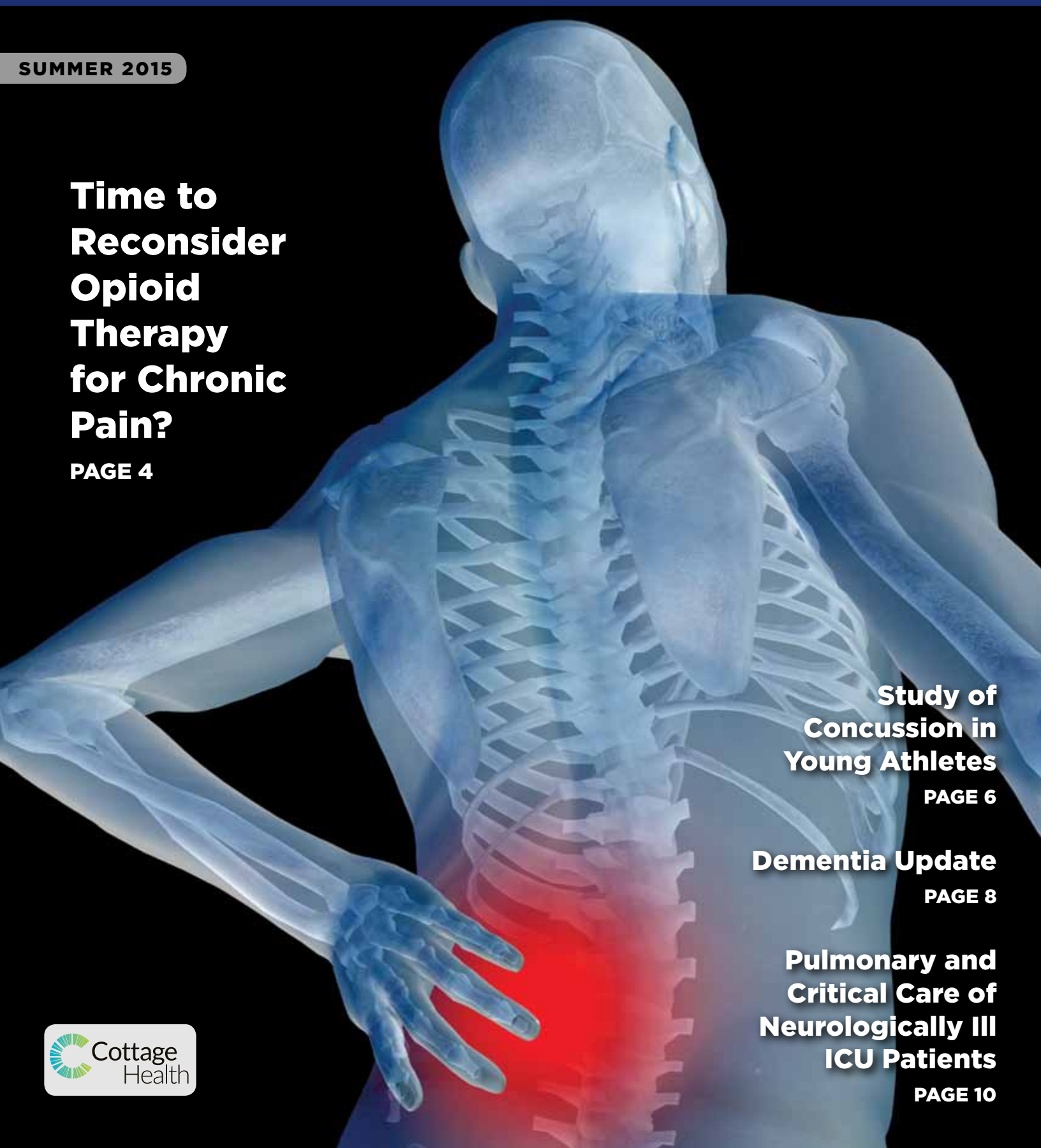
NEUROtransmitter

A PUBLICATION OF SANTA BARBARA NEUROSCIENCE INSTITUTE AT COTTAGE HEALTH

SUMMER 2015



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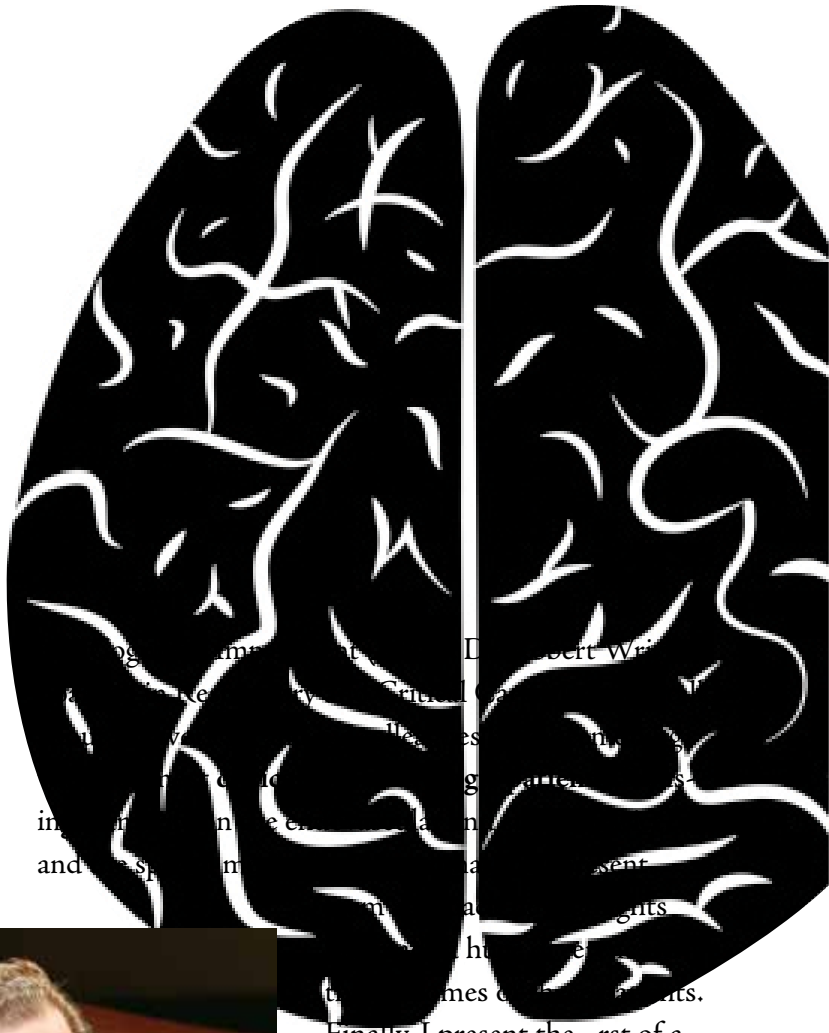
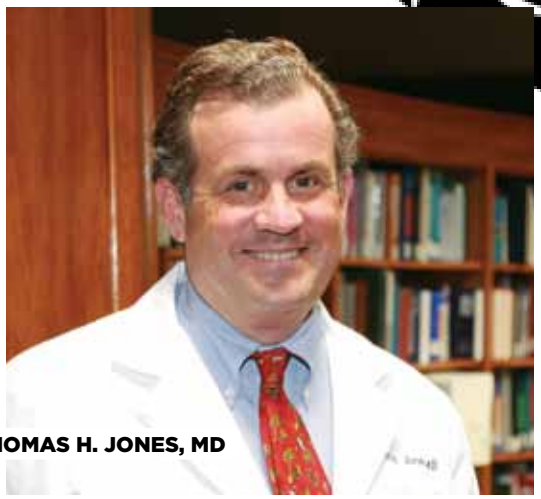
Cottage Health



Dear Colleagues,

is issue of *NEUROtransmitter* is the 14th we have published since our inaugural magazine ve years ago. In each issue, we have tried to inform our regional medical community about the latest neuroscience developments and, perhaps most important, how they impact our care in the community. We have attempted to carry out this mission using Michael Porter s lens of health care quality, de ned by him as health care outcomes achieved per dollar spent.



THOMAS H. JONES, MD

In this issue, we present a variety of articles for your consideration. Dr. Stephen Kaminski, the Director of the Cottage Health Trauma Service, writes an interesting article on **concussions** in young people playing sports and talks about some of his clinical research interests. Dr. Kaminski is boarded not only in Critical Care Medicine but also in Neurocritical Care and, therefore, has special training and interest when it comes to all head injuries. Next, we have a **dementia** update article by Dr. Robert Harbaugh, who adds to the information he provided our medical community in our fall 2012 publication (http://www.cottagehealth.org/app/ les/public/991/Neurotransmitter-Fall-2012.pdf). Dr. Harbaugh, a general neurologist by training, has developed a subspecialty expertise in the clinical diagnosis and management of cognitive dysfunction. In his article in this issue, he breaks down the various common forms of dementia and introduces us to the concept of Mini-

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and

Finally, I present the rst of a two-part article on what I feel is the evolving paradigm shift in the management of **chronic pain**, particularly emphasizing a growing sentiment that opiates may be contraindicated.

We hope that our readers will learn from these experienced clinicians and that this will result in some practice changes that better align with evidence-based medicine. We also invite all interested to attend our **8th Annual Saving the Brain Conference** (www.cottagehealth.org/neuro), which is further referenced on the back cover of this publication. is is the rst year we have held it on a Friday and, as usual, we look forward to clinically relevant talks at a great venue.

Sincerely,

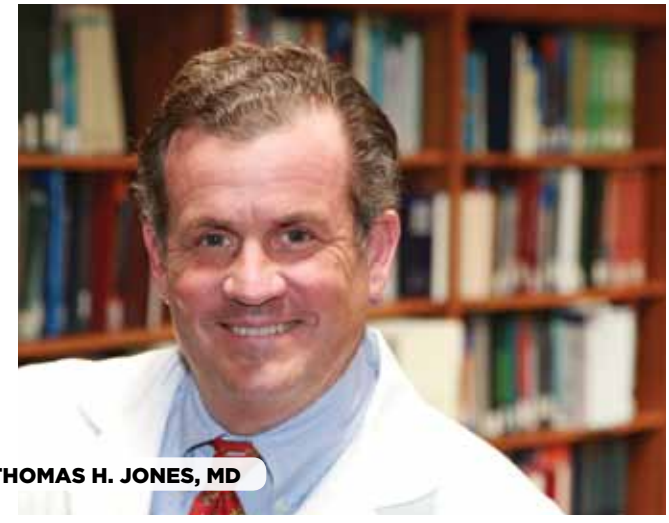
THOMAS H. JONES, MD
Neurosurgeon and Medical Director
Santa Barbara Neuroscience Institute at Cottage Health

Opioid Therapy for Chronic Pain: Time for Reconsideration?

BY THOMAS H. JONES, MD, NEUROSURGEON AND MEDICAL DIRECTOR SANTA BARBARA NEUROSCIENCE INSTITUTE AT COTTAGE HEALTH

"Opium is a bitter, brown, granular powder derived from the seedpod of the poppy. People have used opium for relief of pain and suffering for thousands of years. The morphine alkaloid was isolated in 1806 and it was then that pharmacological production began." (NEJM, 2003; 349:1943-53) Legal use of morphine and its analogues became tightly restricted in the 1940s and soon thereafter became the province of licensed physicians to use for their patients at their discretion.

Throughout the 20th century, physicians were taught that opioids should be used to treat **acute and chronic pain.** Pain management "experts," either self-anointed or unwitting surrogates of the pharmaceutical industry, recommended using a 10-point scale to gauge the level of relief and pushed the concept that slowly escalating the drug dosage and using longer-acting drugs (oxycontin, methadone) might be better. They would routinely push dosages above the 180mg morphine equivalent dosage daily. They would remind all of us that just a small percentage of patients developed drug addiction that could be harmful.



THOMAS H. JONES, MD

The American Pain Foundation, the largest self-proclaimed advocacy group for pain patients, stated that the risk of addiction was overblown and that opiates were, in fact, underutilized. Only later was I made aware that this foundation receives 90 percent of its funding from the drug and medical device companies. Additionally, some of the foundation board members had extensive ties to the drug makers. In this environment that they helped to create, the pharmaceutical industry that produces such opiates has hit the motherlode. "Painkiller" sales have increased fourfold since 1999. In 2004, Purdue Pharma, the maker of oxycontin, alone had gross sales of $1.7 billion from that one drug and a few years later sold $2.8 billion worth. The United States has achieved a 90 percent market share of all the world sales of Vicodin and 80 percent of all the opiates sold! In this environment, by 2008 deaths from prescription opiate overdoses surpassed the combined overdose deaths of cocaine and heroin. Recently, the Centers for Disease Control noted 14,800 deaths from opiate overdosage, representing 74 percent of all prescription overdosage deaths.

Escalating Use of Opiates

As a physician/surgeon practicing for over 30 years, I began to question what I was being told about 20 years ago. I saw chronic pain patients deteriorating yearly and yet their neurologic exams and MRI scans were largely unchanged. The only constant was escalating use of opiates prescribed by physicians. I bought a book to update my knowledge on the changes

in our understanding of pain pathway physiology since I finished medical school; I read the pain literature and looked hard for carefully controlled studies that would support opiate use in chronic pain patients. I was at first shocked and later appalled to realize that almost all the studies had been subsidized – even edited – by the pharmaceutical industry and that there were literally no randomized controlled trials scrutinizing the use of opiates in chronic pain states (i.e., over six months). What trials there were that came close were generally Veterans Administration-sponsored studies, not funded by any pharmaceutical money, that suggested chronic pain was adversely impacted by opiate use.

I then stumbled upon the concept of **opiate induced hyperalgesia (OIH)** and I finally began to realize the fallacy behind the use of opiates for chronic pain. This explained why most of the opiate-dependent patients that I have cared for over the years had much more pain during ordered tests (e.g., discograms, myelograms, lumbar punctures) and surgical procedures than my non-opiate dependent patients. It also explained why most of these patients told me that they didn't think the medications helped them, and they didn't appear to me to be functioning at a higher level than before opiates had been started. A recent self-aware patient told me that she knew something was wrong when just combing her hair hurt her scalp.

Opioid-Induced Hyperalgesia

It turns out that there is a large body of literature looking at animal models, former addicts on methadone and even patients in the recovery room after one dose of a powerful narcotic (fentanyl) intra-operatively. The pathophysiology of this paradoxical effect hasn't been completely worked out yet though probably has something to do with NMDA receptors, glutamate neurotransmitter release, etc. I finally realized why my patients were worsening every year. They were becoming **hyperalgesic on opiates.** The opiates were increasing rather than decreasing their perception of pain. I then found out that those patients who had the fortitude to taper off opiates were actually much better, had more manageable pain and were functioning at a much higher level. Other revelations include the now-known adverse impact of chronic opiates on the immune system, on the hypophyseal-adrenal (low cortisol levels with reduced energy and disordered mentation) and hypophyseal-gonadal (amenorrhea, galactorrhea, low testosterone) systems.

Based upon what I have witnessed as a clinician and what I have read in the world's literature, I would generally not recommend opiates for non-cancer-related chronic pain states. There are few exceptions. They are not only ineffective, they also have an adverse impact upon our pain perception and create other, often sub-clinical physiologic disturbances that may result in permanent harm. I believe that there are better pain control alternatives that are not associated with half of these risks. These will be discussed in a follow-up article in the next edition of this magazine.

For more information on the Santa Barbara Neuroscience Institute at Cottage Health, visit www.sbni.org

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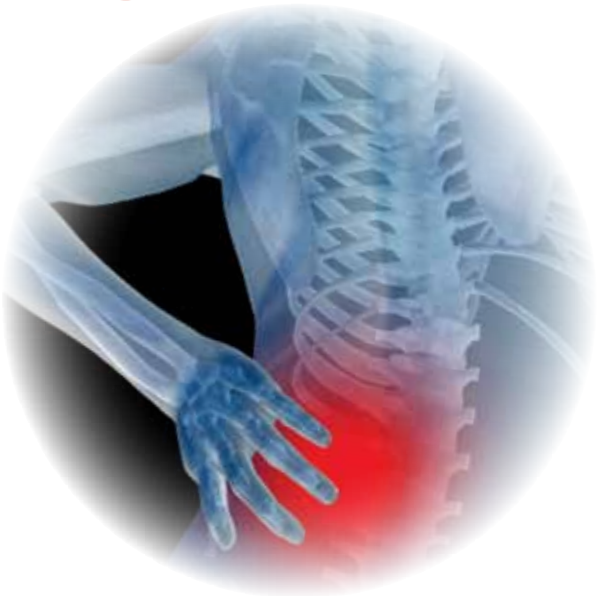


Collaborative Study Focuses on Concussion in Young Athletes

BY STEPHEN S. KAMINSKI, MD

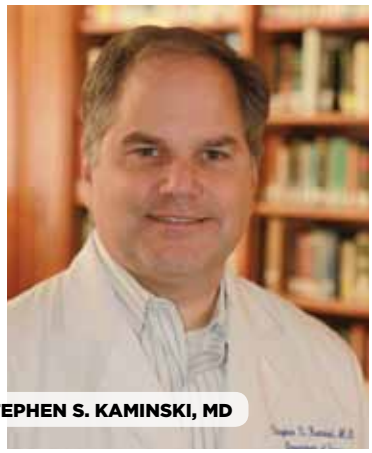
Trauma Service of Santa Barbara Cottage Hospital and Santa Barbara City College Athletic Department examine whether some individuals' brains are more susceptible to force than others.

An estimated 30 million or more children and adolescents participate in sports in the United States. For those suffering a traumatic brain injury as a result of their activities, as presented in case study No. 1 (see opposite page), that injury is associated with a clear pattern. There is also an understanding of both how to treat the initial presentation and how to rehabilitate a patient following the event. Concussion or mild traumatic brain injury (mTBI) as presented in case study No. 2 remains much more of a mystery.

Given the large number of children participating in sports and other contact activities, concussion has become an important topic in the spectrum of brain injury. By some estimates, as many as 1.5 to 3 million children and adolescents per year may suffer a concussion. In adults there has been much reporting on the potential chronic effects of repetitive brain trauma with linkage of behavior change, depression and even suicide to pathologic changes seen at the cellular level in professional athletes. To avoid the risk of chronic traumatic encephalopathy, emphasis has been placed on early recognition and treatment of concussion in youth athletes. A 2011 California law now requires that student athletes suspected of suffering a concussion be removed from activity and not returned to play until they have been evaluated by a health care professional experienced in the management of concussion.

Defining Concussion

The Centers for Disease Control (CDC) and an international consensus statement have defined concussion as a complex pathophysiological process affecting the brain induced by traumatic biomechanical forces. Unfortunately, this somewhat vague terminology describes a broad spectrum of disease, but it does point out the important role of force. Although force is linked to the manifestation of symptoms, it is unclear whether a uniform force results in injury or, rather, if there is individual variability that predisposes one to concussion. In other words, are certain individuals' brains more susceptible to force and have a lower threshold for injury and the development of symptoms than others? This question has formed the basis for a recently conducted collaborative study between the Trauma Service of Santa Barbara Cottage Hospital and the Athletic Department of Santa Barbara City College.



STEPHEN S. KAMINSKI, MD

ImPACT Testing

As one can imagine, it would be difficult both to get approval for and to have youth athletes sign up for a study in which they would be struck in the head until they develop a concussion. Instead, we approached the question by trying to gain information from events that were already occurring. ImPACT testing (Immediate Post-Concussion Assessment and Cognitive Testing) is an increasingly popular evaluation of baseline and post-injury cognitive function used in athletes playing sports in which concussion is a risk. The test is computerized and takes an athlete about 25 minutes to complete. The test measures multiple aspects of cognitive function in athletes, including memory, reaction time, attention and problem solving. As part of the study, students participating in football, men's soccer and women's soccer underwent ImPACT testing pre- and post-season. Students also completed a symptom survey following the season. From these tests we have looked to gain information on concussive symptoms and effects on cognitive function.

PHOTOS: NICOLE HAUN

Triax headbands are worn by Santa Barbara City College football and women's soccer players during practice to track head impact data.

Measuring Force

The next step was to evaluate if there is linkage to the cognitive elements with varying levels of force experienced by athletes during the season. Athletes were required to wear a force-sensing headband manufactured by Triax Technology (triaxtec.com) during the season for both practice and games. There are two types of force an athlete can encounter. One is direct, linear or head-on. The other is rotational. Rotational force is considered by some to be more important. As the brain is fluid within the skull, non-linear force can result in shear stress or impact on the brain that may not be the product of direct contact. The Triax Technology device uses a 3-axis accelerometer and gyroscope to obtain accurate linear and rotational impact measurements.

Using state-of-the-art computerized cognitive function testing and force sensors, our hope is to better understand the relationship between force, the development of concussion symptoms and changes in cognition, as well as to explore differences where they exist between men and women athletes. A better understanding of these elements will assist us in developing the best treatment for concussion, which is ultimately prevention.

For more information on treatments for concussion and other services available at the Santa Barbara Neuroscience Institute at Cottage Health, visit www.sbni.org

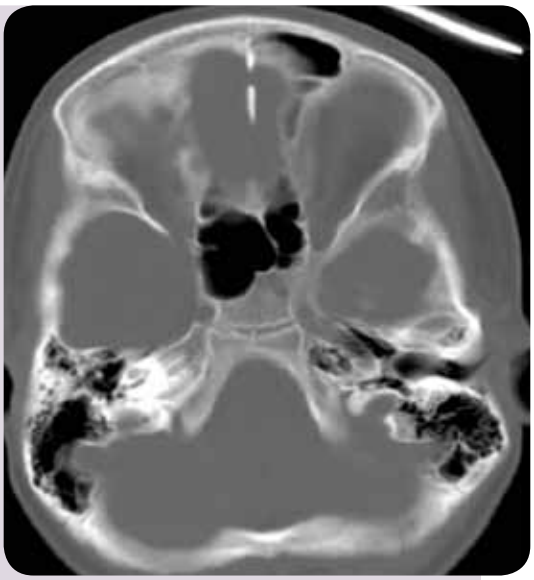
Two Different Sports-Related Brain Injuries

CASE STUDY No. 1

A 14-year-old girl is twirling in a sitting position while skating, commonly called a sit spin. She falls, striking the back of her head on the ice. She suffers a loss of consciousness of less than one minute and subsequently complains of a headache. Her neurologic examination is normal. She undergoes head CT imaging which demonstrates a hairline posterior occipital skull fracture and she has a possible and barely perceptible parenchymal contusion underlying the fracture. She subsequently complains of double vision and dizziness when standing. When she attempts to resume physical activity, she suffers shortness of breath and describes a balance or gait instability. Repeat imaging has shown the contusion to have cleared. She has trouble concentrating in school and her symptoms take three months to resolve.

CASE STUDY No. 2

A 15-year-old boy is playing high school football when he is upended, landing on his head. He denies suffering, and is not observed to have suffered, a loss of consciousness. He is taken out of the game and, on the sideline, complains of headache, nausea and vomiting. He undergoes head CT imaging, which is unremarkable. He has persistent headaches, nausea and difficulty concentrating. His school work suffers. After six months, he starts to show some improvement in his symptoms. Interim repeat imaging remains unremarkable.



Case Study No. 1: The bone windows of a non-contrast head CT reveal a linear left-sided posterior occipital skull fracture.

Dementia Update: Current Concepts Best Not Forgotten …

BY ROBERT HARBAUGH, MD

Following are brief updates spanning the more commonly encountered degenerative dementias. *Be prepared for some major re-thinking!*

◆ Dementia is not a disorder or a disease but an umbrella term that describes a functional state in which an individual demonstrates an acquired loss of cognitive and/or behavioral competence (inability to live independently), usually in later life, evolving over time (months to years). This transition point is not easily defined and typically occurs in various spheres over time. Over 100 distinct disorders have been described as causal, with degenerative disorders creating > 85 percent.

◆ Dementias are exceedingly complex by nature. Persons affected by late-life dementia follow unique clinical profiles, have unique caregiver support systems, and demonstrate dynamic disease expressions over time. In addition, more detailed clinicopathological studies have shown a greater commingling of underlying pathological substrates as well as more diverse clinical expressions than has been previously assumed.

◆ Arguably, Minimal Cognitive Impairment (MCI) is, in fact, the most common member of the dementias when viewed as a clinical spectrum. Also known as "minor neurocognitive impairment" in the new DSM-V nomenclature, MCI describes changed cognitive functioning in which one demonstrates a measurable change in generally only one (or minor changes in other) sphere(s) of cognition (e.g. short-term memory), and in which no other degradation in intellectual or behavioral competence is present (not demented). Approximately 15 percent of those manifesting MCI will develop dementia annually, but not all persons are obliged to become demented.

ROBERT HARBAUGH, MD

◆ We are entering a period of major rethinking about the degenerative dementias – many of our assumptions about their pathophysiological underpinnings and when/how to modify disease expression will need to be reshaped (or discarded), especially with respect to Alzheimer's disease (AD). Degenerative dementias start decades before the "damage is done." The most effective treatments will follow this principle. However, small changes in disease trajectory (a more tangible realization in the short term) are possible.

◆ A strong unifying theme of misfolded, aberrant native proteins within the CNS appears to be the basis of many degenerative dementias. However, the specific molecular sequences remains elusive.

◆ A constellation of clinical tools is needed to accurately diagnose degenerative dementias, with limited biomarkers currently available (mostly limited to research centers).

◆ Alzheimer's disease affects 60–70 percent of those with a dementia, as the

sole or as a contributing disorder, but it is by no means the only "dementia."

◆ Lewy Body disease (LBD, also known as Dementia with Lewy Bodies), a complex disorder only widely described in the past 20 years, is now the second-most common identified dementia. In reality a "spectrum" disorder, overlapping Parkinson's disease clinically and pathologically, LBD retains a distinctive clinical profile, with remarkable fluctuations demonstrable.

◆ Vascular dementia (VaD) is a complex array of several interacting subsets of the cerebrovascular disorders. In addition, VaD is analytically challenging without in-depth information about the patient's precise signs and symptoms (with exacting temporal profiles), coupled with detailed neuroimaging and neuropsychometric studies. At least four subsets of VaD may co-exist.

◆ The frontotemporal dementias are a diverse set of several distinctive, unrelated pathological entities which create primarily "behavioral" dementias, but which can cause language specific disorders initially. These disorders typically appear in the late middle years, challenging AD in prevalence before the age of 60.

◆ FDA-approved anti-dementia drugs (e.g. Aricept, Namenda) are useful for small, but meaningful and cost-effective benefits in treated populations of those affected by AD and Parkinson's disease/Lewy Body disease spectrum. Studies do not show a role in disease modification or a consistent benefit in populations with MCI.

◆ The National Alzheimer's Project Act (NAPA), a large national effort to manage AD and related disorders, created in 2011, estimates the availability of disease-modifying agents possibly by 2025. There is, unfortunately, no scientifically proven disease-modifying agent for any degenerative dementia at this time. Because of the intrinsic nature of the degenerative dementias, redundant proof – several studies unequivocally showing benefit with acceptable adverse effects, administered for a long period (presumed) – will require many years to complete, even if a putative cure for AD or related dementia is available now.

◆ Small improvements in dementia costs, caregiver burden and societal impact can create major leveraged gains.

◆ The behavioral and psychological symptoms of dementia (BPSD), create major disabilities but are, arguably, the most "treatable" elements of the dementia epidemic. Many of these behavioral transgressions are amenable to medical interventions if management is started before crises erupt.

◆ Late-life depression is highly correlated with degenerative dementias, and appears likely to be another early behavioral manifestation of a variety of degenerative dementias.

◆ Bold, innovative strategies will be needed to address the Gray Tsunami or future generations may experience a different standard of living.

For more information on dementia and the Santa Barbara Neuroscience Institute at Cottage Health, visit www.sbni.org

CASE STUDY: From Memory Impairment to Dementia

A 71-year-old right-handed married male, a retired insurance agent, was seen for a one-year history of mild "memory" impairment.

The patient's family had noted subtle, uncharacteristic changes in recall and memory. Conversations were sometimes not well recalled – for example, an important lunch date with a close friend was uncharacteristically overlooked on three occasions during the prior six months, and news items, which previously had been a topic of dinner conversation, were not well recalled in detail or were recalled with erroneous content. In addition, other family members commented about a lack of initiative, coupled with a more complacent personality. The patient is fit, happily married and very active.

The patient attained a four-year college degree. The past medical history was remarkably unrevealing. A great aunt died from apparent "dementia" at age 88. The patient took lisnopril for HTN. The neurological ROS was normal.

The physical and neurological exam were unremarkable, but with a Montreal Cognitive Assessment (MOCA) screening exam demonstrating a score of 25/30 (MMSE variant) – a borderline low score.

A brain MRI and basic dementia labs were unrevealing. On formal neuropsychological evaluation, mild deficits in new learning, work list generation and executive functioning were noted for age, gender and educational achievement.

In follow-up consultation, his neurologist diagnosed "mild cognitive impairment, amnestic type," and recommended continued lifestyle interventions for optimizing brain fitness and a follow-up in three to six months (with his wife in attendance). Limitations in the efficacy of current anti-dementia drugs were outlined. Consideration for referral to a tertiary care center for research consideration was discussed. It was strongly suggested financial activities be overseen or transferred to others.

Two years later, the patient had progressed "measurably." He continued to live independently with oversight, but no longer drove nor engaged in financial transactions. He needed more oversight at home; all parties agreed he had become demented. Donepezil was initiated and family counseling ensued.

Managing the Pulmonary and Critical Care of Neurologically Ill Patients in the ICU

BY ROBERT S. WRIGHT, MD

There is now uniform agreement among physicians and nurses taking care of the most critically ill patients that the team approach enhances outcomes, improves patient and family communication and may lower cost of care. The pulmonologists and intensivists of the Santa Barbara Neuroscience Institute have been involved with the co-management of seriously ill brain-injured patients since the inception of the program at Santa Barbara Cottage Hospital in 2007.

These doctors round daily on the neurologic patients in the ICU, often with the entire care team, and are involved with every intubated patient. Care is coordinated with the attending neurologist/neurosurgeon and also with the nurse practitioners of the neurologic service, the ICU nurse team, case managers and pharmacists.

These patients most commonly have a subarachnoid hemorrhage (SAH), a large intracranial bleed or a stroke. The majority of our patients are intubated and frequently treated with therapeutic hypothermia. These latter patients are heavily sedated and paralyzed with neuromuscular blockers. This, in turn, leads to a unique set of circumstances requiring special expertise. Involvement revolves around three basic management issues: 1) control of ventilation, 2) management of respiratory failure and the pulmonary complications of treatments for the brain injury, and 3) general medical care of the brain-injured patient.

Key Management Issues

Control of ventilation is maintained by monitoring end-tidal CO_2 and arterial blood gases. CO_2 is maintained generally around 32-34 mm Hg to help mitigate brain swelling as hypercarbia can produce cerebral vasodilation and edema.

Respiratory failure initially is iatrogenic due to the sedation, neuromuscular blockage and hypothermia necessary for the treatment of subarachnoid hemorrhage. Due to brain swelling and other processes such as cerebral vasospasm, large volumes of fluids are given to maintain brain perfusion. Unfortunately, this can lead to cardiogenic pulmonary edema, large pleural effusions and hypoxemia. Oxygenation is monitored carefully by oximetry, blood gases and brain tissue oxygen tension ($PbtO_2$). When monitored, the neurologists like to see the $PbtO_2$ above 15 mm Hg. Often times, increasing the blood pressure with vasopressors can augment brain oxygenation. However, frank hypoxemia with SaO_2 saturations less than 92 percent requires aggressive maneuvers. Lobar atelectasis is treated with bronchoscopy and is frequently required multiple times in a hospitalization. Refractory hypoxemia requires recruitment maneuvers and the use of high PEEP. Although we generally start out with conventional volume ventilation, we have found that novel ventilator management is sometimes required. Airway pressure release ventilation, or APRV, is frequently used. This is basically an advanced form of inverse ratio ventilation in which inspiration is prolonged and exhalation is shortened significantly. Additionally, we try to keep peak airway pressures less than 30 cm H_2O. Prolonged intubation is common and we frequently intervene early with percutaneous tracheostomy.


ROBERT S. WRIGHT, MD

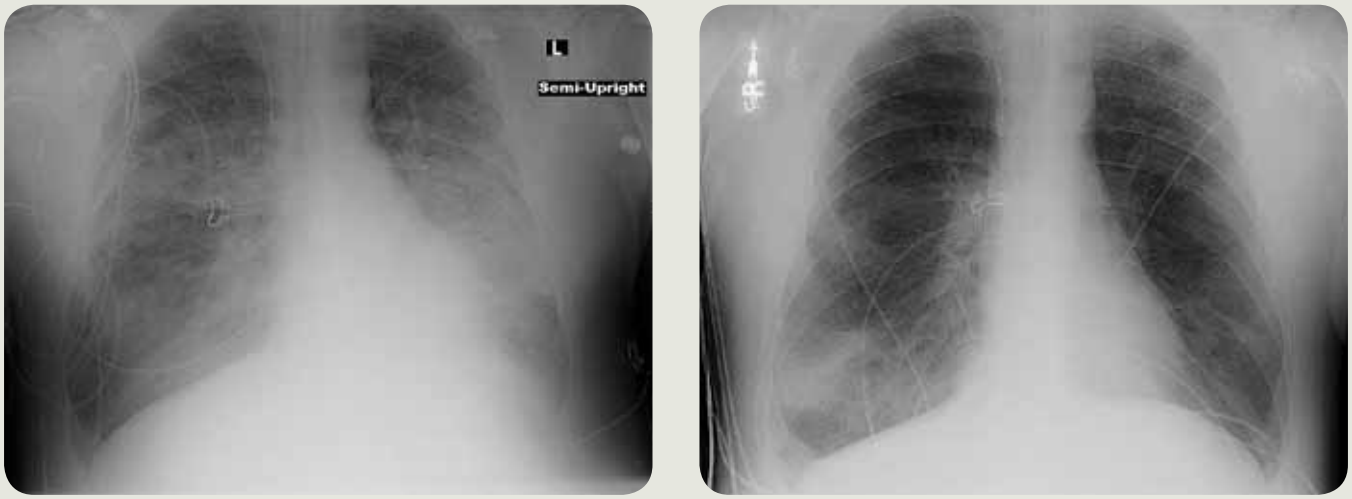
A very important aspect of management is the prevention of ventilator-associated pneumonia, or VAP. VAP not only leads to significant morbidity and mortality but is now considered an avoidable complication of medical care according to the Centers for Medicare and Medicaid. Since pulmonologists have become involved with the care of these patients, the incidence of VAP has dropped ten-fold.

We are also involved with the **general care** of the patients. Important issues are prophylaxis for thromboembolism and nutrition. The use of prophylactic anticoagulants can be a conundrum especially in patients with SAH. Consequently, timing and dosing becomes important. When pulmonary embolism occurs, we often have to resort to placement of inferior vena cava filters due to the risk of intracranial hemorrhage with systemic anticoagulation. As the patients are intubated and heavily sedated, they are unable to eat for days or weeks at a time. Consequently, our team is very aggressive in starting early enteral nutrition. Small nasogastric feeding tubes are placed past the pylorus and enteral nutrition is started almost from the onset of their hospital

CASE STUDY: Complex Critical Care of a Patient with a Severe Aneurysmal Subarachnoid Hemorrhage

A 59-year-old man was found unresponsive in his bathroom. He was taken to a nearby hospital and a head CT scan showed a diffuse subarachnoid hemorrhage (SAH). He was transferred to Santa Barbara Cottage Hospital by air ambulance for a higher level of care. CT angiography was immediately performed and he was found to have a 3 mm anterior communicating aneurysm with a small tête suggesting this was the bleeding site. The aneurysm was coiled. His Hunt and Hess grade was 4 and he was Fisher grade 3. A ventriculostomy was performed, a licox monitor was placed and he was started on cooling measures with an intravascular cooling catheter. Arterial and central lines were placed and he was heavily sedated and paralyzed while intubated and mechanically ventilated.

He required vigorous hydration to prevent and ameliorate vasospasm. He developed deep venous thrombosis requiring the placement of an IVC filter as therapeutic anticoagulation was contraindicated in this setting. However, he had a sudden episode of marked hypoxia and CT scanning showed a pulmonary embolism. He was started on a heparin infusion with tight regulation of his PTT at the lower end of the therapeutic scale. He developed sinusitis requiring ceftriaxone. His pulmonary status became progressively worse and he became more hypoxic and hypercarbic. Conventional volume ventilation was no longer effective, so he was switched to airway pressure release ventilation (APRV) which allows prolonged inspiratory times and a very short expiratory time. This allowed more effective recruitment of atelectatic alveoli and improved his oxygenation. This is demonstrated in the chest radiographs shown below. However, this alone was not effective and a decision was made to prone ventilate the patient. He was carefully moved by a team of six nurses and therapists from the supine position to the prone position while securing all catheters and his endotracheal tube. This was performed two days in a row, each time maintaining him in the prone position for 16 hours at a time. Additionally, he became oliguric and unresponsive to usual dose of diuretics. A high-dose furosemide infusion was started and this led to a significant improvement in his oxygenation. Lobar atelectasis required multiple bronchoscopies by the pulmonologist. After considerable effort and the combination of APRV, aggressive diuresis, multiple bronchoscopies and appropriate antibiotics, he began to improve. Once improved, he remained quite weak and unable to ventilate easily, so a percutaneous tracheostomy was performed. Subsequently, he was weaned off the ventilator and became strong enough to be transferred to a rehabilitation hospital where he is making excellent progress.



Left Image: Chest radiograph showing diffuse alveolar infiltrate consistent with ARDS. Right Image: Chest radiograph showing marked clearing of infiltrates with residual abnormalities in the lower right lung.

stay. Our team has a full-time registered dietician who helps manage the supplements. Most patients have their feeding tubes converted to a percutaneous gastrostomy tube. This lessens the likelihood of sinus infection and inadvertent feeding tube removal. A strong nutritional program clearly helps recovery for these patients, who would otherwise become malnourished.

Patients with neurological problems in the ICU often present with complex clinical problems and, as discussed above, management can be challenging. However, there is nothing more gratifying than helping bring a critically ill patient back from the brink of death and return home to their family. The pulmonologists of this service are understandably proud of the care they provide.

For more information on Pulmonary and Critical Care of Neurologic Patients available at the Santa Barbara Neuroscience Institute at Cottage Health, visit www.sbni.org

Cottage Health

Santa Barbara Cottage Hospital
400 West Pueblo Street
P.O. Box 689
Santa Barbara, CA 93102-0689

On behalf of the Santa Barbara Neuroscience Institute, you are cordially invited to attend

Featuring nationally recognized guest speakers, along with experts in the Neurosciences from Santa Barbara Cottage Hospital.

FRIDAY, OCTOBER 9, 2015 | 7:00 am to 4:30 pm

The Fess Parker - Doubletree | Santa Barbara, California 93103

Registration is $75.00 (before September 25). Register online at:

cottagehealth.org/neuro

Cottage
Santa Barbara
Neuroscience Institute

SAVING THE BRAIN 2015 SPEAKERS

Welcome and Overview
Thomas H. Jones, MD
Neurosurgeon and Medical Director, Santa Barbara Neuroscience Institute at Cottage Health

Failure is Not an Option: A Brain Stem Injury Survivor's Story
Earle Powdrell
Aerospace Engineer, Stroke Survivor

MR Tractography to Visualize Nerve Fibers in the Setting of Nerve Injury
Michel Kliot, MD
Neurosurgeon; Interim Chair, Department of Neurosurgery; and Director of Peripheral Nerve Center, Northwestern University, Feinberg School of Medicine, Chicago IL

Let's See How Far We've Come: Intraoperative MRI
Maureen W. Hemingway, MHA, RN, CNOR
Nursing Practice Specialist, Massachusetts General Hospital, Boston MA

Compassion, Fatigue, Vicarious Trauma and Burnout: What to do When There is Nothing Else to Give.
Jennifer M. Fields, BSN, RN, CCRN
Neuroscience ICU,
University of Cincinnati Medical Center

Brain Tumor Management
John Park, MD, PhD
Neurosurgeon and Medical Director, Brain and Spinal Tumor Program, Santa Barbara Neuroscience Institute at Cottage Health

Treatment of Newly Diagnosed Glioblastoma: Molecular and Clinical Factors and Treatment Options
Jennifer L. Clarke, MD, MPH
Associate Professor of Clinical Neurology and Neurosurgery, Division of Neuro-oncology, University of California, San Francisco

Stroke Care 2015 – What Has Changed?
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Establishing a Regional Stroke Initiative
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Case Studies in Neuroendovascular Care
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