discharge to home after surgery. Fletcher et al[6] reported the outcomes of one such pathway as it compared to traditional discharge planning. Both groups had a small difference in thoracolumbar curve Cobb values (35 degree in accelerated pathway vs. 40 degrees in traditional pathway), but there was no difference in proximal and main thoracic curve dimensions. As part of the accelerated discharge pathway, patients underwent aggressive postoperative rehabilitation with 2 to 3 sessions daily. Patients were transitioned to a regular diet on postoperative day 1, at which time they were started on a multimodal regimen of oral analgesics and diazepam. Bladder catheters were removed on postoperative day 1 and surgical drains removed on day 1 or 2. Finally, patients were discharged to home with a standard bowel regimen if they were tolerating a regular diet, even if they did not yet have a bowel movement post-operatively. This study found a significantly shorter hospital length of stay (2.2 vs. 4.2 days) in the accelerated discharge group. Importantly, the accelerated pathway was safe and did not show an increase in complications or re-admission rates when compared to the traditional discharge pathway. Other benefits of the accelerated pathway cited by the authors include faster return to work by parents, lower healthcare cost, and a faster return to a daily home routine. Sanders et al[7] also demonstrated a statistically significant decrease in LOS with an accelerated rehabilitation pathway (3.7 vs 5.0 days). This pathway was also safe and reduced hospital costs by 22%.[7] The authors have found that most adolescent patients can be discharged within 3 or 4 days with an aggressive rehabilitation approach.

Postoperative Considerations

The postoperative rehabilitation of patients undergoing PSF for AIS builds on the inpatient gains by returning to baseline activity at home and improving strength and confidence. This occurs in parallel with evaluations to ensure adequate healing of the surgical site and arthrodesis of the spine.

After PSF, Tarrant et al[9] found a median time to return to school of 10 weeks and 77% of patients had returned to school by 16 weeks postoperatively. Another study by these authors[11] found that patients with preoperative curves greater than 70 degrees were delayed by approximately 1 additional month in their return to school.

Clearance to return to sporting activity will vary by attending surgeon. A survey of scoliosis surgeons[10] found that 43% of surgeons recommended low-impact, non-contact sports at 6 months after surgery. Additionally, 60% of surgeons permitted contact sports (i.e., soccer, basketball) after 12 months. However, 60% did not recommend participation in higher impact collision style sports (i.e., football, hockey) after PSF.

Most patients can expect to be restricted in their activities until about 3 to 6 months postoperatively. Additionally, it is our practice to wait for resolution of pain and to ensure maintenance correction and progression towards arthrodesis before return to non-contact physical activity. Fabricant et al[8] retrospectively analyzed a cohort of 42 patients undergoing PSF for AIS. In this cohort with a mean age of 15.0±1.7 years, the average preoperative curve magnitude was 57.67 ±9.38 degrees and the average time to return to athletic activity was 7.4±3.4 months. Moreover, 25 (59%) of patients returned to physical activity at the same level or better than before surgery, but 7 had to change their activity. Those who did not return to sports or did so at lower level most

commonly cited a loss of flexibility and back pain as their reasons. Additionally, these patients were more likely to have a higher preoperative Lenke classification and a lower level of fusion at the time of surgery. For example, while 73% of patients with a T12 distal fusion level returned to their prior activities after surgery, only 20% of patients with an L4 fusion level achieved this outcome. What is reassuring is that no complications related to return to play were reported. Taken together, these data suggest that a return to athletics is a safe and realistic rehabilitation goal following PSF. However, some patients may ultimately change their sport or activity level, depending on the complexity of their deformity.

As a result of surgical intervention around the paraspinal musculature and the fusion that is ultimately achieved in the spine, AIS patients may experience changes in range of motion (ROM) and the muscular function of their spine. These patients will go on to make adaptive changes while performing certain activities.[20] This may include increased reliance on leg muscles[20] to compensate for weakened paraspinal musculature with forward bends. Physical therapy in the postoperative period should address the loss of flexibility seen as a result of fusion. Additionally, athletes returning to sports will benefit from specific exercises to improve balance, agility and gait while retraining with sports-specific activities.[22, 21] After the initial healing period, it is the authors' protocol to institute formal physical therapy for sport-specific rehabilitation for patients wishing early return to sports.

CONCLUSION

Adolescent Idiopathic Scoliosis is an entity commonly encountered by pediatric musculoskeletal providers. As outlined above, rehabilitation plays an important role at all stages of the continuum of care of patients with AIS. Preoperative exercise therapies may confer a lasting, albeit modest, benefit to patients not yet indicated for surgery. Coordinated, inpatient postoperative care pathways are proving to be cost-effective, safe, and effective in accelerating the postoperative rehabilitation of patients undergoing PSF for AIS. Lastly, ongoing postoperative rehab will ensure a return to an active lifestyle and, in many patients, a return to a high level of activity.

References

1. Miller NH. Cause and natural history of adolescent idiopathic scoliosis. Orthop Clin North Am. 1999;30:343–52.
2. Sui WY, Ye F, Yang JL. Efficacy of tranexamic acid in reducing allogeneic blood products in adolescent idiopathic scoliosis surgery. BMC Musculoskelet Disord. 2016 Apr 27;17:187.
3. Wang M, Zheng XF, Jiang LS. Efficacy and Safety of Antifibrinolytic Agents in Reducing Perioperative Blood Loss and Transfusion Requirements in Scoliosis Surgery: A Systematic Review and Meta-Analysis. PLoS One. 2015 Sep 18;10(9):e0137886.
4. Bowen RE, Gardner S, Scaduto AA, Eagan M, Beckstead J. Efficacy of intraoperative cell salvage systems in pediatric idiopathic scoliosis patients undergoing posterior spinal fusion with segmental spinal instrumentation. Spine (Phila Pa 1976). 2010 Jan 15;35(2):246-51.
5. Oliveira JAA, Filho FAM, Fernandes FV, Almeida PC, de Oliveira VF, Lima Verde SR. Is cell salvage cost-effective in posterior arthrodesis for adolescent idiopathic scoliosis in the public health system? J Spine Surg. 2017 Mar;3(1):2-8.
6. Fletcher ND, Andras LM, Lazarus DE, Owen RJ, Geddes BJ, Cao J, Skaggs DL, Oswald TS, Bruce RW Jr. Use of a Novel Pathway for Early Discharge Was Associated With a 48% Shorter Length of Stay After Posterior Spinal Fusion for Adolescent Idiopathic Scoliosis. J Pediatr Orthop. 2017 Mar;37(2):92-97.
7. Sanders AE, Andras LM, Sousa T, Kissinger C, Cucchiaro G, Skaggs DL. Accelerated Discharge Protocol for Posterior Spinal Fusion Patients With Adolescent Idiopathic Scoliosis Decreases Hospital Postoperative Charges 22. Spine (Phila Pa 1976). 2017 Jan 15;42(2):92-97.
8. Fabricant PD, Admoni S, Green DW, Ipp LS, Widmann RF. Return to athletic activity after posterior spinal fusion for adolescent idiopathic scoliosis: analysis of independent predictors. J Pediatr Orthop. 2012 Apr-May;32(3):259-65.
9. Tarrant RC, O'Loughlin PF, Lynch S, Queally JM, Sheeran P, Moore DP, Kiely PJ. Timing and predictors of return to short-term functional activity in adolescent idiopathic scoliosis after posterior spinal fusion: a prospective study. Spine (Phila Pa 1976). 2014 Aug 15;39(18):1471-8.
10. Rubery PT, Bradford DS. Athletic activity after spine surgery in children and adolescents: results of a survey. Spine (Phila Pa 1976). 2002 Feb 15;27(4):423-7.
11. Tarrant RC, Queally JM, O'Loughlin PF, Sheeran P, Moore DP, Kiely PJ. Preoperative curves of greater magnitude (>70°) in adolescent idiopathic scoliosis are associated with increased surgical complexity, higher cost of surgical treatment and a delayed return to function. Ir J Med Sci. 2016 May;185(2):463-71.
12. Sanders JO, Haynes R, Lighter D, Niederpruem M, Hollenback C, Johnson L, Nomura S, Arndt D, Bush P, Santiago J, King R, Trottier T. Variation in care among spinal deformity surgeons: results of a survey of the Shriners hospitals for children. Spine (Phila Pa 1976). 2007 Jun 1;32(13):1444-9.
13. Watanabe K, Michikawa T, et al. Physical Activities and Lifestyle Factors Related to Adolescent Idiopathic Scoliosis. The Journal of Bone and Joint Surgery. 99(4):284–294, Feb 2017.
14. Lonstein JE. Scoliosis: surgical versus nonsurgical treatment. Clin Orthop Relat Res. 2006;443:248–259.
15. Mooney V, Gulick J, Pozos R. A preliminary report on the effect of measured strength training in adolescent idiopathic scoliosis. J Spinal Disord. 2000;13:102–10.
16. Ko KJ, Kang SJ. Effects of 12-week core stabilization exercise on the Cobb angle and lumbar muscle strength of adolescents with idiopathic scoliosis. J Exerc Rehabil. 2017 Apr 30;13(2):244-249.
17. Gür G, Ayhan C, Yakut Y. The effectiveness of core stabilization exercise in adolescent idiopathic scoliosis: A randomized controlled trial. Prosthet Orthot Int. 2017 Jun;41(3):303-310.
18. SOSORT guideline committee, Weiss HR, Negrini S, Rigo M, Kotwicki T, Hawes MC, Grivas TB, Maruyama T, Landauer F. Indications for conservative management of scoliosis (guidelines). Scoliosis. 2006 May 8;1:5. doi: 10.1186/1748-7161-1-5.

19. Shahnawaz A, Alghadir A, Abu S, Dilshad A. Effects of Exercise on Spinal Deformities and Quality of Life in Patients with Adolescent Idiopathic Scoliosis. Biomed Res Int. 2015;2015:123848.
20. Wang TY, Pao JL, Yang RS, Jang JS, Hsu WL. The adaptive changes in muscle coordination following lumbar spinal fusion. Hum Mov Sci. 2015 Apr;40:284-97. Epub 2015 Jan 24.
21. Kakar RS, Simpson KJ, Das BM, Brown CN. Review of Physical Activity Benefits and Potential Considerations for Individuals with Surgical Fusion of Spine for Scoliosis. Int J Exerc Sci. 2017 Mar 1;10(2):166-177. eCollection 2017.
22. Pescatello LS, Arena R, Riebe D, Thompson PD. ACSM's Guidelines for Exercise Testing and Prescription. Ninth Edition. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2014.
23. Kuru T, Yeldan İ, Dereli EE, Özdinçler AR, Dikici F, Çolak İ. The efficacy of three-dimensional Schroth exercises in adolescent idiopathic scoliosis: a randomised controlled clinical trial. Clin Rehabil. 2016 Feb;30(2):181-90.
24. Monticone M, Ambrosini E, Cazzaniga D, Rocca B, Ferrante S. Active self-correction and task-oriented exercises reduce spinal deformity and improve quality of life in subjects with mild adolescent idiopathic scoliosis. Results of a randomised controlled trial. Eur Spine J. 2014 Jun;23(6):1204-14.

Authors

Jose M. Ramirez, MD; Department of Orthopaedic Surgery, Alpert Medical School, Brown University, Providence, RI.

Craig P. Eberson, MD; Department of Orthopaedic Surgery, Alpert Medical School, Brown University; Hasbro Children's Hospital; University Orthopedics, Providence, RI.

Correspondence

Jose_Ramirez@Brown.edu

Craig_Eberson@Brown.edu

Pediatric Anterior Cruciate Ligament Rehabilitation: A Review

STEVEN F. DEFRODA, MD, MEng; KATHRYN HILLER, BS; ARISTIDES I. CRUZ, Jr., MD, MBA

ABSTRACT

Rehabilitation is crucial in the treatment of ACL injuries, particularly in the pediatric population. Children are often eager to return to their pre-injury level of athletic participation, which may place them at risk for re-injury if rehabilitation protocols are not adequately followed. Contemporary protocols incorporate functional benchmarks rather than solely time-based milestones to better evaluate if patients have adequate strength and function to return to sport activities. Optimization of rehabilitation can lead to safer return to play and minimize the risk of re-injury. Ultimately, successful rehabilitation requires effective communication between the entire care team, including the patient, family, therapist, coaches, trainers, and orthopaedic surgeon in order to optimize recovery from injury.

INTRODUCTION

Increased organized sport participation in children and adolescents has led to an increase in the number of acute and chronic injuries in youth athletes.[1] Among high school athletes, up to 50% of injuries that require surgery involve the knee and 25% of those knee injuries involve the ACL.[2,3] Beck et al. reviewed the incidence of ACL injury in patients aged 6–18 from 1994–2013 and found an average rate of 121 injuries per 100,000 person-years.[4] The highest rates were in 17-year-old males (422 per 100,000) and 16-year-old females (392 per 100,000). An important finding was that over the 20-year period, there was 2.3% average annual increase in the rate of injury.[4] As the rate of injury has increased, so has the number of ACL reconstruction (ACL-R) surgeries in pediatric patients over the last 20 years.[5] Dodwell et al. examined a state-based database and found that from 1990 to 2009 the rate of pediatric ACL-R increased from 17.6 to 50.9 per 100,000.[5]

Many controversies exist regarding the treatment of ACL injuries in the pediatric population, as they are skeletally immature individuals. Reconstruction of the ACL in a manner similar to skeletally mature patients would require disruption of the physis,[6] which could result in growth disturbances.[7] While there have been favorable outcomes regarding initial nonsurgical treatment and delayed reconstruction (until skeletal maturity), these options have resulted in increased knee instability and a higher risk of osteoarthritis later in life.[8–10] Various surgical techniques have been developed in order to provide long-term knee stability in pediatric patients, including physeal sparing as well as partial and complete transphyseal techniques.[11–15] The specific type of procedure to be performed depends on a number of factors, including patient age, activity level, and surgeon preference.

While recent efforts have focused on understanding optimal ACL injury management in the pediatric population, less time has been spent on determining the proper rehabilitation processes that should follow. Adult studies have proven that both post-operative and pre-operative rehabilitation lead to improved functional outcomes.[16] Specific rehabilitation protocols can impact the speed and safety with which patients return to sporting activities.[17] Rehabilitation protocols may vary based on the type of surgery performed; however, both pre- and post-operative rehabilitation are crucial components in managing pediatric ACL injury.[18] The purpose of this paper is to review current trends in pediatric ACL rehabilitation, as well as to identify future areas of study.

NON-OPERATIVE TREATMENT

Nonsurgical treatment has been the traditional approach to ACL injury management in the pediatric population due to the concern of disrupting the physis through surgical reconstruction. Non-operative treatment typically consists of activity modification, physical therapy, and specialized bracing.[19,20] Moksnes, Engebretsen, and Risberg[21] outline a four-phase nonsurgical treatment program that emphasizes range of motion, neuromuscular training, and strengthening. In Phases 1 and 2, patients work closely with physical therapists and are provided with exercises to perform at home; they proceed to the next phase only after meeting specific functional milestones. Phase 3 incorporates jumping and landing, open- and closed-chain strengthening exercises, and plyometric drills. Phase 4 consists of a secondary injury prevention program that focuses on functional stability. If recurrent instability occurs despite activity modification and progressing through the treatment program, surgical reconstruction is recommended.[20]

While nonsurgical treatment preserves the growth plate, many studies have demonstrated its shortcomings, including

an increased risk of instability, meniscal injuries, and chondral injuries.[8–10,20,22,23] When treated non-operatively, 19.5% of pediatric patients sustained new meniscal injuries after their initial ACL tear, and cartilage injuries had a prevalence of 7.1% two to three years after the initial ACL tear.[22] Surgery should be recommended if nonsurgical treatment does not provide sufficient functional stability, if patients continue to have episodes of giving way, if a satisfactory activity level is not achieved, or if there is a significant concomitant meniscal injury.[20–22] While some surgeons advocate for delayed reconstruction once the physis matures, others have found that if surgery is delayed by ≥12 weeks, there is a significantly increased chance of irreparable meniscus injury and lateral compartment chondral injury. [24] Furthermore, the severity of the injuries increase with time. [25] A recent systematic review of the literature concluded that early ACL-R leads to less meniscal and chondral damage compared to non-operative or delayed surgical treatment.[23] There are currently no high-level studies that directly compare the efficacy of nonsurgical treatment to surgical reconstruction of the ACL in the pediatric population.[26]

"PRE-HABILITATION"

In the adult population, pre-operative rehabilitation has been shown to improve knee-related functioning, muscle strength, and return-to-sport rates after ACL-R.[16,27] There is little documentation, however, of the effects of pre-habilitation protocols in the pediatric population. In a case study, Greenberg et al. described a brief pre-operative physical therapy regimen before an all-epiphyseal ACL-R, with gait training, assessment of the patient's maturity level, and ability to follow post-operative instructions.[28] The functional goals included no effusion, at least 80% quadriceps strength in the affected leg when compared with the unaffected leg, full extension, at least 120 degrees of active knee flexion, and independence with weight-bearing restrictions. Fabricant et al. recommend activity modification and closed chain rehabilitation following ACL tears in the pediatric population, but no specific recommendations were provided.[29]

POST-OPERATIVE WEIGHT-BEARING

Generally, surgeons encourage early post-operative weight-bearing following ACL-R, but in the pediatric population, a more restricted weight-bearing protocol may preserve the graft tissue and the physis.[28] Some surgeons recommend restricting patients to toe-touch weight-bearing (TTWB) for at least the first week following an all-epiphyseal reconstruction. [28,30] Weight-bearing as tolerated (WBAT) is advised during weeks 2–4 post-ACL-R until the patient has a normalized gait pattern, [30] and full weight-bearing is recommended by post-operative week five. [28] Similarly, according to the Hospital for Special Surgery (HSS) protocol, patients should aim to normalize gait patterns while WBAT during post-operative weeks 4–8.[29] In the presence of a concomitant meniscus repair, surgeons generally limit weight-bearing to allow time for the meniscus to heal.[21,30,31]

RANGE OF MOTION

One of the major goals of post-operative rehabilitation is for the patient to obtain full range of motion (ROM) of the knee. Passive and active exercises are suggested in the early post-operative phase to help improve extension and flexion, including a continuous passive motion machine,[28] wall slides, prone dangling, resting extension with a heel prop, and stationary cycling.[30] Surgeons tend to rely on time-based criteria when restricting knee motion during the rehabilitation protocol.[32] Some surgeons recommend locking the post-operative brace in full extension immediately following surgery for up to three[28] or four[30] weeks, while both ambulating and sleeping. There is wide variability in the literature regarding post-operative ROM goals. One article recommends reaching 50 degrees of knee flexion by post-operative week four and 90 flexion by week five,[28] while another suggests 90 flexion by week two and 120 flexion by week four.[30] Others have recommended 90 flexion by week four, 125 flexion by week eight, and full ROM by week sixteen.[29] Only Akinleye et al.[30] provide specific functional criteria that must be met before unlocking the brace and removing restrictions. Makhni et al. found a wide range of variability in adult ACL-R protocols at academic institutions, similar to the pediatric literature.[33] There is variability in post-operative protocols, but the main goals should be to restore strength and motion as much as possible and to achieve a successful return to play.

STRENGTHENING

Quadriceps activation and strengthening are important goals early in the rehabilitation phase and can be attained through muscle contractions and straight leg raises.[28–30] Current rehabilitation protocols advocate for progressive strengthening exercises[29,30] along with neuromuscular training to improve strength, proprioception, balance, and muscle endurance.[30] Home exercise programs will help regain strength in the quadriceps, hamstrings, and hip muscles,[28–30] but it is important to consider the patients' age, maturity level, and parental involvement. Isokinetic testing during postoperative weeks 16 and 24 can help guide the rehabilitation program; if the peak torque deficit is less than 25% of the unaffected leg, more advanced and sport-specific training (including double leg hopping, jogging, agility drills, and double leg plyometric drills) may be initiated.[30] According to the HSS protocol, the patient maximizes leg strength during weeks 16 through 20 while the HSS injury prevention program is implemented.[29] One study has found that over 50% of

Figure 1. Early (**A**–**C**), and late (**D**–**E**) ACL rehabilitation exercises.
A. Quad sets, towel is placed under injured leg and patient contracts and relaxes their quadriceps muscle.
B. Straight leg raises. **C**. Quadriceps extensions. **D**. Step ups. **E**. Squats. **F**. One leg squat.

pediatric patients reach 85% of quadriceps strength between two and six months post-ACLR.[34] Another study, however, concluded that it takes longer for the pediatric population to regain quadriceps strength than the adult population; after 15 months, only 25% achieved a limb symmetry index (LSI) of greater than 90% on all strength and functional tests.[35] These results indicate the need for further research to determine the proper strength exercises and duration of rehabilitation for the pediatric population.

FUNCTIONAL TRAINING

The goal of rehabilitation in post-operative ACL-R patients is to achieve a functional and stable knee.[21] Many authors advocate functional exercises throughout the rehabilitation process for this purpose, including specific exercises that target neuromuscular control and muscle strength.[21,28–30] In the Children's Hospital of Philadelphia (CHOP) rehabilitation protocol, early functional exercises (weeks 4–16) include proprioceptive neuromuscular facilitation, progressive resistive exercises, leg presses, balance training, squats, single-leg squats, and step-ups. Progression to running, double-leg hop, plyometrics, and sport-specific activities is initiated only after certain functional milestones are met (**Figure 1**).[20,28,30] Similarly, the functional goals of the HSS protocol are to demonstrate an athletic-ready stance by week 20 and to feel confident with sport-specific movements by week 28.[29] An injury prevention program of neuromuscular training may help to maintain functional stability of the knee with both post-operative and non-operative management.[21]

RETURN TO SPORT

The ultimate goal of the surgical and/or rehabilitation process is to return the patient to the same type, intensity, and frequency of sport as before the injury occurred.[36,37] Returning to play too early places the patient at a greater risk of re-injury,[38] particularly in pediatric and adolescent patients.[39] Previously, subjective self-report measures and time-based criteria were used to assess sport readiness in both the adult and pediatric population.[35,40,41] Objective, functional testing throughout the rehabilitation process will help determine sport readiness at each stage. [21] Functional testing will reveal strength deficits through the presence of abnormal movement patterns, and it should be considered along with factors such as quadriceps strength, range of motion, and dynamic balance.[34,35]

Research on return to sport in the adult population has seen a paradigm shift moving away from time-based criteria towards more function-based criteria in order to individualize progress and plan the safest time to return to sport.[40] Joreitz et al[36] have created a protocol for adults comprised of functional goals, guidelines, and recommendations for returning to sport that is currently being studied. No criterion-based measures have been adequately studied, especially in the pediatric population.[36] Both the HSS and the CHOP protocols use a combination of time and functional criteria for return to sport. The HSS protocol allows return to sport after 28 weeks and achievement of at least 85% functional single leg hop test compared with the unaffected limb as well as dynamic control and lack of apprehension with sport-specific movements.[29] The CHOP protocol, on the other hand, requires that the patient must meet certain functional criteria and be nine months post-operation in order to return to sport.[28,30] There is a need for further research on the most effective criteria to ensure the safest and most efficient return to sport for the pediatric population.

CONCLUSION

Rehabilitation is crucial in the treatment of ACL injuries, particularly in the pediatric population. Children are often eager to return to their pre-injury level of athletic participation, which may place them at risk for re-injury if rehabilitation protocols are not adequately followed. Newer protocols incorporate functional benchmarks rather than time milestones to evaluate if patients have adequate strength and function to return to sport. Ultimately, the physician and therapist in conjunction with patients, parents, coaches, and trainers should clearly outline the goals and specific phases of ACL-R rehabilitation to align expectations, optimize outcomes, and increase the rates of successful return to sport.

References

1. Adirim TA, Cheng TL. Overview of injuries in the young athlete. *Sport Med*. 2003;33(1):75-81. doi:10.2165/00007256-200333010-00006.
2. Rechel JA, Collins CL, Comstock RD. Epidemiology of Injuries Requiring Surgery Among High School Athletes in the United States, 2005 to 2010. *J Trauma Inj Infect Crit Care*. 2011;71(4):982-989. doi:10.1097/TA.0b013e318230e716.
3. Swenson DM, Collins CL, Best TM, Flanigan DC, Fields SK, Comstock RD. Epidemiology of knee injuries among U.S. high school athletes, 2005/2006-2010/2011. *Med Sci Sports Exerc*. 2013;45(3):462-469. doi:10.1249/MSS.0b013e318277acca.
4. Beck NA, Lawrence JTR, Nordin JD, DeFor TA, Tompkins M. ACL Tears in School-Aged Children and Adolescents Over 20 Years. *Pediatrics*. 2017;139(3):e20161877. doi:10.1542/peds.2016-1877.
5. Dodwell ER, LaMont LE, Green DW, Pan TJ, Marx RG, Lyman S. 20 Years of Pediatric Anterior Cruciate Ligament Reconstruction in New York State. *Am J Sports Med*. 2014;42(3):675-680. doi:10.1177/0363546513518412.
6. Cruz AI, Lakomkin N, Fabricant PD, Lawrence JTR. Transphyseal ACL Reconstruction in Skeletally Immature Patients: Does Independent Femoral Tunnel Drilling Place the Physis at Greater Risk Compared With Transtibial Drilling? *Orthop J Sport Med*. 2016;4(6):2325967116650432. doi:10.1177/2325967116650432.
7. Chudik S, Beasley L, Potter H, Wickiewicz T, Warren R, Rodeo S. The Influence of Femoral Technique for Graft Placement on Anterior Cruciate Ligament Reconstruction Using a Skeletally Immature Canine Model with a Rapidly Growing Physis. *Arthrosc - J Arthrosc Relat Surg*. 2007;23(12):1309-1319.e1. doi:10.1016/j.arthro.2007.07.006.
8. Aichroth PM, Patel DV, Zorrilla P. The natural history and treatment of rupture of the anterior cruciate ligament in children and adolescents. *J Bone Jt Surg*. 2002;84(1):38-41. doi:10.1302/0301-620X.84B1.11773.
9. Schachter AK, Rokito AS. ACL injuries in the skeletally immature patient. *Orthopedics*. 2007;30(5):362-365. http://www.ncbi.nlm.nih.gov/pubmed/17539208. Accessed May 27, 2017.
10. Wojtys EM, Brower AM. Anterior cruciate ligament injuries in the prepubescent and adolescent athlete: Clinical and research considerations. *J Athl Train*. 2010;45(5):509-512. doi:10.4085/1062-6050-45.5.509.
11. Anderson CN, Anderson AF. Pediatric ACL: Evaluation and management. *Curr Orthop Pr*. 2014;25(4):312-320. doi:10.1097/bco.0000000000000134.
12. Kocher MS. Physeal Sparing Reconstruction of the Anterior Cruciate Ligament in Skeletally Immature Prepubescent Children and Adolescents. *J Bone Jt Surg*. 2005;87(11):2371. doi:10.2106/JBJS.D.02802.
13. McConkey MO, Bonasia DE, Amendola A. Pediatric anterior cruciate ligament reconstruction. *Curr Rev Musculoskelet Med*. 2011;4(2):37-44. doi:10.1007/s12178-011-9076-9.
14. Cruz AI, Fabricant PD, McGraw M, Rozell JC, Ganley TJ, Wells L. All-Epiphyseal ACL Reconstruction in Children: Review of Safety and Early Complications. *J Pediatr Orthop*. 2017;37(3):204-209. doi:10.1097/BPO.0000000000000606.
15. Lawrence JTR, Bowers AL, Belding J, Cody SR, Ganley TJ. All-epiphyseal anterior cruciate ligament reconstruction in skeletally immature patients. *Clin Orthop Relat Res*. 2010;468(7):1971-1977. doi:10.1007/s11999-010-1255-2.
16. Failla MJ, Logerstedt DS, Grindem H, et al. Does Extended Preoperative Rehabilitation Influence Outcomes 2 Years After ACL Reconstruction?: A Comparative Effectiveness Study Between the MOON and Delaware-Oslo ACL Cohorts. *Am J Sports Med*. 2016;44(10):2608-2614. doi:10.1177/0363546516652594.
17. van Grinsven S, van Cingel REH, Holla CJM, van Loon CJM. Evidence-based rehabilitation following anterior cruciate ligament reconstruction. *Knee Surgery, Sport Traumatol Arthrosc*. 2010;18(8):1128-1144. doi:10.1007/s00167-009-1027-2.

18. Yellin JL, Fabricant PD, Gornitzky A, et al. Rehabilitation Following Anterior Cruciate Ligament Tears in Children: A Systematic Review. *JBJS Rev*. 2016;4(1):e4. doi:10.2106/JBJS.RVW.O.00001.
19. Ludwig M, Atanda A. Management of anterior cruciate ligament tears in skeletally immature athletes. *Phys Sportsmed*. 2015;43(4):440-447. doi:10.1080/00913847.2015.1084213.
20. Moksnes H, Engebretsen L, Eitzen I, Risberg MA. Functional outcomes following a non-operative treatment algorithm for anterior cruciate ligament injuries in skeletally immature children 12 years and younger: A prospective cohort with 2 years follow-up. *Br J Sports Med*. 2013;47(8):488-494. doi:10.1136/bjsports-2012-092066.
21. Moksnes H, Engebretsen L, Risberg MA. Management of Anterior Cruciate Ligament Injuries in Skeletally Immature Individuals. *J Orthop Sport Phys Ther*. 2012;42(3):172-183. doi:10.2519/jospt.2012.3608.
22. Moksnes H, Engebretsen L, Risberg MA. Prevalence and Incidence of New Meniscus and Cartilage Injuries after a Nonoperative Treatment Algorithm for ACL Tears in Skeletally Immature Children. *Am J Sports Med*. 2013;41(8):1771-1779. doi:10.1177/0363546513491092.
23. Fabricant PD, Lakomkin N, Cruz AI, Spitzer E, Lawrence JTR, Marx RG. Early ACL reconstruction in children leads to less meniscal and articular cartilage damage when compared with conservative or delayed treatment. *J ISAKOS Jt Disord Orthop Sport Med*. 2016. http://jisakos.bmj.com/content/early/2016/01/21/jisakos-2015-000012?utm_source=TrendMD&utm_medium=cpc&utm_campaign=Journal_of_ISAKOS%253A_Joint_Disorders_%2526_Orthopaedic_Sports_Medicine_TrendMD_1. Accessed July 5, 2017.
24. Lawrence JTR, Argawal N, Ganley TJ. Degeneration of the Knee Joint in Skeletally Immature Patients with a Diagnosis of an Anterior Cruciate Ligament Tear. *Am J Sports Med*. 2011;39(12):2582-2587. doi:10.1177/0363546511420818.
25. Newman JT, Carry PM, Terhune EB, et al. Factors Predictive of Concomitant Injuries among Children and Adolescents Undergoing Anterior Cruciate Ligament Surgery. *Am J Sports Med*. 2015;43(2):282-288. doi:10.1177/0363546514562168.
26. Anderson CN, Anderson AF. Management of the Anterior Cruciate Ligament-Injured Knee in the Skeletally Immature Athlete. *Clin Sports Med*. 2017;36(1):35-52. doi:10.1016/j.csm.2016.08.003.
27. Alshewaier S, Yeowell G, Fatoye F. The effectiveness of preoperative exercise physiotherapy rehabilitation on the outcomes of treatment following anterior cruciate ligament injury: a systematic review. *Clin Rehabil*. 2017;31(1):34-44. doi:10.1177/0269215516628617.
28. Greenberg EM, Albaugh J, Ganley TJ, Lawrence JTR. Rehabilitation considerations for all epiphyseal ACL reconstruction. *Int J Sports Phys Ther*. 2012;7(2):185-196. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3325638/pdf/ijspt-07-185.pdf. Accessed May 21, 2017.
29. Fabricant PD, Jones KJ, Delos D, et al. Reconstruction of the Anterior Cruciate Ligament in the Skeletally Immature Athlete: A Review of Current Concepts. *J Bone Jt Surgery-American Vol*. 2013;95(5):e28-1-13. doi:10.2106/JBJS.L.00772.
30. Akinleye SD, Sewick A, Wells L. All-epiphyseal ACL reconstruction: a three-year follow-up. *Int J Sport Phys Ther*. 2013;8(3):300-310. http://www.ncbi.nlm.nih.gov/pubmed/23772346. Accessed June 8, 2017.
31. Nwachukwu BU, McFeely ED, Nasreddine A, et al. Arthrofibrosis after Anterior Cruciate Ligament Reconstruction in Children and Adolescents. *J Pediatr Orthop*. 2011;31(8):811-817. doi:10.1097/BPO.0b013e31822e0291.
32. Makhni EC, Crump EK, Steinhaus ME, et al. Quality and Variability of Online Available Physical Therapy Protocols From Academic Orthopaedic Surgery Programs for Anterior Cruciate Ligament Reconstruction. *Arthrosc J Arthrosc Relat Surg*. 2016;32(8):1612-1621. doi:10.1016/j.arthro.2016.01.033.
33. Makhni EC, Crump EK, Steinhaus ME, et al. Quality and Variability of Online Available Physical Therapy Protocols From Academic Orthopaedic Surgery Programs for Anterior Cruciate Ligament Reconstruction. *Arthrosc J Arthrosc Relat Surg*. 2016;32(8):1612-1621. doi:10.1016/j.arthro.2016.01.033.
34. Wells L, Dyke JA, Albaugh J, Ganley T. Adolescent anterior cruciate ligament reconstruction: a retrospective analysis of quadriceps strength recovery and return to full activity after surgery. *J Pediatr Orthop*. 2009;29(5):486-489. doi:10.1016/S0162-0908(09)79559-5.
35. Greenberg EM, Greenberg ET, Ganley TJ, Lawrence JTR. Strength and Functional Performance Recovery after Anterior Cruciate Ligament Reconstruction in Preadolescent Athletes. *Sports Health*. 2014;6(4):309-312. doi:10.1177/1941738114537594.
36. Joreitz R, Lynch A, Rabuck S, Lynch B, Davin S, Irrgang J. Patient-specific and surgery-specific factors that affect return to sport after ACL reconstruction. *Int J Sports Phys Ther*. 2016;11(2):264-278. http://www.ncbi.nlm.nih.gov/pubmed/27104060. Accessed May 21, 2017.
37. Fabricant PD, Lakomkin N, Cruz AI, Spitzer E, Marx RG. ACL reconstruction in youth athletes results in an improved rate of return to athletic activity when compared with non-operative treatment: a systematic review of the literature. *J ISAKOS Jt Disord Orthop Sport Med*. 2016. http://jisakos.bmj.com/content/early/2016/01/27/jisakos-2015-000013?utm_source=TrendMD&utm_medium=cpc&utm_campaign=Journal_of_ISAKOS%253A_Joint_Disorders_%2526_Orthopaedic_Sports_Medicine_TrendMD_1. Accessed July 5, 2017.
38. Dekker TJ, Rush JK, Schmitz MR. What's New in Pediatric and Adolescent Anterior Cruciate Ligament Injuries? *J Pediatr Orthop*. 2016;0(0):1-8. doi:10.1097/BPO.0000000000000792.
39. Dekker TJ, Godin JA, Dale KM, Garrett WE, Taylor DC, Riboh JC. Return to Sport After Pediatric Anterior Cruciate Ligament Reconstruction and Its Effect on Subsequent Anterior Cruciate Ligament Injury. *J Bone Joint Surg Am*. 2017;99(11):897-904. doi:10.2106/JBJS.16.00758.
40. Keller M, Kurz E, Schmidtlein O, Welsch G, Anders C. [Interdisciplinary Assessment Criteria for Rehabilitation after Injuries of the Lower Extremity: A Function-Based Return to Activity Algorithm]. *Sportverletz Sportschaden*. 2016;30(1):38-49. doi:10.1055/s-0042-100966.
41. Barber-Westin SD, Noyes FR. Factors used to determine return to unrestricted sports activities after anterior cruciate ligament reconstruction. *Arthrosc - J Arthrosc Relat Surg*. 2011;27(12):1697-1705. doi:10.1016/j.arthro.2011.09.009.

Authors

Steven F. DeFroda, MD, MEng; Department of Orthopaedics, Alpert Medical School of Brown University, Providence, RI.

Kathryn Hiller, BS; Department of Orthopaedics, Alpert Medical School of Brown University, Providence, RI.

Aristides I. Cruz, Jr., MD, MBA; Department of Orthopaedics, Alpert Medical School of Brown University, Providence, RI.

Correspondence

Steven F. DeFroda, MD
Department of Orthopaedics
Alpert Medical School of Brown University
593 Eddy Street
Providence, RI 02903
401-444-4030
Fax 401-444-6182

Leadless Cardiac Pacemakers: The Next Evolution in Pacemaker Technology

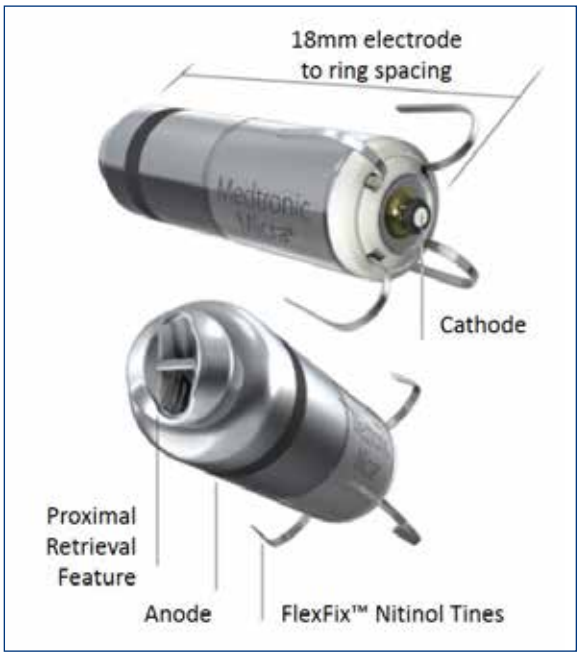
BRIAN D. McCAULEY, MD, MPH; ANTONY F. CHU, MD

ABSTRACT

Implantable pacemakers stand as a mainstay in our therapeutic arsenal, affording those suffering from advanced cardiac conduction system disease both an improved quality of life and reduced mortality. Annually, over 225,000 new pacemakers are implanted in the United States for bradyarrhythmias and heart block. The first implantable transvenous pacemakers appeared in 1965; they were bulky devices, hobbled by a short battery life, and a single pacing mode. Modern transvenous pacemakers have evolved considerably with significant improvements in battery life, pacing options, and lead technology but are still subject to a spectrum of complications stemming from either the subcutaneous pocket or the leads, including: hematoma, infection, wound dehiscence, pneumothorax, cardiac tamponade, lead dislodgment, upper extremity deep vein thrombosis, lead failure, venous obstruction, tricuspid valve insufficiency, and endocarditis. Single-chamber right ventricular (RV) leadless cardiac pacemakers, a concept from the past, has been revitalized to address these complications. Improvements in battery life, device miniaturization, catheter-based delivery tools, and advanced programming have made leadless cardiac pacemakers a viable option. In this review, we will discuss single-component leadless cardiac pacemaker technology, provide an overview of the two approved devices, and discuss their benefits as well as their limitations.

KEYWORDS: arrhythmias, cardiac; cardiac pacing, artificial; leadless pacing; pacemaker, artificial

Medtronic's Leadless Micra Transcatheter Pacing System (TPS).



INTRODUCTION

Implantable pacemakers stand as a mainstay in our therapeutic arsenal, affording those suffering from advanced cardiac conduction system disease both an improved quality of life and reduced mortality (1-4). Annually, over 225,000 new pacemakers are implanted in the United States for bradyarrhythmias and heart block (5). The first implantable transvenous pacemakers appeared in 1965. They were bulky devices, hobbled by a short battery life, and a single pacing mode. Modern transvenous pacemakers have evolved considerably with significant improvements in battery life, pacing options, and lead technology, but are still inserted similar to the original devices from the 1960s; with a pectoral-placed pulse generator connected to intracardiac transvenous pacing leads (6). Accordingly, while modern pacemakers are significantly more elegant devices, unfortunately they share many complications with their predecessors. In the short term, the subcutaneous pocket, in which the generator lies, is subject to hematoma, infection, and wound dehiscence. Intervention for cutaneous pocket complications increases the infection risk 15-fold (7-9). In addition to the risk posed by the subcutaneous pocket, transvenous leads have both short- and long-term sequelae. In the acute setting, the insertion of transvenous leads can lead to pneumothorax, cardiac tamponade, lead dislodgment, or upper extremity deep vein thrombosis and complication rates may be as high as 8% to 12% (10,11). Long-term complications from transvenous leads include lead failure, venous obstruction, tricuspid valve insufficiency, and endocarditis (11,12). In the simplest of the modern pacing configurations, a subcuta-

neous generator with a single-chamber lead, more than 1 in every 40 implants will require surgical intervention within 3 months of implantation; half of which are attributable to lead issues (12-14). Additionally, over the life of the device, lead failures are associated with significant morbidity (11).

In the 1970s, physicians, in concert with device engineers, developed the idea of a leadless pacemaker. They successfully implanted a self-contained RV cardiac pacemaker in a canine model with induced complete heart block, achieving 66 days of captured pacing. However given the technologic limitations of battery life inherent to the 1970s, the idea was shelved (15). Recently, advances in battery life, device miniaturization, improved catheter-based delivery tools, and advanced programming to optimize power consumption have assisted in making leadless cardiac pacemakers a viable option (16-19). In this review, we will discuss single-component leadless cardiac pacemaker technology, provide an overview of the two main devices, and consider their benefits and limitations.

SINGLE-COMPONENT LEADLESS CARDIAC PACEMAKERS

Currently, there are two single-component leadless cardiac pacemakers: 1) the Micra Transcatheter Pacing System (TPS; Medtronic) which received FDA approval in April 2016 and 2) the Nanostim Leadless Cardiac Pacemaker (LCP; St. Jude Medical) which is currently waiting FDA approval. Both devices are fully self-contained units, transcutaneously deployed via the femoral approach and capable of single-chamber RV pacing, sensing, and rate responsiveness. The Nanostim LCP is longer than the Micra TPS (42 mm versus 25.9 mm), but they displace similar volumes (1.0 and 0.8 mL). While the implantation procedure for both devices is similar, the Micra TPS requires a 27 French delivery sheath, while the Nanostim LCP uses a smaller 21 French delivery sheath. Once the device is advanced to the RV, site selection is accomplished via contrast-enhanced visualization. The pacemaker is then deployed using 4 self-expanding nitinol tines (Micra TPS) or an active screw-in helix with 3 angled nitinol tines perpendicular to the helix (Nanostim LCP). Following anchoring of the pacemaker, electrical parameters are assessed, internal fixation is tested using a gentle tug test, and the device is then deployed from the sheath.

Retrievability is an important consideration for leadless cardiac pacemakers. The Nanostim LCP has a dedicated steerable retrieval system. If a retrieval is warranted, the distal cap of the pacemaker is captured by the snare of the retrieval catheter, then rotated counter-clockwise to release the device from the myocardium. The Micra TPS does not have a dedicated retrieval system, but in several cases has been retrieved by employing a conventional goose-neck snare through a guiding sheath. Neither system has demonstrated long-term retrievability in humans, but has been demonstrated in animal models.

CLINICAL DATA: SAFETY AND PERFORMANCE

The data regarding the safety and efficacy of leadless pacemakers was spearheaded by the LEADLESS Trial. Using the Nanostim LCP device, 33 patients (mean age 77 years, 67% male) at 3 centers were enrolled over a four-month period in 2012-2013. The Nanostim LCP was successfully implanted in 32/33 (97%) patients. Overall, freedom from complications was 94% (31/33) at 90-day follow-up. There was one major complication: a 70-year-old male experienced cardiac tamponade with hemodynamic collapse during Nanostim LCP implantation, requiring emergent cardiac surgery, and died of an ischemic stroke due to a sub-therapeutic INR 3 weeks later (17).

A second clinical study, for safety and efficacy of the Nanostim LCP system, LEADLESS III, was prospectively performed in 56 centers in 3 countries (Australia, Canada, and USA) and enrolled 526 patients (mean age 75 years, 62% male), 300 with a minimum follow-up of 6 months. The Nanostim LCP was successfully implanted in 504/526 (95.8%), mean procedure time was 28.6 ± 17.8 minutes, and 70% of deployed devices did not require repositioning. Device-related serious adverse events occurred in 34 (6.5%) of patients and included pericardial effusion 1.5%, vascular complication 1.2%, dislodgment 1.1% (4 to pulmonary vein, 2 to femoral vein), and device retrieval 0.8% due to elevated pacing thresholds (18). Of note, St. Jude, manufacturer of the Nanostim LCP, issued a battery advisory due to a malfunction in 7/1423 (0.5%) devices, occurring between months 29–37 following implantation. The malfunction caused abrupt battery depletion, eliciting a loss of pacing and failed communication.

The Micra TPS Trial was a global, multicenter prospective study aimed at device safety and efficacy. The study enrolled 725 patients (mean age 75, 58.8% male) who met guidelines for RV pacing. The Micra TPS was successfully implanted in 719/725 (99.2%), with a mean procedure duration of 23.0 ± 15.3 minutes. Device-related serious adverse events occurred in 25 (3.4%) patients and included cardiac perforation 1.5%, vascular complications in 0.7%, venous thromboembolism in 0.3%, and increasing pacing thresholds in 0.3% of patients. No device dislodgment or embolization were observed. There was one major complication: a 77-year-old woman with end-stage renal failure, who underwent concomitant atrioventricular nodal ablation during the Micra TPS implant, died. Her death was likely attributable to a metabolic acidosis from her underlying renal failure and a prolonged procedure time (18).

POTENTIAL BENEFITS/LIMITATION TO LEADLESS CARDIAC PACING

The most obvious benefit of leadless pacing technology stems from mitigation of the short- and long-term risks associated with the traditional subcutaneous pocket with transvenous leads. The Nanostim LCP has a battery life that is

comparable to that of standard, single-chamber transvenous pacemakers. The similarity in battery life, despite the discrepancy in battery size, is directly attributable to the direct contact of the leadless cardiac pacemaker with the myocardium. The resulting reduction in impedance yields a significant reduction in current requirements. For the Nanostim LCP, the per beat requirement is 1.0 µA versus 6.24 µA for a standard transvenous pacemaker from the same device manufacturer (17-22).

On the other hand, the Micra TPS has approximately one half the battery life of the Nanostim LCP. The decreased battery life is based on two differences between the devices; the Micra TPS has a smaller battery (120 vs 248 mAh), and it uses radiofrequency telemetry instead of conductive telemetry. The predicted battery life for the Micra TPS at nominal settings is approximately 9.6 years, while the Nanostim LCP at the same settings would last 14.7 years. However, given that pacing is a dynamic, often user dependent process, there may be some variability in the actual device battery life. Only long-term follow-up will accurately delineate the true life of these devices. Additionally, both devices are believed to be safe for conditional use in magnetic resonance imaging due to a lack of ferromagnetic construction materials. Follow-up studies will need to be conducted to confirm their safety.

The most vexing limitation of the current generation of leadless cardiac pacemakers is their ability to only provide single-chamber RV pacing, barring the devices from placement in individuals requiring dual-chambered pacing or those in need of cardiac resynchronization therapy (23). Single-chamber RV pacing represents a relative minority, 15–30% of patients in Western countries (5). Another potentially concerning, but as of yet unseen complication, is the possibility of chronic device embolization. Also, the relatively large femoral venous sheath size (Nanostim LCP at 21F or Micra TPS at 27F) introduces the potential for both vascular and perforative complications, such as the terminal perforation that happened during the LEADLESS trial. Retrievability or the ability to place multiple devices at end-of-device life is a potential problem that remains to be resolved. While long-term device retrieval has been proven in animal models, it has yet to be done in humans. Lastly, given the relative small displacement by either leadless cardiac pacemaker, ~ 5mL, there is a possibility to deploy multiple devices if retrieval becomes onerous, but again this is a theoretical solution without current supporting clinical data.

CONCLUSION

While leadless cardiac pacemakers offer a potential means to circumvent some of the short- and long-term complications inherent to traditional subcutaneous transvenous pacemakers, many questions remain unanswered. Both the Micra TPS and the Nanostim LCP have demonstrated similar performance and safety. However, since no data is currently available to determine the long-term viability of leadless cardiac pacemakers, we will need to rely on future randomized clinical trials, and registry data to see if the potential benefits of the leadless cardiac pacing systems will supersede their conventional counterparts.

References

1. Epstein AE, Dimarco JP, Ellenbogen KA, et al. ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices) developed in collaboration with the American Association for Thoracic Surgery and Society of Thoracic Surgeons. J Am Coll Cardiol. 2008;51(21):e1-62. PMID: 18498951.
2. Ellenbogen KA, Kay GN, Wilkoff BL et al. Clinical Cardiac Pacing, Defibrillation, and Resynchronization Therapy. Saunders; 2011. ISBN: 1437716164.
3. Andersen HR, Nielsen JC, Thomsen PE, et al. Long-term follow-up of patients from a randomised trial of atrial versus ventricular pacing for sick-sinus syndrome. Lancet. 1997;350(9086):1210-6. PMID: 9652562.
4. Toff WD, Camm AJ, Skehan JD. Single-chamber versus dual-chamber pacing for high-grade atrioventricular block. N Engl J Med. 2005;353(2):145-55. PMID: 16014884.
5. Mond HG, Proclemer A. The 11th world survey of cardiac pacing and implantable cardioverter-defibrillators: calendar year 2009--a World Society of Arrhythmia's project. Pacing Clin Electrophysiol. 2011;34(8):1013-27. PMID: 21707667.
6. Jeffrey K, Parsonnet V. Cardiac pacing, 1960-1985: a quarter century of medical and industrial innovation. Circulation. 1998;97(19):1978-91. PMID: 9609092.
7. Mallela VS, Ilankumaran V, Rao NS. Trends in cardiac pacemaker batteries. Indian Pacing Electrophysiol J. 2004;4(4):201-12. PMID: 16943934.
8. Van rees JB, De bie MK, Thijssen J, Borleffs CJ, Schalij MJ, Van erven L. Implantation-related complications of implantable cardioverter-defibrillators and cardiac resynchronization therapy devices: a systematic review of randomized clinical trials. J Am Coll Cardiol. 2011;58(10):995-1000. PMID: 21867832.
9. Klug D, Balde M, Pavin D, et al. Risk factors related to infections of implanted pacemakers and cardioverter-defibrillators: results of a large prospective study. Circulation. 2007;116(12):1349-55. PMID: 17724263.
10. Korkeila P, Nyman K, Ylitalo A, et al. Venous obstruction after pacemaker implantation. Pacing Clin Electrophysiol. 2007;30(2):199-206.
11. Wiegand UK, Bode F, Bonnemeier H, Eberhard F, Schlei M, Peters W. Long-term complication rates in ventricular, single lead VDD, and dual chamber pacing. Pacing Clin Electrophysiol. 2003;26(10):1961-9. PMID: 14516336.
12. Kirkfeldt RE, Johansen JB, Nohr EA, Jørgensen OD, Nielsen JC. Complications after cardiac implantable electronic device implantations: an analysis of a complete, nationwide cohort in Denmark. Eur Heart J. 2014;35(18):1186-94. PMID: 24347317.
13. Udo EO, Zuithoff NP, Van hemel NM, et al. Incidence and predictors of short- and long-term complications in pacemaker therapy: the FOLLOWPACE study. Heart Rhythm. 2012;9(5):728-35. PMID: 22182495.
14. Hauser RG, Hayes DL, Kallinen LM, et al. Clinical experience with pacemaker pulse generators and transvenous leads: an 8-year prospective multicenter study. Heart Rhythm. 2007;4(2):154-60. PMID: 17275749.

15. Spickler JW, Rasor NS, Kezdi P, Misra SN, Robins KE, Leboeuf C. Totally self-contained intracardiac pacemaker. J Electrocardiol. 1970;3(3-4):325-31. PMID: 5517593.
16. Reddy VY, Knops RE, Sperzel J, et al. Permanent leadless cardiac pacing: results of the LEADLESS trial. Circulation. 2014;129(14):1466-71. PMID: 24664277.
17. Reddy VY, Exner DV, Cantillon DJ, et al. Percutaneous Implantation of an Entirely Intracardiac Leadless Pacemaker. N Engl J Med. 2015;373(12):1125-35. PMID: 26321198.
18. Reynolds D, Duray GZ, Omar R, et al. A Leadless Intracardiac Transcatheter Pacing System. N Engl J Med. 2016;374(6):533-41. PMID: 26551877.
19. Knops RE, Tjong FV, Neuzil P, et al. Chronic performance of a leadless cardiac pacemaker: 1-year follow-up of the LEADLESS trial. J Am Coll Cardiol. 2015;65(15):1497-504. PMID: 25881930.
20. Berger T, Roithinger FX, Antretter H, Hangler H, Pachinger O, Hintringer F. The influence of high versus normal impedance ventricular leads on pacemaker generator longevity. Pacing Clin Electrophysiol. 2003;26(11):2116-20. PMID: 14622313.
21. De voogt WG. Pacemaker leads: performance and progress. Am J Cardiol. 1999;83(5B):187D-191D. PMID: 10089864.
22. Markewitz A, Kronski D, Kammeyer A, et al. Determinants of dual chamber pulse generators longevity. Pacing Clin Electrophysiol. 1995;18(12 Pt 1):2116-20. PMID: 8771121.
23. Gillis AM, Russo AM, Ellenbogen KA, et al. HRS/ACCF expert consensus statement on pacemaker device and mode selection. J Am Coll Cardiol. 2012;60(7):682-703. PMID: 22854177.

Authors

Brian D. McCauley, MD, MPH, Internal Medicine Resident, The Warren Alpert Medical School of Brown University, Providence, RI.

Antony F. Chu, MD, is the Director of Complex Ablation, Arrhythmia Services Section, Warren Alpert Medical School of Brown University, Providence, RI.

Correspondence

Antony F. Chu, MD
Cardiovascular Institute
950 Warren Ave., 2nd floor
East Providence, RI 02914
401-606-1004
achu1@lifespan.org

Influenza A Infection and Anaphylaxis in a Pediatric Patient Hospitalized for Asthma Exacerbation

ERIC J. CHOW, MD, MS, MPH; IVONA SEDIVA, MD

ABSTRACT

Influenza infections can cause a variety of different systemic problems beyond respiratory symptoms. A 4-year-old boy with a history of atopy, infected with influenza A, presented to our hospital with an asthma exacerbation developed symptoms of anaphylaxis. He was treated with a full course of oseltamivir and symptoms resolved without recurrence of allergic reaction. Infections have been implicated as causes of anaphylaxis but this has mostly been limited to parasites. While viral infections have been documented as causes of urticarial rashes, anaphylaxis due to viral infection has only been reported once, in an adult patient. There have not been any reports of anaphylaxis related to influenza infections. Early recognition and treatment of patients with influenza may prevent progression of systemic allergic reactions.

KEYWORDS: influenza, anaphylaxis, urticaria, allergy, asthma

INTRODUCTION

Influenza infections are a major cause of morbidity and mortality every year. Between 2015 to 2016, there were 24.6 million cases of influenza, 95.9 per 100,000 patients requiring hospitalization and almost 12,000 cases resulted in death.[1] Symptoms can include fever, cough, rhinitis, headaches and myalgias.[2] While skin manifestations of viral infections are common, they are an uncommon presentation in patients infected with influenza.[3] Influenza is a known precipitant of asthma exacerbation; however, anaphylaxis attributed to influenza has not previously been reported.[4] In this report we describe a case of anaphylaxis associated with influenza A infection in a pediatric patient hospitalized for an asthma exacerbation.

CASE REPORT

Our patient is a 4-year-old boy with a history of moderate persistent asthma and eczema who presented to the hospital with rhinorrhea, cough and shortness of breath, worsening over a two-day period. He was tachypneic with a fever of 39.2°C. He had decreased aeration on auscultation and combined diffuse inspiratory and expiratory wheezes. He had his baseline eczematous rash but no other skin findings. Respiratory viral testing was positive for influenza A by rapid influenza test (Xpert; Cepheid, Sunnyvale, CA). No other laboratory studies were drawn at the time of admission. Standard treatment for his asthma exacerbation was initiated, including beta agonist inhaler treatments and oral corticosteroids. His tachypnea persisted, with intermittent hypoxia. He was given continuous albuterol, magnesium and a normal saline bolus and admitted to the pediatric intensive care unit (PICU) for further management.

The patient's work of breathing improved with continued treatment of his asthma. He was also started on oseltamivir. Four hours later, the patient developed a diffuse urticarial skin eruption encompassing his trunk, face and extremities. He had no swelling or vomiting. Initially, his respiratory status remained stable. He was given intravenous (IV) diphenhydramine, which resulted in improvement of his rash. After a 2-hour observation period, he was transferred to the pediatric floor. Upon arrival, the patient redeveloped diffuse urticaria and became hypoxic to 80% on room air with significantly increased work of breathing. He was placed on a non-rebreather mask and was treated for anaphylaxis. The patient was given intramuscular epinephrine, IV ranitidine, albuterol and IV corticosteroids and he was transferred back to the PICU. Over the next 2 hours, his rash and work of breathing improved. His asthma improved and he had no further rash during his hospitalization. He was discharged home on hospital day three. Since discharge, his fever resolved and he completed his course of oseltamivir without further allergic symptoms. He has not had any further anaphylactic reactions since discharge.

During his hospitalization, the patient only ate food that he had previously tolerated. Latex gloves are not used in the hospital. An allergist has followed the patient in the outpatient setting and reports intermittent compliance with his home asthma controller medications. He has been seen in the emergency department for asthma symptoms four times a year prior to this hospitalization, with only one prior hospitalization. His asthma triggers included dust mites, dogs, mold, pollen and smoke exposure. The patient has a notable allergy to latex. He had a non-urticarial skin reaction when touching tomatoes but this had been localized to his mouth and hands. He had received inactivated influenza vaccines during the 2015–2016 and 2016–2017 influenza seasons without any adverse reactions. Allergy testing 2 years prior was notable for allergic reaction to dust mites, dog dander, mold, cats and pollen. During an outpatient follow-up, the allergy test was repeated and showed no change in test results.

DISCUSSION

Anaphylaxis is a systemic reaction to an allergen through the triggering of T helper 2 cells, activating mast cells and basophils. The resulting symptoms are life threatening.[5] The most common allergens include food, insect stings, and medications.[6] The diagnosis of anaphylaxis is based on the clinical presentation of cardiovascular, dermatologic, respiratory and gastrointestinal symptoms.[5] Three criteria were established to help physicians diagnose anaphylaxis generally involving two or more organ systems with or without the presence of a likely or known allergen.[7] There are two documented cases of anaphylaxis to oseltamivir, both in adults.[8,9] Our patient, however, completed his course of oseltamivir without any further reactions and thus the medication was unlikely the cause of the anaphylaxis experienced in the hospital.

A review of the literature showed that while bacterial, parasitic and viral infections, including influenza, have been implicated in the development of urticarial rashes, reports of infections causing anaphylaxis have been limited to case reports, mostly involving parasitic infections.[10] Only one case of a patient with anaphylaxis and associated viral upper respiratory infection (URI) symptoms was reported. This 64-year- old female had symptoms of cough and rhinitis without known exposures to antigens who developed anaphylactic shock.[11] Anaphylactic shock reoccurred when she developed URI symptoms within the same year; however, no viral testing was reported at the time of her symptoms.

The mechanism for allergic response following mast cell and basophil activation involves allergen-IgE interaction followed by a release of mediators including tryptase, tumor necrosis factor and histamine.[12] Mechanisms for influenza activation of the body's allergic response have been proposed. Influenza infects the body through the respiratory tract. The lung parenchyma is lined with cells that serve as a first line of defense against invading pathogens including macrophages, dendritic cells and mast cells.[13] Activation of mast cells leads to degranulation, which in turn promotes inflammatory responses and further recruitment of mast cells. Influenza A and other viruses are known causes of asthma exacerbation.[14] In patients with asthma, the accumulation of mast cells in the lung leads to worsening respiratory symptoms.[15] Grunewald et al suggest that viruses like influenza A can directly activate mast cell degranulation through IgE and viral antigen interaction.[16] Molecular mimicry is another plausible mechanism whereby viral antigens cross react with IgE specific to other allergens. In some cases, viral infections produce sufficient inflammatory response that they activate T helper 2 cell and subsequent allergic response. Future studies will be needed to further understand the complex molecular explanations for these interactions.

CONCLUSION

Recognizing influenza A and other viral infections as potential causes of systemic allergic response is required to reduce patient morbidity and mortality. Studies have shown that viral infections can induce pathways that trigger allergic reactions. This case of influenza A infection associated with anaphylaxis highlights the need to treat allergic symptoms early in patients with known history of atopy and viral respiratory infections.

References

1. Estimated Influenza Illnesses, Medical Visits, Hospitalizations and Deaths Averted by Vaccination in the United States. 2017; https://www.cdc.gov/flu/about/disease/2015-16.htm-methods. Accessed May 28, 2017.
2. Silvennoinen H, Peltola V, Lehtinen P, Vainionpää R, Heikkinen T. Clinical presentation of influenza in unselected children treated as outpatients. *Pediatr Infect Dis J.* 2009;28(5):372-375.
3. Drago F, Ciccarese G, Gasparini G, et al. Contemporary infectious exanthems: an update. *Future Microbiol.* 2017;12:171-193.
4. Flu and People with Asthma. 2017; https://www.cdc.gov/flu/asthma/. Accessed May 30, 2017, 2017.
5. Ma L, Danoff TM, Borish L. Case fatality and population mortality associated with anaphylaxis in the United States. *J Allergy Clin Immunol.* 2014;133(4):1075-1083.
6. Kemp SF, Lockey RF. Anaphylaxis: a review of causes and mechanisms. *J Allergy Clin Immunol.* 2002;110(3):341-348.
7. Sampson HA, Muñoz-Furlong A, Campbell RL, et al. Second symposium on the definition and management of anaphylaxis: summary report -- Second National Institute of Allergy and Infectious Disease/Food Allergy and Anaphylaxis Network symposium. *J Allergy Clin Immunol.* 2006;117(2):391-397.
8. Hirschfeld G, Weber L, Renkl A, Scharffetter-Kochanek K, Weiss JM. Anaphylaxis after Oseltamivir (Tamiflu) therapy in a patient with sensitization to star anise and celery-carrot-mugwort-spice syndrome. *Allergy.* 2008;63(2):243-244.
9. Canadian Adverse Drug Reaction Newsletter Volume 10, Number 4. 2000; http://publications.gc.ca/collections/Collection/H12-38-10-4E.pdf. Accessed June 25, 2017, 2017.
10. Wedi B, Raap U, Wieczorek D, Kapp A. Urticaria and infections. *Allergy Asthma Clin Immunol.* 2009;5(1):10.
11. Mazur N, Patterson R, Perlman D. A case of idiopathic anaphylaxis associated with respiratory infections. *Ann Allergy Asthma Immunol.* 1997;79(6):546-548.
12. Peavy RD, Metcalfe DD. Understanding the mechanisms of anaphylaxis. *Curr Opin Allergy Clin Immunol.* 2008;8(4):310-315.
13. Graham AC, Temple RM, Obar JJ. Mast cells and influenza a virus: association with allergic responses and beyond. *Front Immunol.* 2015;6:238.
14. Minor TE, Dick EC, Baker JW, Ouellette JJ, Cohen M, Reed CE. Rhinovirus and influenza type A infections as precipitants of asthma. *Am Rev Respir Dis.* 1976;113(2):149-153.
15. Zarnegar B, Mendez-Enriquez E, Westin A, et al. Influenza Infection in Mice Induces Accumulation of Lung Mast Cells through the Recruitment and Maturation of Mast Cell Progenitors. *Front Immunol.* 2017;8:310.
16. Grunewald SM, Hahn C, Wohlleben G, et al. Infection with influenza a virus leads to flu antigen-induced cutaneous anaphylaxis in mice. *J Invest Dermatol.* 2002;118(4):645-651.

Authors

Eric J. Chow, MD, MS, MPH, Departments of Medicine and Pediatrics, Warren Alpert Medical School of Brown University; Rhode Island Hospital and Hasbro Children's Hospital, Providence, RI.

Ivona Sediva, MD, Department of Pediatrics, Warren Alpert Medical School of Brown University; Division of Pediatric Critical Care, Hasbro Children's Hospital, Providence, RI.

Correspondence

Dr. Eric J Chow
Departments. of Medicine and Pediatrics,
Rhode Island Hospital, Hasbro Children's Hospital
245 Chapman Street, Suite 100, Providence, RI 02905
401-444-6072
Fax 401-444-8804

CADASIL as a Multiple Sclerosis Mimic

ANDREW J. BOULEY, MD; SHADI YAGHI, MD

KEYWORDS: CADASIL, stroke in young adults, multiple sclerosis, MRI

INTRODUCTION

Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is a well-described entity that causes progressive neurologic decline due to subcortical infarcts. We describe a patient diagnosed with CADASIL that has many atypical features suggestive of multiple sclerosis (MS) as an alternate diagnosis.

CASE REPORT

A 31-year-old man with no significant past medical or family history presented with a one-day history of left arm weakness and dysarthria. His blood pressure was 124/83 mm Hg, his heart rate was 63 and regular, and he was afebrile. The general physical examination and mental status were normal aside from dysarthria. He had mild, left upper motor neuron facial weakness, mild left hemiparesis of arm and leg, with mild left dysmetria and dysdiadochokinesia. Reflexes were 3+ on the left and 2+ on the right; Babinski sign was present on the left. Gait testing revealed left leg circumduction.

The head CT scan was unremarkable. The brain MRI revealed an area of restriction on diffusion weighted imaging (DWI) in the right corona radiata with extensive periventricular T2/FLAIR hyperintensities (**Figure 1**). Post-contrast brain MRI showed partial peripheral enhancement of the lesion that restricted on DWI (**Figure 2**). Magnetic resonance angiography (MRA) showed normal intracranial and extracranial vasculature. Transthoracic echocardiogram revealed normal left ventricular function without evidence of right-to-left shunting with agitated saline. ECG and inpatient cardiac telemetry showed normal sinus rhythm. Blood tests revealed a normal complete blood count and basic metabolic profile, low-density lipoprotein cholesterol of 165 mg/dl, urine toxicology screen positive for cannabinoids, and normal cerebrospinal fluid (CSF) profile with no oligoclonal bands and a normal IgG index. *NOTCH3* genetic testing revealed a heterozygous missense mutation, diagnostic of CADASIL.

Figure 1. Non-contrast MRI brain.
Axial FLAIR (A-C) and sagittal FLAIR (D) images showing extensive periventricular white matter disease with involvement of the external capsule and sparing of the temporal lobes and brainstem. Axial DWI and ADC (E and F) images showing restricted diffusion.

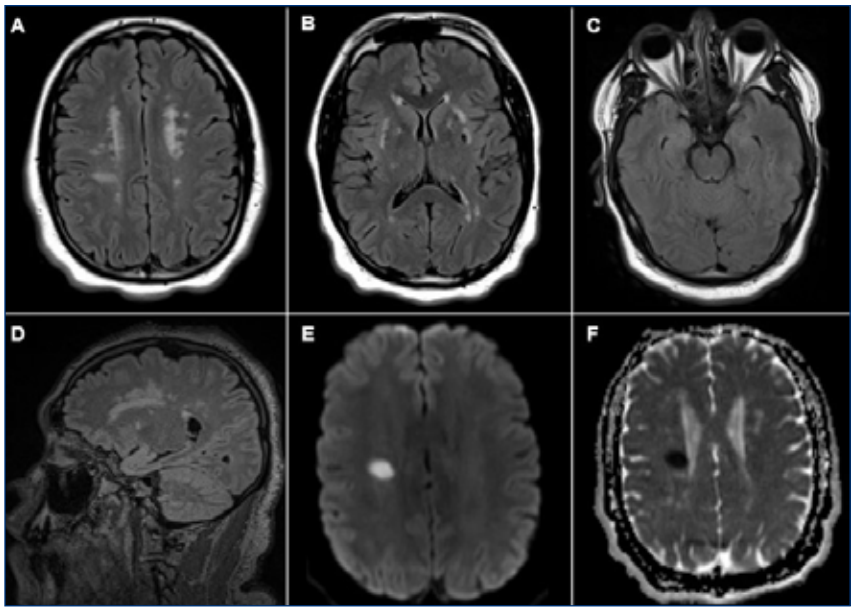
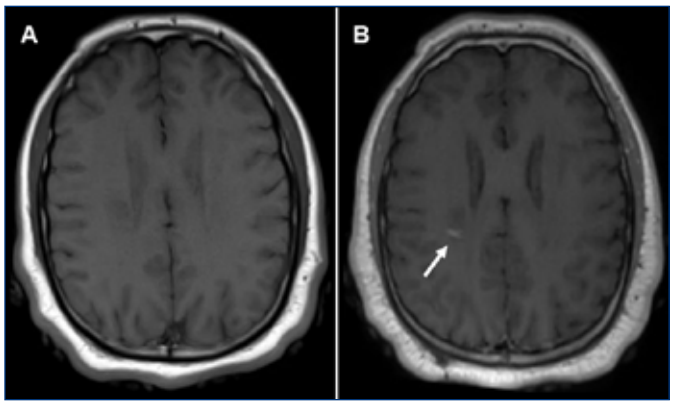


Figure 2. Pre- and post-contrast MRI brain.
Axial T1 pre-gadolinium (A) and post-gadolinium (B) images showing slight peripheral enhancement of the acute restricting lesion (arrow).



DISCUSSION

The clinical presentation and neuroimaging suggested ischemic stroke with small vessel vasculopathy involving the external capsule. This led us to send *NOTCH3* genetic testing in pursuit of CADASIL. However, atypical features of the patient's presentation led us to pursue other diagnoses. Due to the patient's age and the predominance of periventricular white matter disease with Dawson finger-like projections on the sagittal FLAIR MRI, there was strong suspicion for MS. Prior to the return of the *NOTCH3* genetic testing, the patient was empirically treated with a three-day course of intravenous methylprednisolone 1,000 mg daily while simultaneously being treated with aspirin and atorvastatin for secondary stroke prevention. Acute demyelinating lesions in MS typically have increased signal on DWI with increased signal on ADC, presumably due to vasogenic edema and T2 shine-through caused by the breakdown in the blood-brain barrier.[1,2] More rarely, acute demyelinating lesions can display a restricted diffusion pattern, leading to a diagnostic challenge for young adults presenting with stroke-like symptoms.[3] The enhancement of the lesion was not helpful in differentiating stroke from demyelinating disease since both disease processes can demonstrate peripheral ring enhancement. In cases such as these, CSF analysis can be helpful as the presence of oligoclonal bands is highly sensitive for MS.[4] Other disease entities, such as small vessel inflammatory and large vessel vasculitides, were lower on the differential given the lack of systemic symptoms, normal CSF profile, and normal MRA.

Stroke mechanisms in young adults include cardioembolism, dissection, moyamoya, vasculitis, drug abuse, hypercoaguable states, and neurogenetic disorders.[5] Our patient's lack of traditional vascular risk factors suggested a possible genetic or metabolic etiology. MRI findings of lacunar infarcts and white matter changes are particularly prevalent in CADASIL and cerebral autosomal recessive arteriopathy with subcortical infarcts and leukoencephalopathy (CARASIL).[6]

CADASIL is an autosomal dominant inherited small vessel disease of the central nervous system that leads to subcortical infarcts.[7] Over 150 causative mutations of *NOTCH3* on chromosome 19 have been reported.[7] While some groups have proposed incomplete penetrance for specific mutations,[8] the overall penetrance for CADASIL mutations is thought to be complete or near complete.[9] Although most reported cases are familial, de novo mutations can also occur; however, their frequency is not well defined.[10] The various mutations result in a change in the number of cysteines, causing structural changes in the transmembrane receptor *NOTCH3*.[11] This protein is involved in arterial development, and mutation leads to damaged smooth-muscle cells of vessels, fibrosis, and the accumulation of *NOTCH3* and granular osmiophilic material (GOM).[7,11] The vascular disease is an amyloid-negative arteriopathy that affects leptomeningeal and perforating arteries of the brain.[7] *NOTCH3* genetic testing is the gold standard for diagnosing CADASIL and is nearly 100% sensitive and specific. Skin biopsies are also diagnostic, where immunostaining reveals NOTCH3 in the vessel wall and electron microscopy reveals the presence of GOM in vascular smooth-muscle cells.[7]

Clinical features of CADASIL include migraines with aura, recurrent strokes, cognitive decline progressing to dementia, and psychiatric disturbances, typically with migraines being the presenting clinical feature. Intracranial hemorrhage and seizures are also common.[7,11] Commonly, the first clinical ischemic event occurs after 40 years of age, but it can occur by age 20.[7] MRI imaging shows diffuse white matter T2 hyperintensities, often periventricular, and involving the centrum semiovale, multiple circumscribed lacunar infarcts, and early brain atrophy. A propensity for involvement of the external capsule and the anterior poles of the temporal lobes is particularly suggestive of CADASIL.[7] Treatment is limited to symptomatic treatment of migraines and neuropsychiatric features, as well as secondary prevention of additional strokes via antiplatelet therapy, statins, and management of hypertension and diabetes when clinically indicated. Genetic counseling is mandatory.[7]

References

1. Schaefer P, Grant P, Gonzalez R. Diffusion-weighted MR imaging of the brain. *Radiology.* 2000;217(2):331-345.
2. Cotton F, Weiner H, Jolesz F, Guttmann C. MRI contrast uptake in new lesions in relapsing-remitting MS followed at weekly intervals. *Neurology.* 2003;60(4):640-646.
3. Balashov K, Lindzen E. Acute demyelinating lesions with restricted diffusion in multiple sclerosis. *Mult Scler.* 2012;18(12):1745-1753.
4. Przybek J, Gniatkowska I, Mirowska-Guzel D, Czlonkowska A. Evolution of diagnostic criteria for multiple sclerosis. *Neurol Neurochir Pol.* 2015;49(5):313-321.
5. Ji R, Schwamm L, Pervez M, Singhal A. Ischemic stroke and transient ischemic attack in young adults: risk factors, diagnostic yield, neuroimaging, and thrombolysis. *JAMA Neurol.* 2013;70(1):51-57.
6. Craggs L, Yamamoto Y, Deramecourt V, Kalaria R. Microvascular pathology and morphometrics of sporadic and hereditary small vessel diseases of the brain. *Brain Pathol.* 2014;24(5):495-509.
7. Chabriat H, Joutel A, Dichgans M, Tournier-Lasserve E, Bousser M. Cadasil. *Lancet Neurol.* 2009;8(7):643-653.
8. Vyshka G, Kruja J. Clinical variability of the cerebral autosomal-dominant arteriopathy with subcortical infarcts and leukoencephalopathy phenotype in two siblings of a large family showing the same mutation. *Int Med Case Rep J.* 2013;6:59-63.
9. Chabriat H, Vahedi K, Iba-Zizen M, et al. Clinical spectrum of CADASIL: a study of 7 families. Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy. *Lancet.* 1995;346(8980):934-939.
10. Joutel A, Dodick D, Parisi J, Cecillon M, Tournier-Lasserve E, Bousser M. De novo mutation in the Notch3 gene causing CADASIL. *Ann Neurol.* 2000;47(3):388-391.
11. Meng H, Zhang X, Yu G, Lee S, Chen Y, Prudovsky I, Wang M. Biochemical characterization and cellular effects of CADASIL mutants of NOTCH3. *PloS One.* 2012;7(9):e44964.

Authors

Andrew J. Bouley, MD, Department of Neurology, The Warren Alpert Medical School of Brown University, Providence, RI.

Shadi Yaghi, MD, Department of Neurology, The Warren Alpert Medical School of Brown University, Providence, RI.

Correspondence

Andrew J. Bouley, MD
Rhode Island Hospital, 593 Eddy Street, APC 5th Floor
Providence, RI 02903
617-688-4988, Fax 401-444-6858
andrew.j.bouley@gmail.com

Expect the unexpected: Rectus sheath hematoma comes without a notice

UMAMA GORSI, MD; VISHNU PRIYA MALLIPEDDI, MD

INTRODUCTION

Rectus sheath hematoma (RSH) is an unusual clinical entity that results from bleeding into the rectus sheath. RSH was first described nearly 2500 years ago, by Hippocrates and Galen who described it as a consequence of abdominal trauma (1). The first published case of RSH in the United States was in 1857 (2). The most common predisposing factors are anticoagulants, strenuous activities (e.g., cough, vomiting, exercise) and blunt abdominal trauma (3). There is an estimated rise in the RSH cases based on the increasing use of anticoagulants(2). Delayed recognition of RSH may result in complications like hemodynamic instability, abdominal compartment syndrome, multi-organ dysfunction and even death (4). We present a case of spontaneous rectus sheath hematoma resulting in hemodynamic instability, during anticoagulation therapy for acute pulmonary embolism, in a middle-aged female. We highlight the need for the physicians to consider RSH in the differential diagnosis list in high-risk patients on anticoagulants.

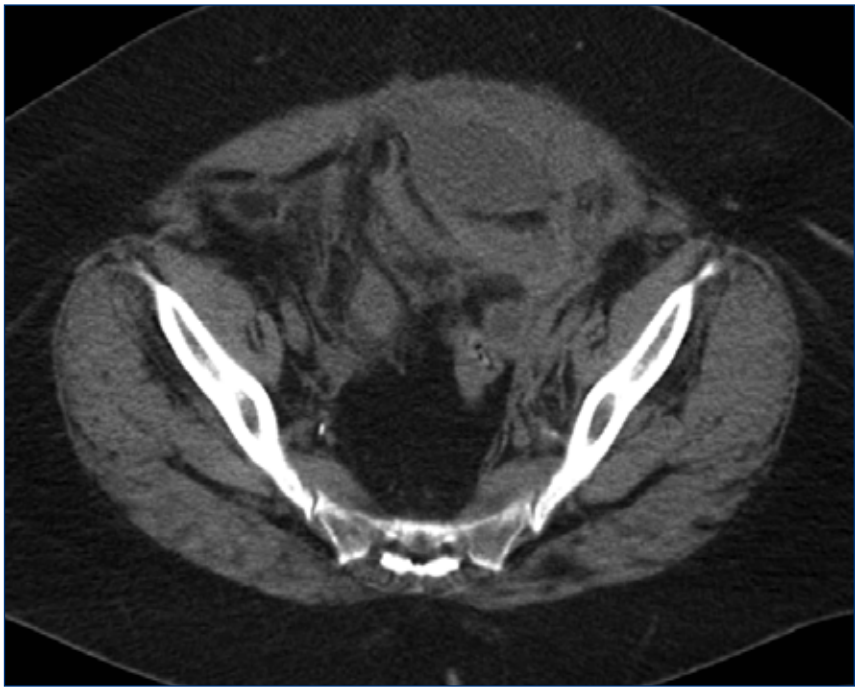
CASE SUMMARY

A 57-year-old obese woman with a history of hypertension, renal artery stenosis which was stented, presented with the complaints of worsening left-sided chest pain and shortness of breath for two days. She was taking clopidogrel as her only medication. She had a blood pressure of 108/56 mm Hg, pulse rate of 66 beats/min, respiratory rate of 22 breaths/min and oxygen saturation of 97%. Lungs were clear and auscultation of the precordium revealed no murmurs. The abdomen was soft, non-tender but an ecchymosis was noted in the left lower quadrant. The remainder of the physical examination was unremarkable. Laboratory investigations showed elevated D-dimer level. Computed tomography (CT) of the chest showed a right upper lobe and right lower lobe segmental pulmonary embolism. Immediately, IV heparin was started. But within 24 hours, the patient's blood pressure dropped to 80 mm Hg and heart rate rose to 110 beats/min. There was a drop in hemoglobin from 11.3 to 8.2g/dl in the preceding three hours. The patient was given intravenous fluids. IV heparin and clopidogrel were held temporarily. She had developed a 20cm ecchymosis in the left lower abdominal wall, severe tenderness in the left lower abdominal quadrant and severe abdominal pain on straight leg raise (positive Carnett's sign) in a supine position. CT of the abdomen confirmed the diagnosis of rectus sheath hematoma (RSH). The patient was transferred to the intensive care. Fluid resuscitation was started. IV Heparin and clopidogrel were discontinued. An inferior vena cava filter was placed and anticoagulation was resumed. Close monitoring of bleeding and coagulation markers for 3 months was planned along with a decision to remove the filter at that time.

DISCUSSION

Rectus sheath hematoma is the most common primary non neoplastic disorder of the rectus abdominis muscle(5). It occurs due to the accumulation of blood within the rectus abdominis muscle either due to bleeding from the inferior

Figure 1. CT scan showing bilateral hematoma left more than right.



epigastric artery or the superior epigastric artery or their branches, or occasionally from direct tears of the rectus abdominis muscle (6). RSH affect women twice as often as men, generally in the fifth and seventh decades of life (7) due to a smaller rectus abdominis muscle mass and an inability to tamponade the bleeding (2). The main risk factors for RSH are anticoagulant therapy, hematological disorders, trauma, strenuous physical activity, coughing, sneezing, and pregnancy (8). In a review of 126 cases of rectus sheath hematoma, almost 70% of the patients were on anticoagulation therapy, while 24% of them were on simultaneous anticoagulation and antiplatelet therapies (8).

Eliciting signs on physical examination helps in differentiating abdominal wall pathologies (9). Carnett and Fothergill signs are elicited by flexing the neck with the patient supine. In Carnett's sign, the pain and tenderness persist or increase with palpation of the abdominal mass in RSH and decrease with intra-abdominal pathology. In Fothergill sign, the hematoma remains fixed and palpable in RSH whereas, impalpable in an intra-abdominal mass(10). The patient had positive Carnett's and Fothergill signs.

Although ultrasonography of the abdomen is preferred in pregnant women, pediatric population and in patients with acute renal failure, its sensitivity for RSH is only 71%. CT abdomen with IV contrast is the diagnostic imaging modality of choice with 100% diagnostic success rate (11)and is considered superior to ultrasonography. A hyperdense mass posterior to the rectus abdominis muscle with ipsilateral anterolateral muscular enlargement are characteristic of acute RSH, although chronic RSH may present as an isodense or hypodense mass relative to the rectus abdominis muscle on CT scan abdomen (12).

Three tiers of RSH severity have been proposed. Type I RSH is intramuscular, does not cross the midline or dissect along the fascial planes. Type II RSH is intramuscular, may cross the midline, with blood seeping between the muscle and the transversalis fascia excluding prevesical space. Type III RSH may or may not involve muscle but blood is found between the muscle and the transversalis fascia, in the peritoneum or prevesical space of Retzius (13). Although rectus sheath hematoma is self-limiting, it is associated with an overall mortality of approximately 4% whereas for those on anticoagulant therapy, it is 25% (7).

RSH is most commonly managed conservatively in the majority of RSH cases (14). It consists of bed rest, analgesia, hematoma compression, ice packs application, fluid resuscitation and most importantly, discontinuation of anticoagulants. Type I and type II RSH are managed conservatively. Type III RSH is usually managed by blood transfusion and invasive treatment (13). We encountered a Type III RSH in our patient who was hemodynamically unstable, managed conservatively by aggressive fluid resuscitation, discontinuation of IV heparin and clopidogrel and placing an IVC filter.

Reinitiating anticoagulation therapy is always a concern and the decision must be individualized.

References

1. Manier JW. Rectus sheath hematoma. Am J *Gastroenterol*. 1972;57(5):443-52.
2. Hatjipetrou A, Anyfantakis D, Kastanakis M. Rectus sheath hematoma: a review of the literature. *Int J Surg*. 2015;13:267-71.
3. Alla VM, Karnam SM, Kaushik M, Porter J. Spontaneous rectus sheath hematoma. *West J Emerg Med*. 2010;11(1):76-9.
4. Kocayigit I, Can Y, Sahinkus S, Aydin E, Vatan MB, Kilic H, et al. Spontaneous rectus sheath hematoma during rivaroxaban therapy. *Indian J Pharmacol*. 2014;46(3):339-40.
5. Lambroza A, Tighe MK, DeCosse JJ, Dannenberg AJ. Disorders of the rectus abdominis muscle and sheath: a 22-year experience. *Am J Gastroenterol*. 1995;90(8):1313-7.
6. Fitzgerald JE, Fitzgerald LA, Anderson FE, Acheson AG. The changing nature of rectus sheath haematoma: case series and literature review. *Int J Surg*. 2009;7(2):150-4.
7. Buffone A, Basile G, Costanzo M, Veroux M, Terranova L, Basile A, et al. Management of patients with rectus sheath hematoma: Personal experience. *J Formos Med Assoc*. 2015;114(7):647-51.
8. Cherry WB, Mueller PS. Rectus sheath hematoma: review of 126 cases at a single institution. *Medicine* (Baltimore). 2006;85 (2):105-10.
9. Suleiman S, Johnston DE. The abdominal wall: an overlooked source of pain. *Am Fam Physician*. 2001;64(3):431-8.
10. Auten JD, Schofer JM, Banks SL, Rooney TB. Exercise-induced bilateral rectus sheath hematomas presenting as acute abdominal pain with scrotal swelling and pressure: case report and review. *J Emerg Med*. 2010;38(3):e9-12.
11. Moreno Gallego A, Aguayo JL, Flores B, Soria T, Hernandez Q, Ortiz S, et al. Ultrasonography and computed tomography reduce unnecessary surgery in abdominal rectus sheath haematoma. *Br J Surg*. 1997;84(9):1295-7.
12. Fukuda T, Sakamoto I, Kohzaki S, Uetani M, Mori M, Fujimoto T, et al. Spontaneous rectus sheath hematomas: clinical and radiological features. *Abdom Imaging*. 1996;21(1):58-61.
13. Berna JD, Garcia-Medina V, Guirao J, Garcia-Medina J. Rectus sheath hematoma: diagnostic classification by CT. *Abdom Imaging*. 1996;21(1):62-4.
14. Rimola J, Perendreu J, Falco J, Fortuno JR, Massuet A, Branera J. Percutaneous arterial embolization in the management of rectus sheath hematoma. *AJR Am J Roentgenol*. 2007;188(6): W497-502.

Authors

Umama Gorsi, MD, Memorial Hospital of Rhode Island, Warren Alpert Medical School of Brown University; Division of Cardiology, Mayo Clinic.

Vishnu Priya Mallipeddi, MD, Division of Cardiology, Mayo Clinic

Correspondence

Umama Gorsi, MD

Preventive Cardiology Fellow, Mayo Clinic

1805 Quarry Ridge

Rochester, MN 55901

umamasardar@hotmail.com

Rhode Island Lyme Disease Surveillance Summary 2014–2016

JONATHAN BARKLEY, MPH; DANIELA N. QUILLIAM, MPH; UTPALA BANDY, MD, MPH

INTRODUCTION

Cases of Lyme disease are concentrated in the Northeast and Midwest of the United States, following the distribution of the *Ixodes scapularis* ticks that transmit the disease. Early symptoms of Lyme disease include fever, chills, headache, fatigue, and muscle aches. The characteristic erythema migrans (EM) rash, is identified in 70–80% of cases and typically presents 3–30 days following a tick bite.[1] Later signs and symptoms appear days to months after tick bite and include arthritis, facial palsy, heart palpitations, and problems with short term memory. The peak transmission risk season in Rhode Island is from May to September.

In 2015, Rhode Island had the fourth highest rate of confirmed Lyme disease, behind Vermont, Maine, and Pennsylvania.[2] However, surveillance data used to determine these rates can depend on a state's ability to capture and classify cases, which is dependent on personnel and other resources. Since May 2013, funding to enhance Lyme surveillance has enabled RI to actively reach out to providers to obtain additional clinical information necessary to classify cases as "Confirmed" or "Probable" based on the national Centers for Disease Control and Prevention (CDC) case definition.[3] Note that the CDC case definition does not include post-treatment Lyme disease syndrome or chronic Lyme disease cases. Prior to May 2013, RI employed a passive system of Lyme surveillance, which has been previously described.[4] This article will summarize findings from 2014-2016, which represents three full years of data from RI's enhanced Lyme disease surveillance system. This article will discuss observed case counts and rates over time, geographic trends, clinical characteristics, and co-infections.

METHODOLOGY

The Rhode Island Department of Health's Center for Acute Infectious Disease Epidemiology (CAIDE) staff receives Lyme disease reports from healthcare providers, hospitals, and laboratories via mail, fax, or electronic laboratory reporting. When laboratory tests are positive, CAIDE staff follow up actively with providers to obtain clinical information. The lab information, combined with the clinical information, is used to classify cases as "Suspect", "Probable", or "Confirmed" based on the CDC Lyme disease case definition.[4] Importantly, an EM rash greater than or equal to 5cm is sufficient to classify a case as confirmed and no laboratory results are necessary. All clinical case information is entered into RI's web-based electronic disease surveillance system, reviewed for accuracy, and then transmitted to CDC.

Lyme data were exported from the surveillance system to calculate rates over time, describe clinical characteristics, and identify co-infections with other reportable tickborne diseases during the surveillance period. ArcGIS was used to show disease burden geographically by city/town.

RESULTS

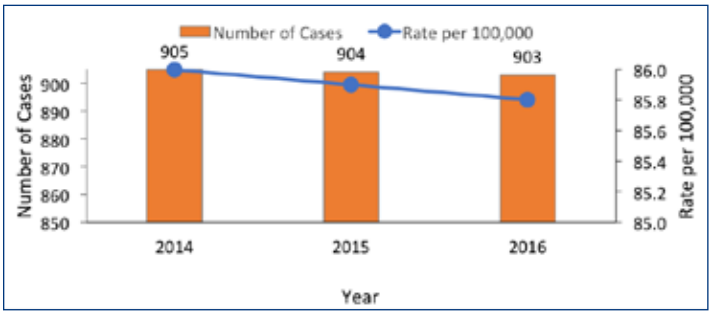
Case counts and rates over time

Reports of Lyme disease were observed to be stable from 2014 to 2016 (**Figure 1**). Approximately 900 confirmed and probable Lyme cases were identified each year and the incidence rate was in the range of 85–86 cases per 100,000 population.

Geographic distribution

From 2014 and 2016, rates of Lyme disease were consistently observed to be highest among Washington County residents and consistently lowest among Providence County residents. The three-year average incidence rate among Washington County residents was 197.0 cases per 100,000 population, compared to 59.6 per 100,000 among Providence County residents. Newport County had the second highest average incidence rate of 115.8 cases per 100,000 population, followed by Bristol County with an average

Figure 1. Number and rate of confirmed and probable cases of Lyme disease reported in Rhode Island, 2014-2016. Cases classified using CDC's Lyme disease case definition.



incidence rate of 105.6 per 100,000, and then Kent County at 78.6 per 100,000. A gradient of average three-year incidence rates is shown by city/town (**Figure 2**). Consistent with the observed county trends, a high incidence of Lyme is observed in the southwest part of the state, among cities and towns in Washington County. Although, the incidence of Lyme is lowest overall in Providence County, a high rate of Lyme disease is observed in the more rural cities and towns of the county, such as Foster and Scituate.

Clinical characteristics of confirmed cases

Erythema migrans greater than 5cm was reported among at least 45% of confirmed Lyme disease cases for each of the years (**Table 1**). Arthritis was the most commonly reported late-stage manifestation, ranging from approximately 35% in 2014 to approximately 48% in 2016. Other later-stage manifestations were less common, reported among fewer than 10% of confirmed cases. Note that probable cases are not included in this analysis as clinical features are not always available in the report to classify a case as probable.

Co-infections

Among the probable and confirmed cases of Lyme disease that were reported each year, between 2 and 3% were reported to be co-infected with at least one other tickborne disease (**Table 2**). For all years, babesia was the most commonly reported co-infection, followed by anaplasma.

DISCUSSION

This article summarizes Rhode Island's Lyme disease surveillance data from 2014–2016. While performing the enhanced surveillance methodology described, case counts and rates were high, but stable during this time period. The case counts and rates observed from 2014–2016 are higher than the case counts and rates observed during the three years prior (2011–2013), but these differences are likely attributable to enhanced surveillance and not increased illness. Underreporting still exists in the enhanced surveillance system; however, by following a consistent methodology CAIDE can better understand these data and identify relative increases and decreases that may be related to true changes in Lyme incidence.

Figure 2. Three-year average rates of Lyme disease by City/Town, Rhode Island, 2014–2016. Rates shown as a gradient and expressed per 100,000 population based on estimates obtained from RI Department of Labor and Training website: http://www.dlt.ri.gov/lmi/census/pop/townest.htm.

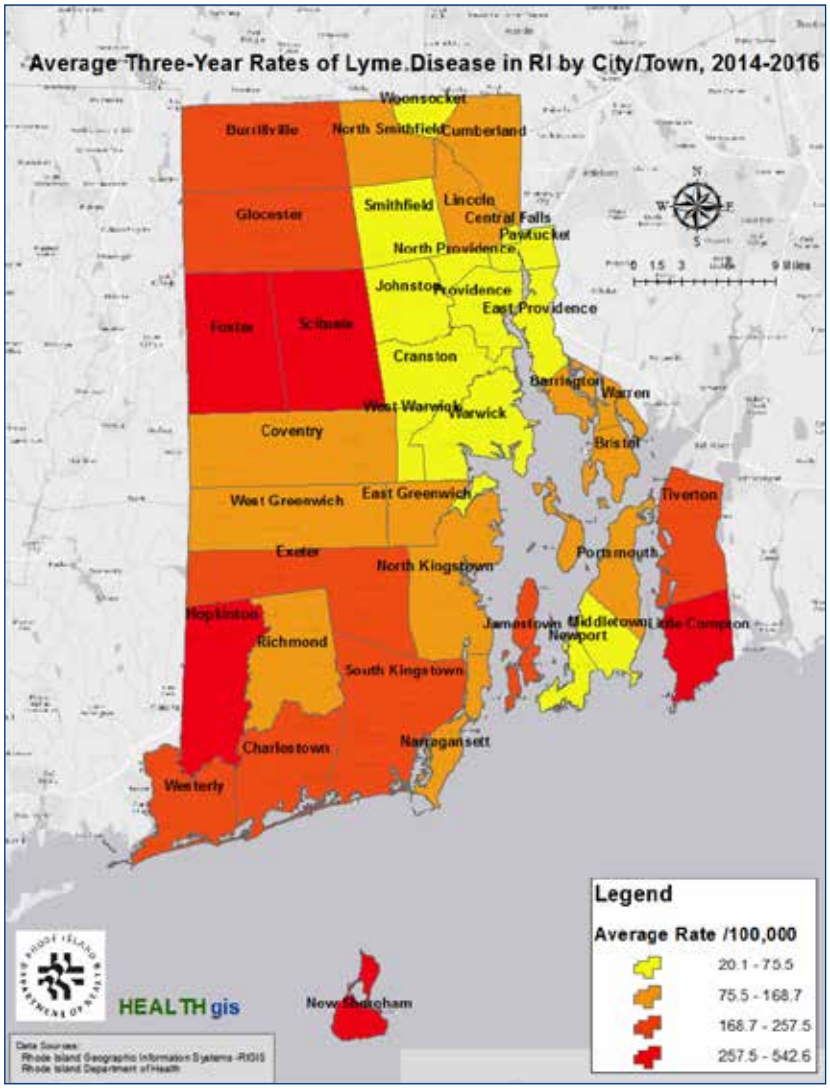


Table 1. Clinical characteristics of Lyme disease cases,[1] Rhode Island, 2014–2016.

	2014 (N=570)		2015 (N=564)		2016 (N=535)	
Clinical Characteristics	**N**	**% of confirmed cases**	**N**	**% of confirmed cases**	**N**	**% of confirmed cases**
Erythema migrans	313	54.9	289	51.2	242	45.2
Arthritis	198	34.7	212	37.6	255	47.7
Bell's Palsy	50	8.8	52	9.2	41	7.7
Radiculoneuropathy	19	3.3	21	3.7	16	3.0
Lymphocytic meningitis	10	1.8	11	2.0	5	0.9
Encephalitis	6	1.1	7	1.2	1	0.2

[1]Based on CDC Lyme disease case definition for confirmed cases.

Table 2. Reported co-infections[1] with Lyme disease in Rhode Island, 2014-2016.
Co-infections displayed as percentages of the total Lyme disease cases and of the total co-infections identified each year.

	2014 (N=905)			2015 (N=904)			2016 (N=903)		
Co-infections with Lyme	**N**	**% of total Lyme cases**	**% of co-infections**	**N**	**% of total Lyme cases**	**% of co-infections**	**N**	**% of total Lyme cases**	**% of co-infections**
Babesia	19	2.1	86.4	19	2.1	65.5	11	1.2	57.9
Anaplasma	2	0.2	9.1	9	1.0	31.0	6	0.7	31.6
Babesia and Anaplasma	0	0	0	1	0.1	3.4	0	0.0	0.0
Ehrlichia	1	0.1	4.5	0	0.0	0.0	1	0.1	5.3
Rocky Mountain Spotted Fever	0	0	0	0	0.0	0.0	1	0.1	5.3
TOTAL	**22**	**2.4**	**100.0**	**29**	**3.2**	**100.0**	**19**	**2.1**	**100.0**

[1]Co-infection identified if case met CDC's probable or confirmed case definition for Lyme and at least one other tickborne disease, with reported illness onset dates or specimen collection dates between conditions differing by 30 days or fewer.

Generally, the highest Lyme burden was observed in rural areas of the state, with rates tending to be the highest among cities/towns in Washington County. Adopting personal protection measures is critical in these areas and any area where high grass and levels of leaf litter can be found, as these conditions are favorable tick habitats. Individuals can protect themselves by wearing long pants and sleeves, using repellents containing DEET or products that contain permethrin on shoes and clothing, and bathing as soon as they come indoors. It is important to conduct frequent tick checks and wear light colored clothing so ticks can be spotted more easily. Anticipatory guidance and prevention counseling in physician offices as well as widespread public information education campaigns are key to informing the public about risk and prevention for this common disease.

Data from RI's enhanced surveillance allowed CAIDE to characterize clinical features and co-infections. An erythema migrans (EM) rash was reported in 45 to 55% of the confirmed cases, which is lower than the 70–80% reported by CDC.[1] This discrepancy could be due to underreporting by clinicians, incomplete clinical information being provided on the case reporting form, or providers not having the opportunity to diagnose an EM if the patient is seen after the rash has resolved or if the EM rash is marginally smaller than 5cm. A case is considered "Confirmed" if an EM rash greater than 5cm in size is observed, thus it is important for providers to report these cases to ensure they are included in the surveillance system.

Consistent with other literature, babesia was the most common co-infection that was identified among reported Lyme cases.[5] Among all reported Lyme cases from 2014–2016, 2–3% were reported to be co-infected with another tickborne disease, which may be accurate, but is also subject to limitations. In an attempt to distinguish concurrent from subsequent infections, a case was considered to be co-infected if the illness onset dates or specimen collection dates differed by 30 days or fewer between the two reported conditions. However, as noted in the literature, the case definition used can determine whether a case is counted as a co-infection.[6] Since patients are often tested for many tickborne diseases at the same time, it is possible that they meet the case definition for multiple tickborne diseases but may not be true co-infections. Literature has shown that cases co-infected with multiple tickborne diseases may have more severe symptoms and longer illness durations[5], so this is something that could be explored as part of active surveillance in the future. In conclusion, CAIDE's enhanced Lyme disease surveillance system has consistently been employed for three full years, which has allowed for analyses that documents a stable endemicity of disease in recent years and has described some basic clinical characteristics of cases. Continued enhanced surveillance is critical to identify the true and changing burden of Lyme disease in RI.

References

1. Centers for Disease Control and Prevention (CDC). Signs and Symptoms of Untreated Lyme Disease. https://www.cdc.gov/lyme/signs_symptoms/index.html. Accessed September 14, 2017.
2. Centers for Disease Control and Prevention (CDC). Lyme disease data tables. https://www.cdc.gov/lyme/stats/tables.html. Accessed September 14, 2017.
3. Centers for Disease Control and Prevention (CDC). Lyme Disease (Borrelia burgdorferi) 2017 Case Definition. https://wwwn.cdc.gov/nndss/conditions/lyme-disease/case-definition/2017/. Accessed September 14, 2017.
4. Lawrence M, Quilliam DN, Bandy U, Fulton JP, Marak TP, Berns A. Rhode Island tick-borne disease surveillance summary 2012-2013. *RI Med J*. 2014;97(9):46.
5. Belongia EA. Epidemiology and impact of coinfections acquired from Ixodes ticks. Vector-borne Zoonot. 2002;2(4):265-73.
6. Horowitz HW, Aguero-Rosenfeld ME, Holmgren D, McKenna D, Schwartz I, Cox ME, Wormser GP. Lyme disease and human granulocytic anaplasmosis coinfection: impact of case definition on coinfection rates and illness severity. *Clin Infect Dis*. 2012;56(1):93-9.

Acknowledgments

We thank Normand Laliberte, RN, and Jason Garrett, BSN, MPH, for their work supporting RI's Lyme surveillance system. Also, thanks to Caroline Gummo, MHS, for her assistance to the surveillance team as an intern.

This publication was supported by the Grant or Cooperative Agreement FOA CDC-FOA-CK14-1401PPHF, funded by the Centers for Disease Control and Prevention. Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the Centers for Disease Control and Prevention or the Department of Health and Human Services.

Authors

Jonathan Barkley, MPH, is Public Health Epidemiologist, Center for Acute Infectious Disease Epidemiology, Division of Preparedness, Response, Infectious Disease, and Emergency Medical Services, Rhode Island Department of Health.

Daniela N. Quilliam, MPH, is Chief, Center for Acute Infectious Disease Epidemiology, Division of Preparedness, Response, Infectious Disease, and Emergency Medical Services, Rhode Island Department of Health, and Teaching Associate of Epidemiology, Warren Alpert Medical School, Brown University.

Utpala Bandy, MD, MPH, is State Epidemiologist and Medical/Division Director, Division of Preparedness, Response, Infectious Disease, and Emergency Medical Services, and Clinical Assistant Professor, Department of Health Services, Policy, and Practice, Brown School of Public Health.

Rhode Island Monthly Vital Statistics Report
Provisional Occurrence Data from the Division of Vital Records

VITAL EVENTS	REPORTING PERIOD		
	MAY 2017	12 MONTHS ENDING WITH MAY 2017	
	Number	Number	Rates
Live Births	944	11,506	10.9*
Deaths	859	10,246	9.7*
Infant Deaths	6	70	6.1#
Neonatal Deaths	5	55	4.8#
Marriages	628	7,182	6.8*
Divorces	278	3,002	2.8*
Induced Terminations	172	2,099	182.4#
Spontaneous Fetal Deaths	74	656	57.0#
Under 20 weeks gestation	67	585	50.8#
20+ weeks gestation	7	71	6.2#

* Rates per 1,000 estimated population
\# Rates per 1,000 live births

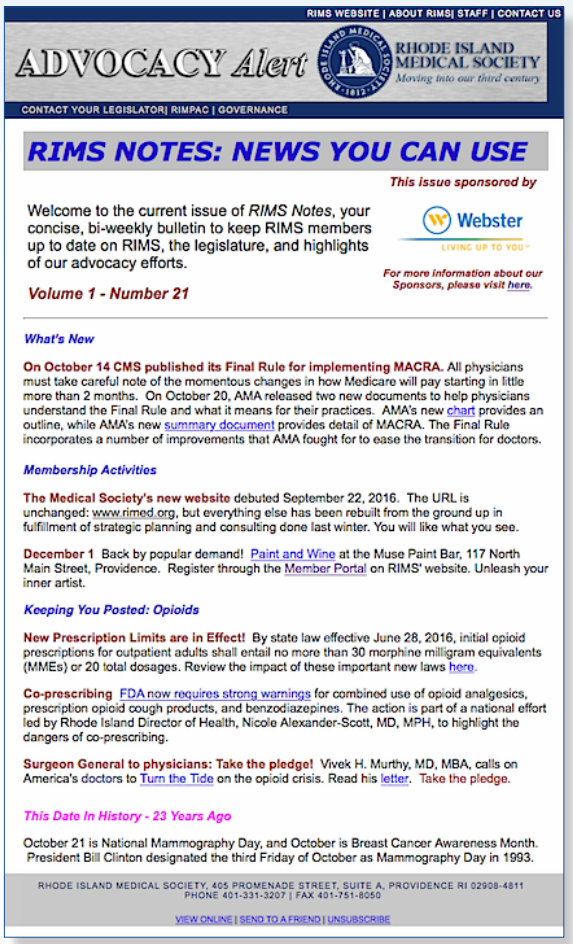
Underlying Cause of Death Category	REPORTING PERIOD			
	NOVEMBER 2016	12 MONTHS ENDING WITH NOVEMBER 2016		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	206	2,329	220.5	3,417.0
Malignant Neoplasms	154	2,169	205.3	5,474.5
Cerebrovascular Disease	37	429	40.6	492.5
Injuries (Accident/Suicide/Homicide)	79	884	83.7	13,736.5
COPD	41	459	43.5	497.5.

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.
(b) Rates per 100,000 estimated population of 1,056,298 (www.census.gov)
(c) Years of Potential Life Lost (YPLL).

NOTE: Totals represent vital events, which occurred in Rhode Island for the reporting periods listed above.
Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.

Are *you* e-reading

RIMS NOTES: *News You Can Use*



RIMS WEBSITE | ABOUT RIMS| STAFF | CONTACT US

ADVOCACY *Alert* — RHODE ISLAND MEDICAL SOCIETY *Moving into our third century*

CONTACT YOUR LEGISLATOR| RIMPAC | GOVERNANCE

RIMS NOTES: NEWS YOU CAN USE

This issue sponsored by Webster LIVING UP TO YOU™

Welcome to the current issue of *RIMS Notes*, your concise, bi-weekly bulletin to keep RIMS members up to date on RIMS, the legislature, and highlights of our advocacy efforts.

For more information about our Sponsors, please visit here.

Volume 1 - Number 21

What's New

On October 14 CMS published its Final Rule for implementing MACRA. All physicians must take careful note of the momentous changes in how Medicare will pay starting in little more than 2 months. On October 20, AMA released two new documents to help physicians understand the Final Rule and what it means for their practices. AMA's new chart provides an outline, while AMA's new summary document provides detail of MACRA. The Final Rule incorporates a number of improvements that AMA fought for to ease the transition for doctors.

Membership Activities

The Medical Society's new website debuted September 22, 2016. The URL is unchanged: www.rimed.org, but everything else has been rebuilt from the ground up in fulfillment of strategic planning and consulting done last winter. You will like what you see.

December 1 Back by popular demand! Paint and Wine at the Muse Paint Bar, 117 North Main Street, Providence. Register through the Member Portal on RIMS' website. Unleash your inner artist.

Keeping You Posted: Opioids

New Prescription Limits are in Effect! By state law effective June 28, 2016, initial opioid prescriptions for outpatient adults shall entail no more than 30 morphine milligram equivalents (MMEs) or 20 total dosages. Review the impact of these important new laws here.

Co-prescribing FDA now requires strong warnings for combined use of opioid analgesics, prescription opioid cough products, and benzodiazepines. The action is part of a national effort led by Rhode Island Director of Health, Nicole Alexander-Scott, MD, MPH, to highlight the dangers of co-prescribing.

Surgeon General to physicians: Take the pledge! Vivek H. Murthy, MD, MBA, calls on America's doctors to Turn the Tide on the opioid crisis. Read his letter. Take the pledge.

This Date In History - 23 Years Ago

October 21 is National Mammography Day, and October is Breast Cancer Awareness Month. President Bill Clinton designated the third Friday of October as Mammography Day in 1993.

RHODE ISLAND MEDICAL SOCIETY, 405 PROMENADE STREET, SUITE A, PROVIDENCE RI 02908-4811
PHONE 401-331-3207 | FAX 401-751-8050

VIEW ONLINE | SEND TO A FRIEND | UNSUBSCRIBE

The new biweekly e-newsletter exclusively for RIMS members.

Clear.

Concise.

Informative.

Respectful of your time.

RIMS NOTES
is published electronically on alternate Fridays.

Contact Sarah if you've missed an issue, sstevens@rimed.org.

Working for You:
RIMS advocacy activities

October 2, Monday
Kids Count release of "*Preventing Youth Tobacco Use in Rhode Island*"

October 3, Tuesday
RIMS Physician Health Committee: Herbert Rakatansky, MD, Chair

Former US Attorney Peter Neronha announcement for Attorney General

AMA Advocacy Resource Center conference call regarding medical licensing board issues

October 5, Thursday
Meeting with NHPRI regarding legislation

Retirement event for David Spenser, Substance Use and Mental Health Leadership Council (formerly DATA and the Rhode island Council of Community Mental Center Organizations)

October 6, Friday
OHIC Care Transformation and Alternative Payment Methodology Advisory Committees: Peter A. Hollmann, MD, President-elect

October 10, Tuesday
Conference Call, DOH and Anchor Medical Associates regarding RIMS' diabetes prevention initiative

October 11, Wednesday
Meeting with Board of Licensure and Discipline (BMLD)

Governor's Opioid Overdose and Prevention Task Force meeting: Sarah J. Fessler, MD, Immediate Past President

October 12, Thursday
Department of Health Community Review of proposed regulations regarding All-Payer Claims Data Base

SIM Steering Committee: Peter A. Hollmann, MD, President-elect

RI Academy of Physician Assistants event marking 50th anniversary of the physician assistant profession

October 13, Friday
Annual Meeting, Rhode Island Business Group on Health

Rhode Island affiliate American Civil Liberties Union annual meeting (allies with RIMS, RI-ACP, RISA, in opposing warrantless access to the Prescription Drug Monitoring Program)

RIMS' **Prevent Diabetes STAT–Rhode Island: Screen/Test/Act Today** CME event, held October 28 at the Providence Marriott Downtown, focused on the need for routine pre-diabetic screenings and referring patients to diabetes prevention programs.

October 16, Monday
Meeting with Blue Cross Blue Shield of Rhode Island: Bradley Collins, MD, President; Peter A. Hollmann, MD, President-elect, and staff

RI Health Center Association 45th anniversary event; RIMS Immediate Past President Sarah J. Fessler, MD, honored

October 17, Tuesday
OHIC Alternative Payment Methodology Advisory Committee: Peter A. Hollmann, MD, President-elect

Conference call American Academy of Dermatology Association regarding Modifier 25 policy of BCBSRI

October 18, Wednesday
Primary Care Physicians Advisory Committee (RI Department of Health)

October 19, Thursday
OHIC Care Transformation Advisory Committee: Peter A. Hollmann, MD, President-elect

Meeting with potential candidate RI Representative District 4

Conference call, American College of Rheumatology regarding state-level legislative and regulatory issues affecting rheumatology

Fundraiser, Representative Ranglin-Vassell

October 20, Friday
Meeting with Health Director Nicole Alexander-Scott, MD, MPH, on a spectrum of issues relating to public health

October 23, Monday
RI Health Professionals Loan Repayment Program

Conference Call, AMA Advocacy Resource Center Executive Committee (Steve DeToy, member)

October 24, Tuesday
Improving End of Life Care Coalition

October 25, Wednesday
Meeting with RIMS past president, Charles "Bud" Kahn, MD, regarding potential RIMS initiative

Special Legislative Commission to Study the Effects of Legalizing Marijuana: RIMS Member John Femino, MD

October 26, Thursday
Meeting with US House of Representatives' Energy and Commerce Committee, Democratic Staff, regarding gun violence prevention: Megan Ranney, MD, MPH; Washington, DC

October 26–27, Thursday and Friday
Meeting of state medical and specialty societies with Pharmaceutical Research and Manufactures Association (PhRMA)

October 28, Saturday
RIMS diabetes prevention seminar (2.5 CME credits), Marriott Hotel, Providence, 9–11:30 am (part of RIMS' diabetes prevention initiative)

RIMS CORPORATE AFFILIATES

The Rhode Island Medical Society continues to drive forward into the future with the implementation of various new programs. As such, RIMS is expanded its Affinity Program to allow for more of our colleagues in healthcare and related business to work with our membership. RIMS thanks these participants for their support of our membership.

Contact Marc Bialek for more information: 401-331-3207 or mbialek@rimed.org

www.nhpri.org

Neighborhood Health Plan of Rhode Island is a non-profit HMO founded in 1993 in partnership with Rhode Island's Community Health Centers. Serving over 185,000 members, Neighborhood has doubled in membership, revenue and staff since November 2013. In January 2014, Neighborhood extended its service, benefits and value through the HealthSource RI health insurance exchange, serving 49% the RI exchange market. Neighborhood has been rated by National Committee for Quality Assurance (NCQA) as one of the Top 10 Medicaid health plans in America, every year since ratings began twelve years ago.

www.ripcpc.com

RIPCPC is an independent practice association (IPA) of primary care physicians located throughout the state of Rhode Island. The IPA, originally formed in 1994, represent 150 physicians from Family Practice, Internal Medicine and Pediatrics. RIPCPC also has an affiliation with over 200 specialty-care member physicians. Our PCP's act as primary care providers for over 340,000 patients throughout the state of Rhode Island. The IPA was formed to provide a venue for the smaller independent practices to work together with the ultimate goal of improving quality of care for our patients.

RIMS gratefully acknowledges the practices who participate in our discounted Group Membership Program

ANCHOR MEDICAL ASSOCIATES

AQUIDNECK MEDICAL
Member of University Medicine

THE CENTER FOR orthopaedics INC.

COASTAL MEDICAL
LEADERS IN HEALTHCARE

Ortho Rhode Island

NorthMain Radiation Oncology

Ob-Gyn Associates
A Lifespan Physician Group Practice

Orthopaedic Associates, Inc.

Rhode Island Eye INSTITUTE

RI ENT

SKIN PROS

UEMF
university EMERGENCY MEDICINE foundation

UNIVOTO
University Otolaryngology

Women's Medicine Collaborative
A program of The Miriam Hospital
Lifespan. Delivering health with care®

For more information about group rates, please contact Marc Bialek, RIMS Director of Member Services

Congratulations

to the ***Rhode Island Medical Journal*** for
100 years of advancing healthcare in our state

At Blue Cross & Blue Shield of Rhode Island, we're proud to support the *Rhode Island Medical Journal* and work with the Rhode Island Medical Society in making healthcare accessible, affordable, and of the highest quality.

We are partnering with healthcare providers across the state to establish accountable care organizations that combine innovative payment models, patient data, and improved coordination of care to ensure our state continues to remain a healthcare leader for the next century.

Blue Cross Blue Shield of Rhode Island

bcbsri.com

The Good News is . . .

that Thousands More Rhode Islanders, Formerly Ineligible, May Now Enjoy the Protection of

Blue  *Cross*

As recommended by many Rhode Island physicians, Blue Cross now extends protection to Self-employed, unemployed, retired, or other persons employed in establishments having five or less employees. Opportunity for this new individual enrollment is *open to October 18th only.*

This new, widespread Blue Cross service, bringing the important protection of prepaid hospital service to the vast majority of Rhode Island citizens, will undoubtedly have your active support through recommendation to all eligible people within your sphere of professional and social contacts.

Farmers, fishermen, domestic employees, professional people, small business men—are among those who may now join. The age limit is 65 years and the usual Blue Cross health statement is required. The waiting period for maternity cases will remain at 9 months.

Prospective applicants may obtain full information and enrollment blanks by applying to Blue Cross headquarters. You will help this greater Blue Cross plan to complete success by requesting and using descriptive folders for your outgoing mail and a small display cut-out in color for your waiting room.

BLUE CROSS

338 HOSPITAL TRUST BUILDING

PROVIDENCE, RHODE ISLAND

GAspee 1451

Care New England Board takes action on the future of Memorial Hospital

Authorizes end of negotiations with Prime Healthcare Foundation

In action taken at a special meeting October 17, the Care New England (CNE) Board of Directors authorized the termination of negotiations with Prime Healthcare Foundation regarding their planned acquisition of Memorial Hospital of Rhode Island as a result of both sides being unable to reach mutually acceptable terms. The Board also authorized Care New England management to prepare necessary plans and filings with the Rhode Island Department of Health to maintain vital access to primary care and outpatient services in the community, while closing Memorial's inpatient units and Emergency Department.

The impetus for the changes includes the chronic financial losses being incurred at Memorial, continuing a nearly 10-year slide. The 294-bed hospital has averaged a daily inpatient census of just 15 to 20 patients resulting in an operating loss in the past fiscal year of $23 million. According to **CHARLES R. REPPUCCI**, chairman of the CNE board, "The magnitude of the losses at Memorial cannot be sustained and jeopardizes our other hospitals and provider organizations. We have exhausted our very best efforts and those of some nationally-recognized consultants to improve the situation without the outcomes we had hoped to achieve."

Care New England recorded a $68 million loss from operations in fiscal year 2016 and is projected to show a $49 million operating loss for the fiscal year that just ended on September 30. Its plan to restore financial well-being to the health care system focuses on continued work on revenue improvement and cost reduction, and resolution of the ongoing losses at Memorial.

Since the 2013 acquisition of Memorial by Care New England, **DENNIS D. KEEFE**, president and CEO, said the leadership of the system and the hospital have worked diligently to try to make Memorial successful. This includes significant investment in clinical information systems and facility improvements, bringing in new administrative leadership, establishment of new services, initiation of marketing plans to promote the hospital and its programs, and the hiring of restructuring experts to help turn around the hospital's dire financial situation.

Despite these efforts and a 2016 improvement plan to relocate the obstetric unit and scale back inpatient capacity, Memorial has not drawn enough patient and community support to meet meaningful volume thresholds that would sustain a safe and viable inpatient operation.

Accordingly, in early 2017, Care New England initiated an exhaustive search to engage more than 70 potential parties that might be interested in the acquisition of Memorial. Prime ultimately emerged as the single bidder, and the execution of the Letter of Intent (LOI) between Prime and Care New England was announced in April 2017. In the time since, extensive work on due diligence and the negotiation of terms has taken place. However, the parties were unable to reach an ultimate agreement. Confidentiality provisions in the LOI prevent both organizations from sharing further details.

Care New England's **JAMES E. FANALE, MD, EVP**, chief operating officer and chief clinical officer, said it will be the utmost priority in the plan for Memorial to continue to provide high quality patient care, while working to address options to ensure access to care for patients in the community including maintaining a robust primary care presence. CNE will also develop a plan to address the Memorial-based medical residency training program with The Warren Alpert Medical School of Brown University.

"While difficult, these actions represent a necessary and critical step in restoring financial health to Care New England while ensuring the future of hospitals and facilities many have come to rely on for their care," continued Reppucci. "This has been a long and complex process that has been met with unrelenting dedication and compassionate care from all who work at Memorial Hospital. We will support both the employees and the community in this transition so the well-established legacy of care in Pawtucket is not lost, but rather adapted for the demands of today's health care landscape."

Care New England will begin immediately working with the Memorial staff on transition plans. Until a definitive plan is developed and approved, there are no estimates on the number of employees who might be displaced. Care New England will attempt to place affected employees in other vacancies across Butler, Kent, Women & Infants, the VNA of Care New England, and The Providence Center.

Care New England officials say they hope to file the necessary regulatory application (Reverse Certificate of Need) as soon as possible.

Care New England remains committed to its affiliation with Partners HealthCare, and looks forward to ongoing progress in the negotiations and due diligence process. ❖

Brookdale Overview

Independent Living *An ideal retirement living experience*

- Spacious apartments with minimal maintenance
- Restaurant-style dining
- Plenty of planned activities every day

Assisted Living *The right choice for people who need extra help with daily activities*

- Qualified staff assists with taking medication, dressing, bathing, etc.
- Floor plans, from studio to two-bedroom apartments
- Activities and events for various levels of acuity

Alzheimer's & Dementia Care *Person-centered care for people at various stages*

- Programs that leverage the latest dementia care research
- A care philosophy defined by more than the symptoms of Alzheimer's & dementia
- An experienced staff who help residents thrive

Rehabilitation & Skilled Nursing *For short-term surgerical recovery or long-term rehabilitation*

- Around-the-clock, licensed nursing care
- Providing clinical resources in a comfortable setting that feels like home
- A mission and focus to helping residents get well and then get home as quickly as possible

---

Personalized Living *For people who just need a little help with things*

- One-on-one non-medical services for home care needs
- Additional personal needs for those in assisted living or home such as escorts to doctor appointments and more

Home Health *For qualified people in need of therapy or rehabilitation — all in the comfort of home*

- Get Medicare-certified assistance from experienced professionals
- Many healthcare services such as wound care and stroke therapy

Therapy *Specialized programming personalized to encourage recovery*

- An emphasis on education, fitness and rehabilitation that helps seniors retain or enhance their independence
- Most insurances accepted

Hospice *Promoting comfort by addressing the full range of needs of patients and families*

- Primary focus of quality of life
- Specially trained staff help families and patients cope with overwhelming feelings accompanying end-of-life care

Not all services are available at all communities. Contact community for details

The Rhode Island Network

Brookdale Center of New England
Brookdale Cumberland
Brookdale Smithfield
Brookdale Greenwich Bay
Brookdale Pocasset Bay
Brookdale Sakonnet Bay
Brookdale East Bay
Brookdale West Bay
Brookdale South Bay

For more information about how we can help you serve your patients' needs

Click Here

77025 CB

Bringing **New Life** to Senior Living®

BROOKDALE
—SENIOR LIVING SOLUTIONS—

brookdale.com

Miriam receives NIH grant to study RI Department of Corrections opioid program

$215,157 grant to be led by principal investigator Josiah "Jody" Rich, MD

The Miriam Hospital has been awarded a National Institutes of Health grant to study an innovative opioid addiction treatment program for incarcerated individuals that was expanded at the Rhode Island Department of Corrections last year.

The $215,157 federal grant will fund research into medication-assisted treatment to be led by principal investigator **JOSIAH "JODY" RICH, MD**, an infectious disease specialist and director of the hospital's Center for Prisoner Health and Human Rights.

The program at the Rhode Island Department of Corrections treats individuals diagnosed with opioid use disorder by initiating and continuing them on synthetic narcotics – methadone and buprenorphine (Suboxone). The program also provides access to naltrexone (Vivitrol), which deters opioid abuse by blocking any high from narcotics. As they re-enter into the community, participants are linked up with providers of medication-assisted treatment to further decrease risks of relapse, overdose, and re-incarceration.

The program, the first of its kind in a statewide correctional system, has garnered national attention.

The grant from the National Institute on Drug Abuse is for "evaluating the implementation and impact of a novel medication-assisted treatment program in a unified jail and prison system."

"People with opioid use disorder who leave the correctional setting without medications are among those at the highest risk for overdose and death," said Dr. Rich, who in addition to his work at The Miriam is a professor of medicine and epidemiology at Brown University's Warren Alpert Medical School. "The comprehensive program developed at the Rhode Island Department of Corrections, in partnership with CODAC and others, is having and will continue to have a substantial impact on reducing overdose deaths in Rhode Island. This grant will allow this program to be optimized and replicated across the nation."

Dr. Rich serves as an expert advisor to Gov. Gina Raimondo's Overdose Prevention and Intervention Task Force. In response to a request from the governor, the General Assembly provided $2 million in the state's 2017 budget to expand the treatment program in the prisons.

ASHBEL T. WALL, II, director of the RI Department of Corrections (RIDOC), said, "With the support of the Governor and the General Assembly, we have been able to roll out treatment for opioid addiction to currently incarcerated individuals and link those individuals to ongoing treatment in the community. Our approach has been so successful that the Bureau of Justice Assistance has designated RIDOC as a 'Center of Innovation' and we have been receiving calls from colleagues around the country who are interested in our approach."

The corrections program is run by CODAC Behavioral Healthcare, Rhode Island's oldest and largest community provider of services for opioid use disorder.

Dr. Rich's research team is an interdisciplinary collaboration between Lifespan, Brown University's Center for Alcohol and Addiction Studies, the University of Rhode Island's Academic Health Collaborative, and the University of North Carolina at Chapel Hill. **LAUREN BRINKLEY-RUBINSTEIN, PhD**, is the lead co-investigator for the study and will oversee the qualitative component of this research. Other members of the research team are **TRACI GREEN, PhD**, **BRANDON MARSHALL, PhD**, **LYN STEIN, PhD**, and **ROSEMARIE MARTIN, PhD**. ❖

Lifespan's Comprehensive Spine Center expands to Newport

The Comprehensive Spine Center anchored at Rhode Island Hospital is now offering its full range of services at Newport Hospital. The Center opened there on October 10. Patients will be seen by interventional pain specialist and physiatrist **KYLE SILVA, DO**, neurosurgeon **JARED FRIDLEY, MD**, and **ALEXIOS G. CARAYANNOPOULOS, DO, MPH**, medical director of the Lifespan Comprehensive Spine Center and division director of Pain and Rehabilitation Medicine in the Department of Neurosurgery at Rhode Island Hospital. Dr. Carayannopoulos is also an interventional pain specialist and physiatrist. Services at Newport Hospital will be integrated with the Vanderbilt Rehabilitation Center. ❖

Brown launches global public health MA program

PROVIDENCE – Beginning with the fall 2018 semester, Brown University will offer a global public health master's degree that combines traditional public health training in population sciences with rigorous social science and international fieldwork experience. The Corporation of Brown University approved the new degree program at its annual fall meeting on October 21.

Graduate program director Abigail Harrison said the University expects to enroll a cohort of 10 domestic and international students in the first year and more in future years.

Only about five other universities offer a master's in global health, Harrison said – and only about half a dozen more provide a global track within a traditional master's in public health. Among those, not all require fieldwork. Students in the new Brown program, however, will work in one of several low- or middle-income countries such as Ghana, Samoa, South Africa, the Philippines or Mexico, during the summer between the program's two years to "learn public health by doing public health." The projects they perform will form the basis of their master's theses.

Classroom-based curricular highlights include new courses on the ethics of community engagement, global organizations and policy priorities, and implementation science and public health interventions, Harrison said.

The program will begin accepting applications late in fall 2017. ❖

Proud to sponsor the Rhode Island Medical Society

More than $1 out of every $4 of practice revenue is the patient's responsibility!

24/7 Online

Client Management System

Patient portion has been growing by five to six percent per year. Getting control of patient portion an important focus of any private practice that expects to say private.

On average practices send 3.3 statements before receiving payment

*The average cost of printing, handling and stamping a statement is $1.25 per statement, not including employee cost.

COLLECTIONS WITHOUT ALIENATING YOUR PATIENTS

DMI
DEBT MANAGEMENT, INC.

Local
High Recovery
Customized Programs
Improved Patient Retention

For a free consultation call Carmella Beroth at 508-553-1916 or visit www.debtmanagementinc.com

Debt Management, Inc.
"Collecting the Uncollectible"

Hep C care falls short for young RI opioid users

PROVIDENCE – As public health officials worry that the increase of opioid use among young adults has helped to spread the hepatitis C virus to a new generation, a study in Rhode Island finds that while screening is common, the follow-up measures needed to stop the spread of the virus are significantly less so.

"Many young people who are at risk for hepatitis C may acquire the infection and then not know it, and then through drug injection practices may transmit it to others," said **BRANDON MARSHALL**, associate professor of epidemiology in the Brown University School of Public Health and corresponding author of the new study in the Journal of Adolescent Health. "For this reason, we need to not only be screening, but also providing care to young people who test positive for hepatitis C."

Between January 2015 and February 2016, the researchers recruited 196 people between the ages of 18 and 29 from the streets of Rhode Island who use prescription opioids recreationally, rather than for medical reasons. Of those, 154 (78.6 percent) reported receiving HCV screening, which Marshall said was a high and encouraging rate. That said, the proportion receiving screening was much higher among those ages 24 to 29 (89.5 percent) than among those ages 18 to 23 (59.7 percent), he noted.

Among those who were screened, 18 said they tested positive for HCV, which was 30 percent of the 59 people in the study who said they have injected drugs. When study staff asked about follow-up care, they found several gaps: Among the 18 with a positive test, 13 received a confirmatory follow-up test, 12 were referred for specialty care, only 10 received information about how not to transmit the virus to others, and nine received education about living with HCV.

"Screening for HCV is free in many parts of the state, but financial and other barriers exist for youth who test positive and are in need of additional resources and hepatitis C care," Marshall said. "We need to work on improving access to hepatitis C treatment programs and other referral services for young people."

Co-author **DR. LYNN TAYLOR**, an associate professor of medicine at Brown and physician at the Miriam Hospital, said the clear overlap of opioid use and hepatitis C infection requires a tightly coupled public health effort.

"This work points to our next steps: We must act to integrate overdose and hepatitis C prevention in Rhode Island," Taylor said. "In locales where people are injecting opiates, there are an estimated five new hepatitis C infections for every fatal overdose. Rhode Island is the ideal state to address the connections between the opioid and hepatitis C crises and demonstrate the benefits that are possible for public health preventive efforts."

Brown University School of Public Health graduate alumnus **AYORINDE SOIPE** led the study. Other authors include **AJIBOLA ABIOYE**, **TRACI GREEN** and **DR. SCOTT HADLAND**.

The National Institutes of Health funded the Rhode Island Young Adults Prescription Drug Study (grant R03-DA03770), from which the data were derived, and provided additional funding (P30AI042853). ❖

Kent Union employees and Kent Hospital reach agreement

WARWICK – The United Nurses and Allied Professionals and Kent Hospital reached a tentative agreement October 22, on a first contract for 400 newly organized employees and a contract extension for the 800 employees currently under contract. Members voted by an overwhelming margin to approve the two agreements Monday night. Both contracts will run through at least June 30, 2020.

The difficult and complex negotiations dealt with two fundamental issues. The first was bringing the newly organized employees pay and benefits up to the level of already-organized employees. The second issue was a contract extension, providing employees with security and stability while Care New England seeks to establish an organizational partner for the future. The first contract for the 400 newly organized employees largely places all members in the same pay benefit structure. The contract extension includes a one-year wage freeze in 2018 with wage increases in 2019 and 2020.

Union president **ROSE DESNOYERS, RN**, said, "This agreement achieves two major goals for our members. First, we are very pleased that our 400 new members now have the benefit of a union contract. Second, the contract extensions provide stability and certainty for all of our members over the next several years."

MICHAEL DACEY, MD, Kent Hospital president and COO, said, "I am extremely pleased with the agreement reached and the tremendous support indicated by Monday's vote. I believe this new contract is the result of both sides coming together and achieving positive outcomes that put the future of the hospital first, while addressing the important concerns of the union membership." ❖

Multi-site study to examine cognitive behavior therapy for traumatic brain injury-induced seizures

PROVIDENCE – Seizures are a common result of traumatic brain injury, especially in Veterans. A new study funded by the Department of Defense Congressionally Directed Medical Research Programs, and conducted in Providence and Birmingham, Ala. – at the Veterans Affairs Medical Centers in Providence and Birmingham, Rhode Island Hospital, Brown University and the University of Alabama at Birmingham – hopes to shed new light on the mechanism behind seizures associated with post-traumatic epilepsy and psychogenic nonepileptic seizures.

The $3.6 million award, W81XWH-17-1-0619, will examine whether a form of cognitive behavior therapy – a short-term, goal-oriented psychotherapy approach to problem-solving – could be effective in reducing the frequency and/or severity of seizures in those with traumatic brain injury, or TBI. Cognitive behavior therapy has been widely used for improving mental health. It focuses on developing coping strategies to treat specific problems and decrease symptoms.

PROVIDENCE VA MEDICAL CENTER PHOTO BY WINFIELD DANIELSON

Dr. W. Curt LaFrance Jr., co-principal investigator for a new study that hopes to shed light on the mechanism behind seizures associated with post-traumatic epilepsy and psychogenic nonepileptic seizures, conducts a finger-tapping exercise with Army and Navy Veteran Ernest J. Avery of West Warwick, as part of an exam at the Providence VA Medical Center February 2, 2016.

"Individuals can develop pathological responses, including seizures, from major, life-changing events such as traumatic brain injury," said **Jerzy Szaflarski, MD, PhD**, director of the Epilepsy Center in the UAB School of Medicine and co-principal investigator of the study. "The overall goal of the study is to see if cognitive behavior therapy will modify brain changes and response to stressful events, and whether these changes will result in improved seizure control."

"Non-pharmacologic approaches for seizures are gaining acceptance as a therapy," said **W. CURT LAFRANCE JR., MD**, co-principal investigator and member of the VA RR&D Center for Neurorestoration and Neurotechnology, associate professor of Psychiatry and Human Behavior and Neurology at Brown University's Warren Alpert Medical School, director of Neuropsychiatry and Behavioral Neurology at Rhode Island Hospital, and neuropsychiatrist at the Providence VA Medical Center. "Building off of our previous pilot studies, this will be the first large-scale examination of the neuroimaging brain signals in response to an intervention for patients with seizures."

The study teams will enroll Veteran and civilian patients with a history of TBI, divided into three groups of 88 patients each. One group will consist of patients with TBI without a history of seizures, another group will have TBI with epileptic seizures, and the last will have TBI with nonepileptic seizures.

Patients with seizures will receive cognitive behavior informed therapy for 12 weeks, administered by trained medical professionals. All patients will receive functional magnetic resonance imaging, or MRI, at baseline and again at approximately 14 weeks. An earlier study conducted by LaFrance and Szaflarski in 36 patients showed that cognitive behavior therapy improved seizure control in patients with nonepileptic seizures.

Epileptic seizures can be treated medically and with surgery, but there is not a standard therapy for nonepileptic seizures, which also occur in Veterans. Between 10–20 percent of the general population with seizure disorders experience nonepileptic seizures.

"The anticipated long term scientific gains will contribute to the goal of validating a neurological biomarker for patients with seizures that may be used for identifying treatment response," LaFrance said. "The effort could ultimately affect individuals and caregivers by providing a diagnostic tool that may aid in identifying treatment targets and response in reducing seizures and common comorbidities in Veterans and civilians."

"This project, combining functional neuroimaging with patient interventions, should provide a deeper understanding of neuroanatomic and neurophysiologic processes in patients with seizures," Szaflarski said. "The information gained will generate further hypotheses on neural processes and biomarkers for both epileptic and nonepileptic seizures."

The research is being funded through an Idea Development Award by the Department of Defense Congressionally Directed Medical Research Programs' Epilepsy Research Program. The ERP was initiated in 2015 to develop an understanding of the magnitude of post-traumatic epilepsy within the military and to expand research into the basic mechanisms by which TBI produces epilepsy. ❖

Pictured here at the groundbreaking (from left to right) are Cheryl Dunnington, RN, Chief Nursing Officer; Dr. Vincent Armenio, Chairman of Medicine; Demetra Ouellette, President, Roger Williams; Dr. Sheri Smith, Chair of the Roger Williams' board; John Holiver, Chief Executive Officer of CharterCARE; Edwin Santos, Chair of the CharterCARE board; Dr. N. Joseph Espat, Chairman of Surgery, and Candace Wray, RN, clinical nurse manager of the Roger Williams' Emergency Department.

Groundbreaking held at RWMC for major Emergency Department renovation

PROVIDENCE – On October 3, 2017, Roger Williams Medical Center held a groundbreaking ceremony to renovate the existing Roger Williams' Emergency Department space by building an addition of approximately 12,000 SF to accommodate increased patient bay sizes and an expanded behavioral health program.

The new addition will provide 28 private patient treatment bays including six behavioral health beds, a bariatric treatment bay, two procedure rooms and a trauma room alongside typical state-of-the-art treatment bays and exam space.

Substantial renovations will occur in the existing portion of the Emergency Department to further provide for a separate behavioral health tract. The existing Emergency Department will remain operational during construction. Upon completion of the project, there will be improved patient parking and a covered patient drop-off along with increased support space and modernized fixtures, furniture and equipment.

"Throughout our health system, we continue to make investments to improve the care we deliver to the community," said **JOHN HOLIVER**, CharterCARE CEO. "We pride ourselves on speedy and appropriate emergency care, which will be enhanced greatly by this renovation at Roger Williams."

Holiver also noted the significant economic boost this 18-month project will bring to the construction, trade and affiliated industries. The project was designed by Robinson Green and Beretta (RGB) Architects and is being constructed by Gilbane. ❖

Recognition

Southcoast Health named one of America's 100 Best Hospitals for Cardiac Care by Healthgrades

Southcoast Health has been recognized as one of America's 100 Best Hospitals for Cardiac Care for the seventh year in a row by Healthgrades (2012–2018), the leading online resource for healthcare consumers. Southcoast Health is one of just four hospitals in Massachusetts to receive this distinction for seven consecutive years.

In all, Southcoast Health was recognized for superior cardiovascular services with 11 recognitions, including receiving the Healthgrades Cardiac Care Excellence Award for the 12th year in a row (2007–2018).

"These recognitions are a much-deserved acknowledgment of the tremendous dedication of the team of physicians, nurses and clinical staff at the Southcoast Health Cardiovascular Care Center," said **DR. MARGARET FERRELL**, Physician-in-Chief of Cardiovascular Services at Southcoast Health.

Southcoast Health also received awards in pulmonary, gastrointestinal and critical care.

These achievements are part of new findings and data released on Healthgrades.com and in the Healthgrades 2018 Report to the Nation. For its analysis, Healthgrades evaluated approximately 45 million Medicare-patient records for nearly 4,500 short-term acute care hospitals nationwide, assessing hospital performance relative to each of 34 common conditions and procedures.

Healthgrades recognizes a hospital's quality achievements for cohort-specific performance, specialty area performance and overall clinical quality. Individual procedure or condition cohorts are designated as 5-star (statistically better than expected), 3-star (statistically as expected) and 1-star (statistically worse than expected) categories.

Detailed performance information, such as cohort-specific outcomes data and quality achievements, as well as more information on the Healthgrades 2018 Report to the Nation, including the complete methodology, can be found at www.healthgrades.com/quality. ❖

Michael Fine, MD, honored with Primary Care Leadership Award

Patient-Centered Primary Care Collaborative (PCPCC) honors Michael Fine, MD, with the prestigious Barbara Starfield Primary Care Leadership Award. The award was presented during the Annual Conference at the PCPCC's Annual Awards Dinner on October 11, 2017.

Dr. Fine holds the titles of Health Policy Advisor to Mayor James Diossa of Central Falls, Rhode Island, as well as Senior Population Health and Clinical Services Officer at Blackstone Valley Community Health Care (BVCHC). In addition to these roles, Dr. Fine is the driving force behind the Central Falls Neighborhood Health Station, which is slated to open in 2018, and will provide all health services, including primary care and population health, for most of the municipality's residents. Fine is the co-author of, "The Nature of Health," and a new book, "Healthcare Revolt," slated to be released in 2018.

"We are elated to recognize Dr. Fine," said PCPCC's President and CEO, Ann Greiner. "As a family physician, community organizer, and advocate, Dr. Fine has devoted his career to reforming primary care to better reduce costs, improve access, and help the underserved community. He has also been a policy leader, successfully driving primary care and public health collaboration."

"There is no greater honor than to receive the Barbara Starfield Award," said Dr. Fine. "Dr. Starfield's lifetime of research and advocacy helped me understand the important relationship between health equity and democracy as well as the reason that primary care must be at the center of a health care system." ❖

Ann Meers, RN, from Women & Infants receives award from American Urogynecologic Society

PROVIDENCE – **ANN MEERS, BS, RN, CCRC**, of Barrington, research supervisor in the Division of Urogynecology and Reconstructive Pelvic Surgery at Women & Infants Hospital, was recently presented with the Robin Haff Research Award by the American Urogynecologic Society (AUGS).

According to AUGS, this award is presented annually to an individual in recognition of outstanding and sustained contributions to female pelvic floor disorders research. The Robin Haff Research Award recognizes the important role that clinical research nurses and coordinators play in the clinical research process.

Meers has worked in women's health research for two decades, with a particular focus on pelvic floor disorders. She has served as chair of the Research Coordinator Committee of the *Eunice Kennedy Shriver* National Institutes of Child Health and Development Pelvic Floor Disorders Network. ❖

Appointments

Dr. Vanessa M. Britto named Executive Director of Health and Wellness at Brown

VANESSA M. BRITTO, MD, currently the director of health services for Wellesley College, has been appointed assistant vice president and executive director of health and wellness at Brown University.

Reporting to the vice president for campus life and student services, Dr. Britto will provide strategic, administrative and clinical leadership for medical and mental health services for students at Brown. She will oversee a wide range of health and wellness departments – including Health Services and Counseling and Psychological Services – to provide high-quality, comprehensive and collaborative health care on campus.

Dr. Britto has served since 2001 in her current role at Wellesley, and previously was the college physician at Stonehill College. Prior to her career in higher education, Dr. Britto practiced medicine both in community health centers and in private practice.

Having completed her internal medicine residency and a fellowship in general internal medicine at Rhode Island Hospital and with a master of science in community health from Brown, Dr. Britto also brings to the position firsthand experience on the Brown campus. She says the position is a natural extension of her commitment to student health and well-being and the foundational role health plays in the ability of students to achieve their educational goals.

As part of her work at Brown leading Health Services and Counseling and Psychological Services, Dr. Britto will oversee services including BWell Health Promotion, Emergency Medical Services, nursing, pharmacy and more. She will lead a staff of approximately 106 employees and 125 student workers, who provide clinical services for more than 34,000 patient encounters annually.

In addition, Dr. Britto will develop programs for students and residents at the Warren Alpert Medical School and will hold an appointment on the medical school faculty. Her appointment follows a search conducted by a committee that included faculty and staff from Brown along with undergraduate, graduate and medical school students.

In addition to her master's degree from Brown, she holds a bachelor of arts from Dartmouth College and an MD from the University of Illinois College of Medicine.

Dr. Britto will begin her work at Brown on January 16, 2018. ❖

Appointments

Dr. Alexander-Scott named President-Elect of national organization of state health directors

State health directors from across the nation have elected **NICOLE ALEXANDER-SCOTT, MD, MPH**, Director of the Rhode Island Department of Health (RIDOH), to be President-Elect of the Association of State and Territorial Health Officials (ASTHO), providing Dr. Alexander-Scott with a national platform to advocate for the health priorities of Rhode Islanders and people throughout the country.

ASTHO is comprised of the chief health official in each U.S. state and territory. Dr. Alexander-Scott will spend a year working with the current President to help steer the organization before assuming the role of President in September, 2018. Dr. John Wiesman, Secretary of Health at the Washington State Department of Health, is beginning his term as ASTHO's President as Dr. Alexander-Scott begins her term as President-Elect.

"I was both humbled and honored to be elected President-Elect of ASTHO. State health directors from across the country made this decision after looking at the tremendous successes brought about in Rhode Island by the 480 individuals that I am honored to call colleagues at RIDOH," said Dr. Alexander-Scott. "Our focus at RIDOH is ensuring that every person and community in Rhode Island has an equal opportunity to be as healthy as possible, regardless of zip code, race, ethnicity, sexual orientation, gender identity, level of education, level of income, or insurance status. As President-Elect of ASTHO, I will be partnering with the top public health officials in the nation whose expertise will help us make this vision a reality."

"We are honored to have Nicole Alexander-Scott, MD, MPH serve as ASTHO president-elect," said ASTHO Executive Director Michael Fraser. "She brings unique experience to this role as a practicing physician who specializes in adult and pediatric infectious diseases. We applaud her commitment to public health and appreciate her ongoing leadership to ensure optimal health for all."

ASTHO develops and guides public health policy and ensures excellence in state-based public health practice. Through its leadership team, ASTHO coordinates and advises state health directors on the impacts of health policy and provides them with guidance and technical assistance on improving the nation's health. In addition to the numerous public health policy areas that ASTHO contributes to, each President selects an ASTHO President's Challenge for her or his term. Secretary Wiesman is continuing the 2017 focus (Public Health Approaches to Preventing Substance Misuse and Addictions).

Dr. Alexander-Scott has been the Director of Health at RIDOH since April 2015. She is board-certified in Pediatrics, Internal Medicine, Pediatric Infectious Diseases, and Adult Infectious Diseases. ❖

Appointments

Drs. B. Mona Wirk, Scott LeTellier join Roger Williams Cancer Center

PROVIDENCE – **DR. B. MONA WIRK** has joined the Blood and Marrow Transplantation Unit at Roger Williams, the only unit of its kind in Rhode Island. She completed a postdoctoral fellowship in Hematopoietic Stem Cell Transplantation at the Fred Hutchinson Cancer Research Center/Seattle Cancer Care Alliance in Seattle. Dr. Wirk also completed a one-year Blood and Marrow Transplant Fellowship in the BMT program at Fox Chase Cancer Center at Temple University Hospital in Philadelphia. Her professional experience includes serving as an assistant professor in the Blood and Marrow Transplant Programs of both Stony Brook University in New York and University of Florida. She also worked in the Department of Hematology Oncology at the Mayo Clinic in Jacksonville, Florida.

DR. SCOTT LETELLIER has joined the Medical Oncology division at Roger Williams. Dr. LeTellier completed a fellowship in Hematology Oncology from University of Mississippi Medical Center. He attended medical school at the Autonomous University of Guadalajara in Mexico where he became proficient in Spanish. He completed another year of medical school through New York Medical College and later went on to complete his residency at Staten Island University Hospital. ❖

Anne Schmidt, RN, to serve on national nursing board

VP of patient care services, chief nursing officer at South County Health

ANNE SCHMIDT, RN, vice president of patient care services and chief nursing officer at South County Health in Wakefield, RI was elected to the board of directors for the American Organization of Nurse Executives (AONE). She will serve a three-year term on the AONE Board of Directors, beginning Jan. 1, 2018.

She served in the United States Navy Nurse Corps, received the State of Connecticut Nightingale Award for Nursing Excellence and Leadership, and was named San Diego Naval Hospital's Navy Nurse of the Year. She holds a Master of Science in Nursing degree from the University of San Diego and certification as an adult nurse practitioner and certified executive of nursing practice.

The American Organization of Nurse Executives (AONE) is the national professional organization for nurses who design, facilitate and manage care. With more than 9,700 members, AONE is the leading voice of nursing leadership in health care. The organization provides leadership, professional development, advocacy and research to advance nursing practice and patient care, promote nursing leadership excellence and shape public policy for health care. AONE is a subsidiary of the American Hospital Association. ❖

Obituary

DR. LOUIS M. DAMIANI, 71, of Naples, FL, passed away peacefully at his home after a long illness. He was the beloved husband of Elaine for 44 years.

After graduating from E. Providence High School and URI, he chose to serve in the U.S. Army and later the Army Reserve.

Dr. Damiani graduated from medical school in Guadalajara, Mexico and completed his studies at the State University of New York, Buffalo. He returned to Rhode Island to do his internship and residency in internal medicine at Rhode Island Hospital.

Dr. Damiani practiced medicine in East Providence for over 25 years. He was a past member of the Rhode Island Medical Society, the American College of Physicians and the Aurora Civic Association.

He leaves 2 brothers, Michael J. (Sharlene) of East Providence, Steven R. (Linda) of Barrington and a sister-in-law Dianne Zoglio of Cranston. He also leaves several nieces and nephews. He will be greatly missed and fondly remembered as a loving husband and a witty, intelligent gentleman who loved medicine and had boundless compassion for his patients. ❖