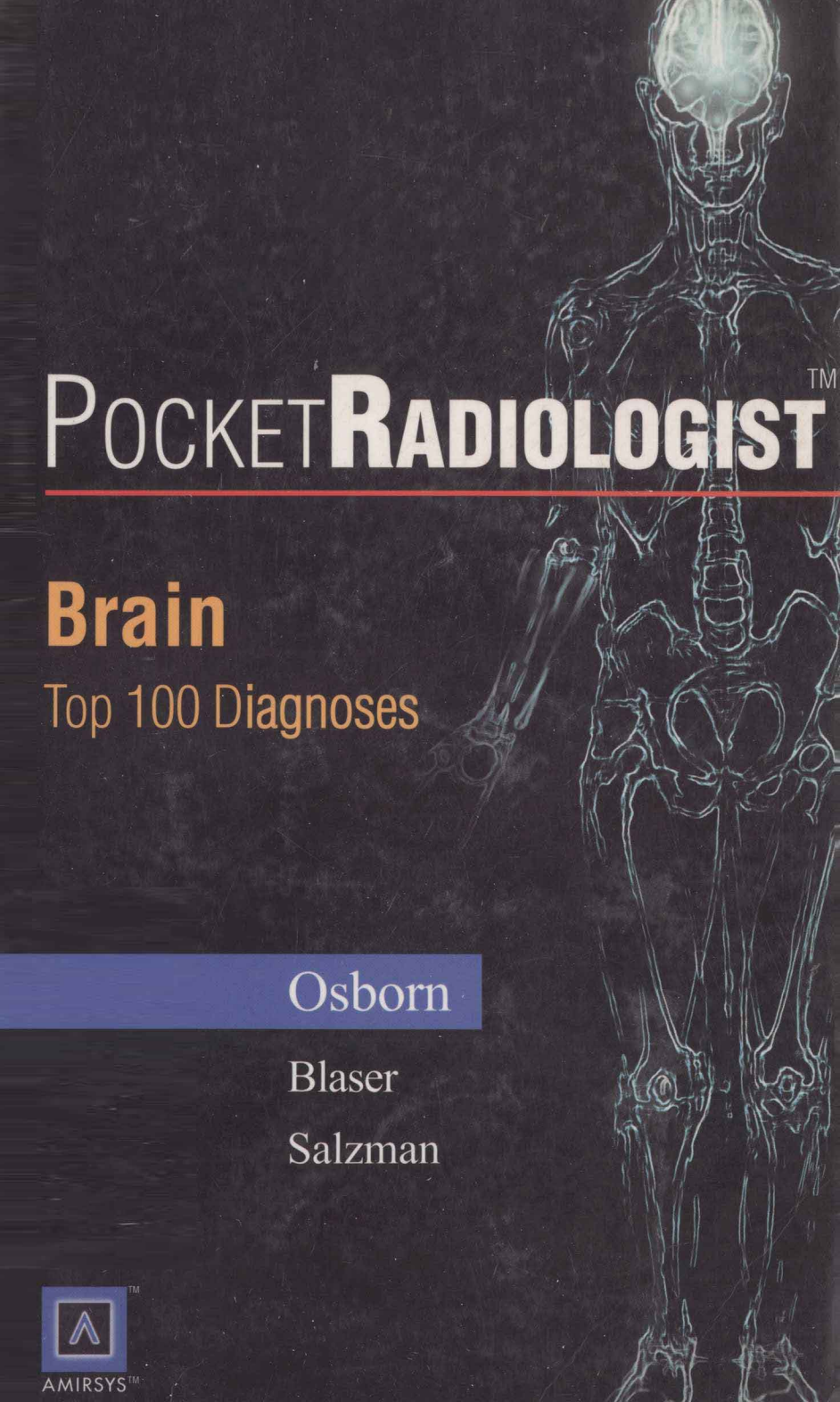

POCKET RADIOLOGIST™

Brain

Top 100 Diagnoses

Osborn

Blaser

Salzman

AMIRSYS™

PocketRadiologist™
Brain
100 Top Diagnoses

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Preface

The **PocketRadiologist™** series is an innovative, quick reference designed to deliver succinct, up-to-date information to practicing professionals "at the point of service." As close as your pocket, world-renowned authors write each title in the series. These experts have designated the "top 100" diagnoses or interventional procedures in every major body area, bulleted the most essential facts, and offered high-resolution imaging to illustrate each topic. Selected references are included for further review. Full color anatomic-pathologic computer graphics model many of the actual diseases.

Each **PocketRadiologist™** title follows an identical format. The same information is in the same place - every time - and takes you quickly from key facts to imaging findings, differential diagnosis, pathology, pathophysiology, and relevant clinical information. The interventional modules give you the essentials and "how-tos" of important procedures, including pre- and post-procedure checklists, common problems and complications.

PocketRadiologist™ titles are available in both print and hand-held PDA formats. Currently available modules feature Brain, Head and Neck, Orthopaedic (Musculoskeletal) Imaging, Pediatrics, Spine, Chest, Cardiac, Vascular, Abdominal Imaging and Interventional Radiology. 2003 topics that will round out the PocketRadiologist™ series include Obstetrics, Gynecologic Imaging, Breast, Temporal Bone, Pediatric Neuroradiology and Emergency Imaging.

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PocketRadiologist™
Brain
Top 100 Diagnoses

The diagnoses in this book are divided into 11 sections in the following order:

Trauma
Infection
Aneurysms
Vascular Malformations
Stroke and Vascular Disease
Neoplasms
Cysts
Meninges
Ventricles and Cisterns
Metabolic White Matter, Degenerative Disease
Congenital Disorders

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TRAUMA

Table of Diagnoses

Aneurysms

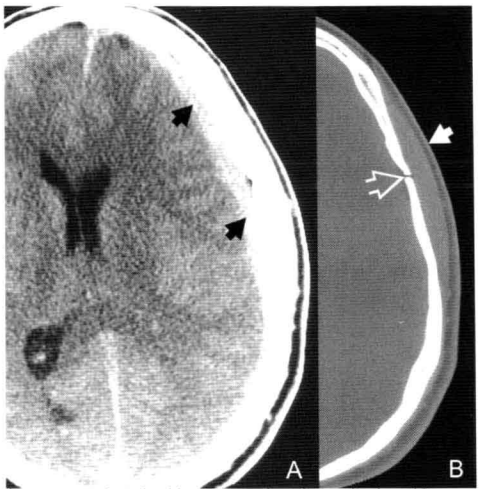
Vascular Malformations

Stroke and Vascular Disease

Neoplasms

Congenital

Skull Fracture (Fx)



NECT scan shows acute SDH (A, black arrows) with nondisplaced linear skull fracture (B, open arrow), overlying soft tissue swelling (white arrow).

Key Facts

- Trauma = #1 cause of death/disability in young
- Skull films ineffective as trauma screen
- 1/3 of patients with severe brain injury don't have fracture (fx)
- Fxs can be linear, depressed, diastatic
- Skull base fxs can damage vessels, dura, involve cranial nerves
- Sequelae include pneumocephalus, CSF leak

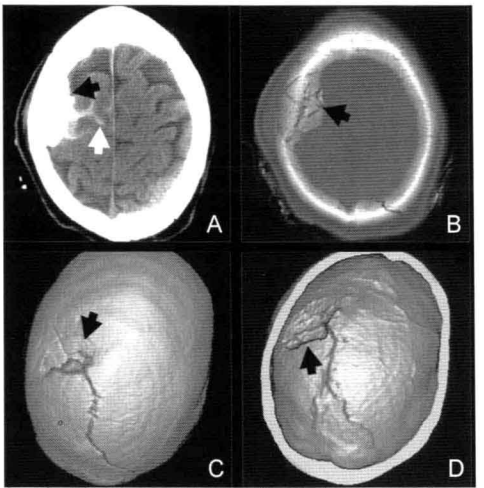
Imaging Findings

General Features

- Linear fx
  - Acute fx = sharply delineated lucent line
  - Overlying soft tissue swelling almost always present
- Depressed fx
  - Fragment(s) displaced inwards
- Diastatic fx
  - Sutures spread
  - +/- Co-existing linear fx
  - May tear venous sinus, cause venous EDH
  - Cranial "burst fracture"
    - Unique in infants
    - Wide diastasis (>4mm)
    - Brain herniates through fx, extrudes under scalp
- Skull base fx
  - Can be longitudinal, transverse
  - 50% have associated intracranial mass lesion

Imaging Recommendations

- Skip the skull films
- Get NECT scan
  - Patients with high-risk mild head injury
    - GCS = 13, 14



NECT scan (A, B) with 3-D reconstruction (C, D) show a comminuted, depressed anterior parietal skull fracture (black arrows). Note traumatic SAH (A, white arrow).

    - 10% of patients with GCS = 15 + loss of consciousness or amnesia have abnormalities on CT scan
    - 5% of patients with GCS = 15 + normal neurological exam have significant intracranial injury revealed on CT
- Use both bone, soft-tissue algorithms
- View/photograph using 3 window settings
    - Soft tissue (level = 40H, window-75-100H)
    - Bone (level = 500H, window = 3000H)
    - Intermediate (level = 75H, window = 150-200H) to show small SDHs
- Evaluate for vascular injury if carotid canal involved

Differential Diagnosis

Suture Line
- Acute fx lucent, has sharp noncorticated margins
- Suture less distinct, has dense sclerotic borders

Vascular Groove
- Corticated margins
- Typical location (i.e., MMA)

Venous Lake
- Corticated margins
- Typical location (i.e., parasagittal)

Arachnoid Granulation
- Corticated margins
- Typical location (parasagittal, transverse sinus)

Pathology

General
- Etiology-Pathogenesis
    - Direct blow to skull

- Epidemiology
  - Fx present in majority of severe head injury cases
  - 25%-35% of severely injured patients do not have skull fx
  - Skull fx absent in 25% of fatal injuries at autopsy!

Gross Pathologic or Surgical Features

- Types: Linear, depressed, diastatic
- Associated injuries
  - Linear fx: Extra-axial hematoma
  - Depressed fx: Lacerated dura, arachnoid; parenchymal injury
  - Skull base fx: Cranial nerve injury, CSF leak, epistaxis, periorbital bruising
  - 10%-15% of patients with severe head trauma (GCS 3-6) have C1 or C2 fracture

Clinical Issues

Presentation

- Varies with underlying injury

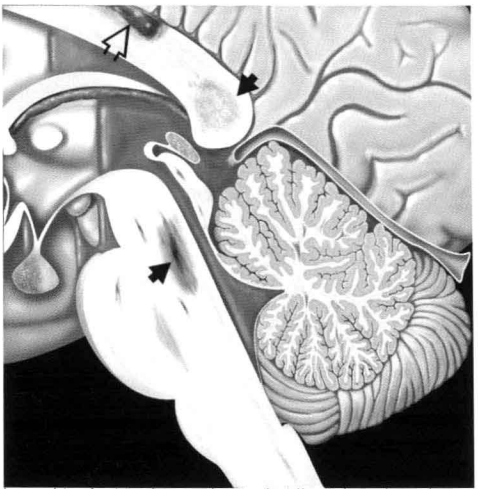
Natural History

- Varies with underlying injury
- Patients who return to ED have a remarkable incidence of missed intracranial lesions, poor outcome

Selected References

1. Hofman PAM et al: Value of radiological diagnosis of skull fracture in the management of mild head injury. J Neurol Neurosurg Psychiatr 68: 416-22, 2000
2. Vilks GM et al: Use of a complete neurological examination to screen for significant intracranial abnormalities in minor head injury. Am J Emerg Med 18: 159-63, 2000
3. Kleinman PK et al: Soft tissue swelling and acute skull fractures. J Pediatr 121: 737-9, 1992

Axonal Injury



Mid-sagittal graphic depicts hemorrhagic (midbrain) and nonhemorrhagic (CC splenium) axonal injury (black arrows), traumatic IVH, SAH, and dorsolateral CC contusion (open arrow).

Key Facts
- Diffuse axonal injury (DAI) = second most common traumatic brain injury (TBI)
- **Not** caused by mechanical "shearing"
- Impaired axoplasmic transport, axonal swelling, disconnection
- Most DAI lesions are microscopic, nonhemorrhagic
- MR >> CT for detection

Imaging Findings

General Features
- Multiple lesions in nearly all cases
- Best diagnostic sign = hemorrhages at gray/white matter, corpus callosum, fornix, upper brainstem, basal ganglia, internal capsule

CT Findings
- May be normal, especially with mild TBI
- 20%-50% demonstrate petechial hemorrhage(s)
- 10%-20% evolve to focal mass lesion

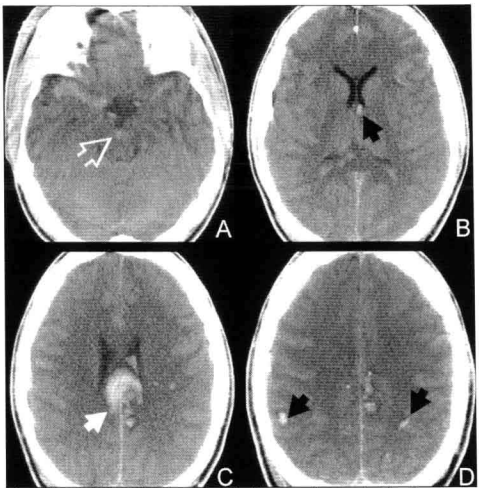
MR Findings
- Multiple high-signal foci on T2WI, FLAIR
- Low signal on T2* sequences

Other Modality Findings
- Decreased ADC
- MRS: Decreased NAA/Cr correlates with outcome
- MSI: Abnormal low-frequency magnetic activity

Imaging Recommendations
- Follow-up at 24h (1/6 evolve)

Differential Diagnosis

Nonhemorrhagic DAI
- Multifocal white matter hyperintensities on T2WI
  - Demyelinating disease (ovoid, may enhance)



NECT scans show DAI with multiple petechial hemorrhages in the fornix and subcortical WM (black arrows), dorsolateral CC contusion (white arrow), and tSAH (open arrow).

- Small vessel disease, lacunar infarcts (older patients)
- Metastases (enhance)

Hemorrhagic DAI

- Multiple "black dots" on T2-, T2*
  - Hypertensive microhemorrhages (longstanding chronic HTN)
  - Amyloid angiopathy (elderly, normotensive, often demented)
  - Cavernous/capillary malformations (mixed hemorrhages)

Pathology

General

- Etiology-Pathogenesis
  - Inertial forces in nonimpact injuries
  - Rapid head rotation
  - Differential acceleration/deceleration
  - Axons rarely disconnected or "sheared" (only in most severe injury)
  - Non-disruptively injured axons undergo
    - Traumatic depolarization
    - Massive ion fluxes, spreading depression
    - Excitatory amino acid release
    - Metabolic alterations
    - Cellular swelling
    - Accelerated glycolysis, lactate accumulation
  - Result
    - Secondary "axotomy"
    - Impaired axoplasmic transport
    - Disconnection
    - Wallerian degeneration, diffuse deafferentation
- Epidemiology
  - Second most common lesion in CHI
  - Occurs in slightly less than 50% of cases