Osborn's BRAIN

IMAGING, PATHOLOGY, AND ANATOMY

Anne G. Osborn

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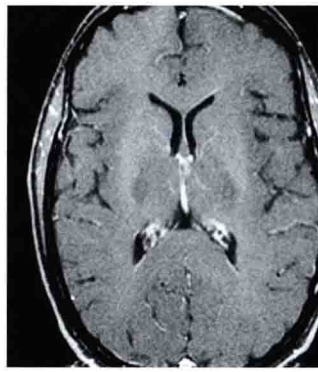

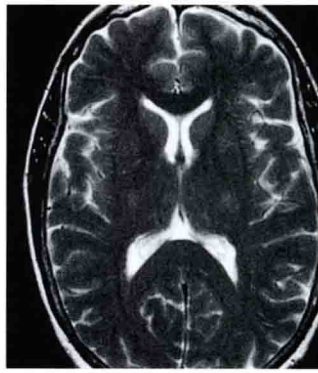

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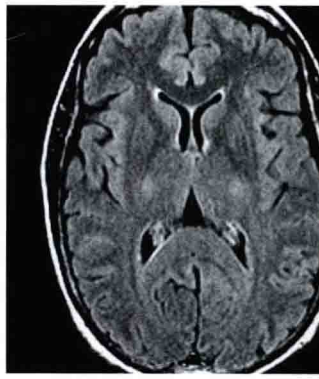

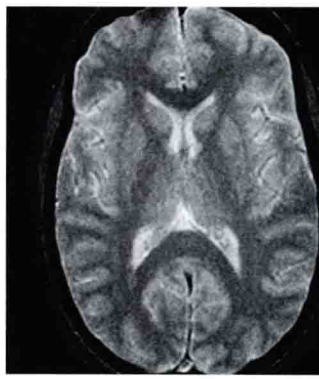

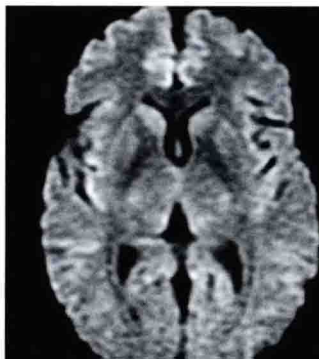

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FOR RON

Beloved sweetheart and eternal companion, you didn't live to see the book completed. Nevertheless, your unconditional love and supportive spirit sustained me throughout the process—from beginning to the very end. I hope it makes you proud! Until we meet again, all my love and devotion right back at you!

PREFACE

With the publication of *Osborn's Brain*, I'm breaking a longstanding promise to myself: I swore I'd never, *EVER*, write another prose-based book. And yet here it is. But let me tell you, this isn't just "another prose book." Far from it! When my colleagues and I published the first edition of *Diagnostic Imaging: Brain*, Dr. Michael Huckman said in his Foreword to the book, "[Osborn] has decided to abandon the usual conventions of medical textbooks." Amirsys's now-classic bulleted format does indeed deliver more information in less space than traditional prose. And I do love those efficiencies! However, I want to give more than, "Just the facts, ma'am." I want to show the thinking *behind* the facts. The reasoning. The framework that facilitates understanding a tough, complex subject.

That's why I've structured the book as a learning curriculum. We start with the most immediate "must know" topics, beginning with trauma. We next discuss nontraumatic hemorrhage, stroke, and vascular lesions. In other words, we jump right into emergent imaging issues before delving into infections, demyelinating and inflammatory diseases, neoplasms, toxic-metabolic-degenerative disorders, and congenital brain malformations.

If you're just starting your residency in radiology, neurosurgery, or neurology, I suggest you begin at the beginning. Read the first three chapters and digest them. Then go part by part, chapter by chapter, straight through the book. If you are a senior resident or fellow, this is a great way to review what you think you already know pretty well. I guarantee you, there's stuff in here that will be new to you. If you're a practicing general radiologist, neuroradiologist, or neurosurgeon, consider this a neuroimaging refresher course. And if you are an honest-to-goodness neuroradiologist, I've tucked a number of cool tidbits into every chapter that I hope you will find intriguing and thought-provoking.

Many of you have asked, emailed, and even written (yes, old-fashioned written) me with your pleas for a new "Osborn." So here it is. I wrote every word of it myself, so the style is mine alone and the approach is therefore consistent from chapter to chapter. I've combined essential anatomy together with gross pathology and imaging to show you just why diseases appear the way they do. The book is illustration-rich, with loads of high definition state-of-the-art imaging and glorious color. My trademark summary boxes are scattered throughout the text, allowing for quick review of the essential facts.

I've drawn on an entire career of accumulated knowledge and intense interest in neuropathology, neurosurgery, and clinical neurosciences to select the very most relevant information for you. It's been fun to do this, the culmination of my decades of continued learning in our beloved subspecialty. I hope you enjoy the journey!

Best regards and good reading!

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No one truly ever produces a text of this magnitude alone. While I am the sole author, there are many individuals who have contributed everything from images to suggestions, opinions, and ideas. Thank you from the bottom of my heart. You know who you are.

Several individuals and groups deserve special mention. First of all, thanks to our neuroradiology colleagues at the University of Utah for their support. A big one to Brian Chin, my 2011-12 clinical neuroradiology research fellow who tirelessly searched out cases and references for the book. Couldn't have done it without you!

Ever since my sabbatical as Distinguished Scientist at the world-renowned Armed Forces Institute of Pathology in Washington, D.C. (which alas is no more, a victim of government downsizing), pathology has formed the foundation of how I view and teach neuroradiology. It's a big part of this text. Special thanks to Richard H. Hewlett and his colleague, the late Stuart Rutherfoord, whose elegant gross photographs make the book sing. Thanks also to Peter Burger, the late Bernd Scheithauer, and their wonderful neuropathology colleagues. Some of the images reproduced here come from their wonderful synoptic text, *Diagnostic Pathology: Neuropathology* (Amirsys Publishing, 2012).

Thanks to the entire Amirsys team. Special mention and profound thanks goes to our inimitable, indomitable, and beloved colleague Paula Woodward, MD. Paula stepped into the editing and production wherever needed. At the end, she jumped in to do a bunch of heavy lifting with the medical edits to keep us on a very tight, unforgiving schedule. Ashley Renlund, our chief editor, made invaluable suggestions, polishing the text and layout. She worked tirelessly to fit almost all the images on the same or facing pages with the referenced text.

Thanks to the international Amirsys Brain, Spine, and Head and Neck case teams. You guys have contributed amazing stuff over the years to the Amirsys database, which now reaches tens of thousands of radiologists and trainees through STATdx™ and RadPrimer™. You have enhanced teaching and improved patient care around the world. Thanks for your superb work...and prompt response when any of us emailed an urgent "request for cases" for one of our many projects. It didn't matter how common or obscure the diagnosis, someone somewhere (and often several of you) sent a perfect case.

Thanks also to the many colleagues who have generously given me fascinating cases over the years. I've tried to keep track of which case came from whom and to acknowledge you appropriately in the captions. Special thanks to all of the image contributors.

-Anne G. Osborn

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Trauma Overview

Trauma is one of the most frequent indications for emergent neuroimaging. Because imaging plays such a key role in patient triage and management, we begin this book by discussing skull and brain trauma.

We start with a brief consideration of epidemiology. Traumatic brain injury (TBI) is a worldwide public health problem that has enormous personal and societal impact. The direct medical costs of caring for acutely traumatized patients are huge. The indirect costs of lost productivity and long-term care for TBI survivors are even larger than the short-term direct costs.

We then briefly discuss the etiology and mechanisms of head trauma. Understanding the different ways in which the skull and brain can be injured provides the context for understanding the spectrum of findings that can be identified on imaging studies.

Introduction

Epidemiology of Head Trauma

Trauma—sometimes called the "silent epidemic"—is the most common worldwide cause of death and disability in children and young adults. Neurotrauma is responsible for the vast majority of these cases. In the USA alone, more than two million people annually suffer a traumatic brain injury. Of these, 500,000 require hospital care. At least 10 million people worldwide sustain TBI each year.

Of all head-injured patients, approximately 10% sustain fatal brain injury, and an additional 5-10% have serious permanent neurologic deficits. Even more have subtle deficits ("minimal brain trauma"), while 20-40% of TBI survivors have moderate disability.

Etiology and Mechanisms of Injury

Trauma can be caused by missile or non-missile injury. Missile injury results from penetration of the skull, meninges, and/or brain by an external object such as a bullet.

Non-missile closed head injury (CHI) is a much more common cause of neurotrauma than missile injury. High-speed motor vehicle collisions exert significant acceleration/deceleration forces, causing the brain to move suddenly within the skull. Forcible impaction of the brain against the unyielding calvaria and hard, knife-like dura results in gyral contusion. Rotation and sudden changes in angular momentum may deform, stretch, and damage long vulnerable axons, resulting in axonal injury.

The etiology of TBI also varies according to patient age. Overall, almost 30% of TBIs are caused by falls. Falls are the leading cause of TBI in children younger than four years and in elderly patients older than 75. Gunshot wounds are most common in adolescent and young adult

males but relatively rare in other groups. Motor vehicle and auto-pedestrian collisions occur at all ages without gender predilection.

Classification of Head Trauma

The most widely used *clinical* classification of brain trauma, the Glasgow Coma Scale (GCS), depends on the assessment of three features: Best eye, verbal, and motor responses. Using the GCS, TBI can be designated as mild, moderate, or severe injury.

TBI can also be divided chronologically and *pathoetiologically* into primary and secondary injury, the system used in this text. **Primary injuries** occur at the time of initial trauma. Skull fractures, epi- and subdural hematomas, contusions, axonal injury, and brain lacerations are examples of primary injuries.

Secondary injuries occur later and include cerebral edema, perfusion alterations, brain herniations, and CSF leaks. Although vascular injury can be immediate (blunt impact) or secondary (vessel laceration from fractures, occlusion secondary to brain herniation), for purposes of discussion, it is included in the chapter on secondary injuries.

CLASSIFICATION OF HEAD TRAUMA

Primary Effects
- Scalp and skull injuries
- Extraaxial hemorrhage/hematomas
- Parenchymal injuries
- Miscellaneous injuries

Secondary Effects
- Herniation syndromes
- Cerebral edema
- Cerebral ischemia
- Vascular injury (can be primary or secondary)

Imaging Acute Head Trauma

Imaging is absolutely critical to the diagnosis and management of the patient with acute traumatic brain injury. The goal of emergent neuroimaging is twofold: (1) identify treatable injuries, especially emergent ones, and (2) detect and delineate the presence of secondary injuries such as herniation syndromes and vascular injury.

How to Image?

A broad spectrum of imaging modalities can be used to evaluate patients with TBI. These range from outdated, generally ineffective techniques (i.e., skull radiographs) to very sensitive but expensive studies (e.g., MR). Techniques that are still relatively new include CT and MR perfusion, diffusion tensor imaging (DTI), and functional MRI (fMRI).

Skull Radiography

For decades, skull radiography (whether called "plain film" or, more recently, "digital radiography") was the only noninvasive imaging technique available for the assessment of head injury.

Skull radiography is reasonably effective in identifying calvarial fractures. Yet skull x-rays cannot depict the far more important presence of extraaxial hemorrhages and parenchymal injuries.

Between one-quarter and one-third of autopsied patients with fatal brain injuries have no identifiable skull fracture! Therefore, skull radiography obtained solely for the purpose of identifying the presence of a skull fracture has no appropriate role in the current management of the head-injured patient. With rare exceptions, it's the brain that matters—not the skull!

NECT

CT is now accepted as the worldwide screening tool for imaging acute head trauma. Since its introduction almost 40 years ago, CT has gradually but completely replaced skull radiographs as the "workhorse" of brain trauma imaging. The reasons are simple: CT depicts both bone and soft tissue injuries. It is also widely accessible, fast, effective, and comparatively inexpensive.

Nonenhanced CT (NECT) scans (four or five millimeters thick) from just below the foramen magnum through the vertex should be performed. Two sets of images should be obtained, one using brain and one with bone reconstruction algorithms. Viewing the brain images with a wider window width (150-200 HU, the so-called subdural window) should be performed on PACS (or film, if PACS is not available). The scout view should always be displayed as part of the study (see below).

Because delayed development or enlargement of both extra- and intracranial hemorrhages may occur within 24-36 hours following the initial traumatic event, repeat CT should be obtained if there is sudden unexplained clinical deterioration, regardless of initial imaging findings.

Multidetector Row CT and CT Angiography

Because almost one-third of patients with moderate to severe head trauma also have cervical spine injuries, multidetector row CT (MDCT) with both brain and cervical imaging is often performed. Soft tissue and bone algorithm reconstructions with multiplanar reformatted images of the cervical spine should be obtained.

CT angiography (CTA) is often obtained as part of a whole-body trauma CT protocol. Craniocervical CTA should also specifically be considered (1) in the setting of penetrating neck injury, (2) if a fractured foramen transversarium or facet subluxation is identified on cervical spine CT, or (3) if a skull base fracture traverses the carotid canal or a dural venous sinus. Arterial laceration or dissection, traumatic pseudoaneurysm, carotid-cavernous fistula, or dural venous sinus injury are nicely depicted on high-resolution CTA.

MR

There is general agreement that NECT is the procedure of choice in the initial evaluation of brain trauma. With one important exception—suspected child abuse—using MR as a routine screening procedure in the setting of *acute* brain trauma is uncommon. Standard MR together with new techniques such as diffusion tensor imaging is most useful in the subacute and chronic stages of TBI. Other modalities such as fMRI are playing an increasingly important role in detecting subtle abnormalities, especially in patients with mild cognitive deficits following minor TBI.

Who and When to Image?

Who to image and when to do it is paradoxically both well-established and controversial. Patients with a GCS score indicating moderate (GCS = 9-12) or severe (GCS ≤ 8) neurologic impairment are invariably imaged. The real debate is about how best to manage patients with GCS scores of 13-15.

GLASGOW COMA SCALE

Best eye response (maximum = 4)
- 1 = no eye opening
- 2 = eye opening to pain
- 3 = eyes open to verbal command
- 4 = eyes open spontaneously

Best verbal response (maximum = 5)
- 1 = none
- 2 = incomprehensible sounds
- 3 = inappropriate words
- 4 = confused
- 5 = oriented

Best motor response (maximum = 6)
- 1 = none
- 2 = extension to pain
- 3 = flexion to pain
- 4 = withdrawal to pain
- 5 = localizing to pain
- 6 = obedience to commands

Sum = "coma score" and clinical grading
- 13-15 = mild brain injury
- 9-12 = moderate brain injury
- ≤ 8 = severe brain injury

In an attempt to reduce CT overutilization in emergency departments, several organizations have developed clinical criteria that help separate "high-risk" from "low-risk" patients. (Several of these are delineated in the boxes below.) Yet the impact on the emergency department physician ordering behavior has been inconsistent. In places with high malpractice rates, many emergency physicians routinely order NECT scans on every patient with head trauma regardless of GCS score or clinical findings.

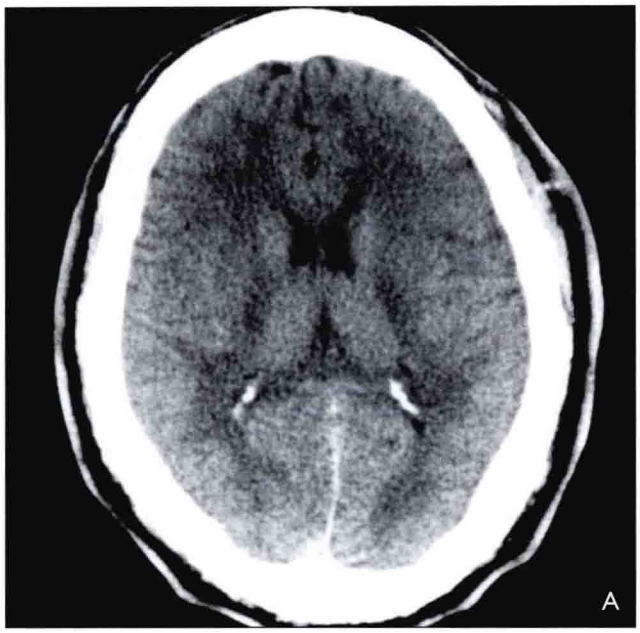
Whether—and when—to obtain follow-up imaging in trauma patients is also controversial. In a large study of children with GCS scores of 14 or 15 and a normal initial CT scan, only 2% had follow-up CT or MR performed. Of these, only 0.05% had abnormal results on the follow-up study, and *none* required surgical intervention. The negative predictive value for neurosurgical intervention for a child with an initial GCS of 14 or 15 and normal CT was 100%. From this, the authors concluded that children with a GCS of 14 or 15 and a normal initial head CT are at very low risk for subsequent traumatic findings on neuroimaging and extremely low risk of needing neurosurgical intervention. Hospitalization of children with minor head trauma after normal CT scan results for neurologic observation was deemed unnecessary.

Appropriateness Criteria

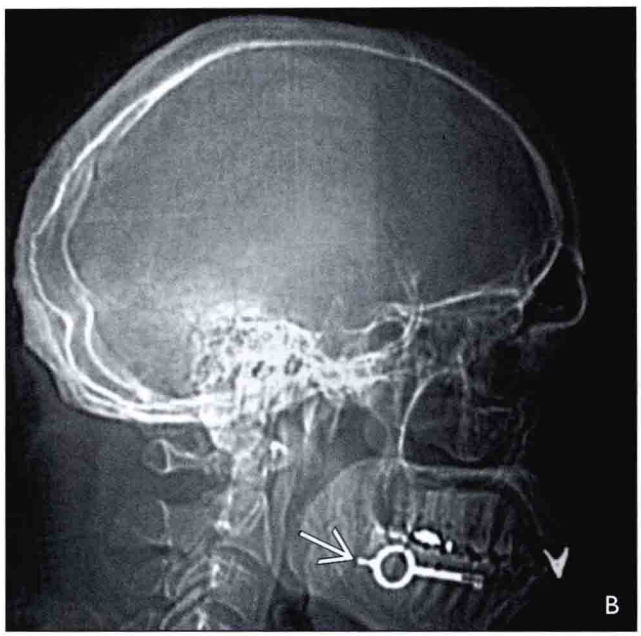
Three major and widely used Appropriateness Criteria for Imaging Acute Head Trauma have been published: The American College of Radiology (ACR) Appropriateness Criteria, the New Orleans Criteria (NOC), and the Canadian Head CT Rule (CHCR).

ACR Criteria. Emergent NECT in mild/minor CHI with the presence of a focal neurologic deficit and/or other risk factors is deemed "very appropriate," as is imaging all traumatized children under 2 years of age. While acknowledging that NECT in patients with mild/minor CHI (GCS ≥ 13) without risk factors or focal neurologic deficit is "known to be low yield," the ACR still rates it as 7 out of 9 in appropriateness.

NOC and CHCR. Both the New Orleans Criteria and Canadian Head CT Rule attempt to triage patients with minimal/mild head injuries in a cost-effective manner. A GCS score of 15 (i.e., normal) without any of the NOC indicators is a highly sensitive negative predictor of clinically important brain injury or need for surgical intervention.



1-1A. *Axial NECT scan of a prisoner imaged for head trauma shows no gross abnormality.*



1-1B. *Scout view in the same case shows a foreign object ➡ (a handcuff key!) in the prisoner's mouth. He faked the injury and was planning to escape, but the radiologist alerted the guards and thwarted the plan. (Courtesy J. A. Junker, MD.)*

NEW ORLEANS CRITERIA IN MINOR HEAD INJURY

CT indicated if GCS = 15 plus any of the following

- Headache
- Vomiting
- Patient > 60 years old
- Intoxication (drugs, alcohol)
- Short-term memory deficits (anterograde amnesia)
- Visible trauma above clavicles
- Seizure

Adapted from Stiell IG et al: Comparison of the Canadian CT head rule and the New Orleans criteria in patients with minor head injury. JAMA 294(12):1511-1518, 2005

According to the CHCR, patients with a GCS score of 13-15 and witnessed loss of consciousness (LOC), amnesia, or confusion are imaged, along with those deemed "high risk" for neurosurgical intervention or "medium risk" for brain injury.

Between 6-7% of patients with minor head injury have positive findings on head CT scans. Most also have headache, vomiting, drug or alcohol intoxication, seizure, short-term memory deficits, or physical evidence of trauma above the clavicles. CT should be used liberally in these cases as well as in patients over 60 years of age and in children under the age of two.

CANADIAN HEAD CT RULE IN MINOR HEAD INJURY

CT if GCS = 13-15 and witnessed LOC, amnesia, or confusion

High risk for neurosurgical intervention

- GCS < 15 at 2 hours
- Suspected open/depressed skull fracture
- Clinical signs of skull base fracture
- ≥ 2 vomiting episodes
- Age ≥ 65 years

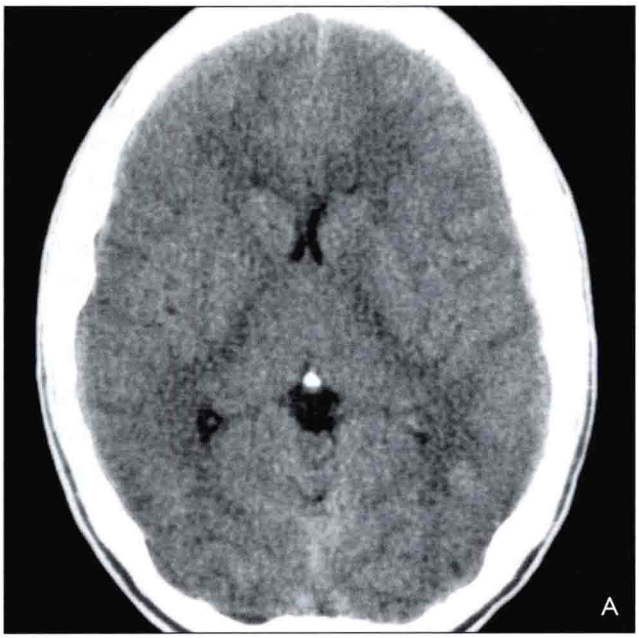
Medium risk for brain injury detected by head CT

- Antegrade amnesia ≥ 30 minutes
- "Dangerous mechanism" (i.e., auto-pedestrian, ejection from vehicle, etc.)

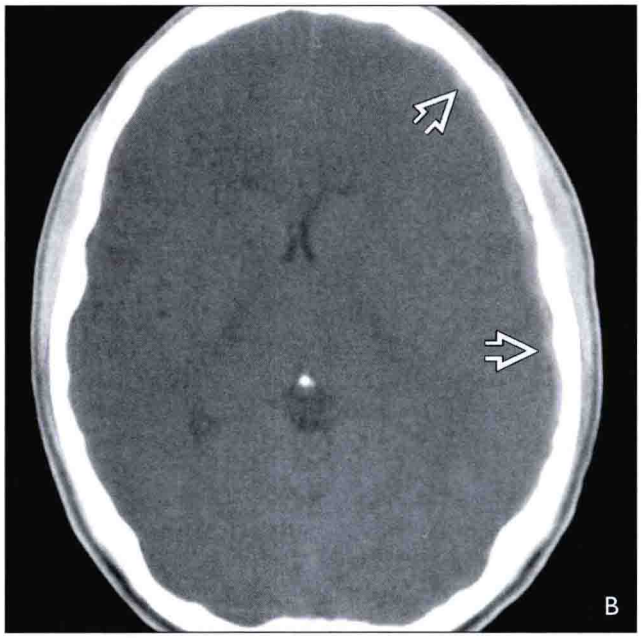
Adapted from Stiell IG et al: Comparison of the Canadian CT head rule and the New Orleans criteria in patients with minor head injury. JAMA 294(12):1511-1518, 2005

Trauma Imaging: Keys to Analysis

Four components are essential to the accurate interpretation of CT scans in patients with head injury: The scout image plus brain, bone, and subdural views of the NECT dataset. Critical information may be present on just one of these four components.



1-2A. *NECT scan at standard brain windows (80 HU) shows no definite abnormality.*



1-2B. *Intermediate window width (175 HU) shows a small left subdural hematoma. Thin subdural hematomas may be visible only with wider window widths.*

Suggestions on how to analyze NECT images in patients with acute head injury are delineated below.

Scout Image

Before you look at the NECT scan, examine the digital scout image! Look for cervical spine abnormalities such as fractures or dislocations, jaw and/or facial trauma, and the presence of foreign objects **(1-1)**. If there is a suggestion of cervical spine fracture or malalignment, MDCT of the cervical spine should be performed before the patient is removed from the scanner.

Brain Windows

Methodically and meticulously work your way from the outside in. First evaluate the soft tissue images, beginning with the scalp. Look for scalp swelling, which usually indicates the impact point. Carefully examine the periorbital soft tissues.

Next look for extraaxial blood. The most common extraaxial hemorrhage is traumatic subarachnoid hemorrhage (tSAH), followed by sub- and epidural hematomas. The prevalence of traumatic SAH in moderate to severe TBI approaches 100%. tSAH is usually found in the sulci adjacent to cortical contusions, along the sylvian fissures, and around the anteroinferior frontal and temporal lobes. The best place to look for subtle tSAH is the interpeduncular cistern, where blood collects when the patient is supine.

Any hypodensity within an extraaxial collection should raise suspicion of rapid hemorrhage with accumulation of unclotted blood or (especially in alcoholics or older patients) an underlying coagulopathy. This is an urgent finding that mandates immediate notification of the responsible clinician.

Look for intracranial air ("pneumocephalus"). Intracranial air is always abnormal and indicates the presence of a fracture that traverses either the paranasal sinuses or mastoid.

Now move on to the brain itself. Carefully examine the cortex, especially the "high-yield" areas for cortical contusions (anteroinferior frontal and temporal lobes). If there is a scalp hematoma due to impact (a "coup" injury), look 180° in the opposite direction for a classic "contre-coup" injury. Hypodense areas around the hyperdense hemorrhagic foci indicate early edema and severe contusion.

Move inward from the cortex to the subcortical white and deep gray matter. Petechial hemorrhages often accompany axonal injury. If you see subcortical hemorrhages on the initial NECT scan, this is merely the "tip of the iceberg." There is usually a *lot* more damage than what is apparent on the first scan. A general rule: The deeper the lesion, the more severe the injury.

Finally, look inside the ventricles for blood-CSF levels and hemorrhage due to choroid plexus shearing injury.

Subdural Windows

Look at the soft tissue image with both narrow ("brain") and intermediate ("subdural") windows **(1-2)**. Small sub-