CHAPTER 4 ■ CERE

HOWARD A. ROWLEY

Stroke is a clinical term applied to any abr
brain insult—literally "a blow from an unse
are caused by either brain infarction (75%
(25%), and must be distinguished from
causing abrupt neurologic deficits. *Infarcti*
injury which occurs when tissue perfusion
enough to cause necrosis, typically due to
feeding artery. *Transient ischemic attacks*
cally defined as transient neurologic sympto
less than 24 hours, which may serve as a "w
infarction occurring in the next few weeks
are often due to temporary occlusion of a fe
though signs and symptoms may be transien
considered to have had a stroke if imaging
lesion. *Hemorrhage* is seen when blood rup
arterial wall, spilling into the surrounding
arachnoid space, or ventricles.

Stroke is the third leading cause of dea
States and major source of long-term disabi
vors. The approach to treatment of ischemi
largely preventative or supportive in the pa
of intravenous (IV) thrombolysis for acute s
outcome benefits of endovascular devices
imaging and intervention a critical part of str
The patient with hemorrhage may harbor a
cular malformation, or other condition, eac
ant differences in treatment options. The ra
critical role in the triage and evaluation of a
Selection of the proper imaging protocol, re
ischemic changes, differentiation of stroke
disorders, and recognition of important str
have a significant impact on therapy and out

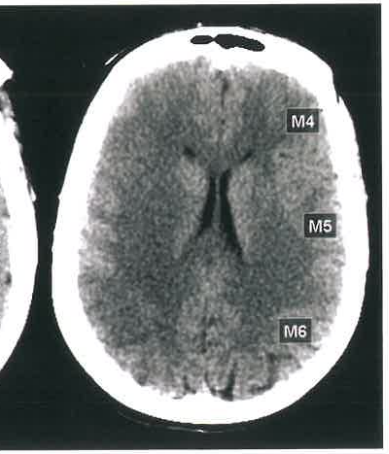
This chapter reviews the pathophysiolo
time course of findings on computed tomo
netic resonance imaging, patterns of arte
occlusions, and overall radiologic approach
the stroke patient.

AGE

	ELDERLY
	Atherosclerosis
	Cardiac emboli
	Coagulopathy
	Amyloid
	Vasculitis
	Venous thrombosis

ter. This leads to cellular ("cytotoxic") edema, ically as increased water content in the affected *ges in brain water are key to understanding signs by CT and MR.* Even a small increase in water es characteristic decreased attenuation on CT, T1-weighted MR, and high signal on T2- and ghted MR. Cytotoxic edema peaks 3 to 7 days n and is maximum in the gray matter. A smaller f vasogenic edema also develops as the more llary endothelial cells lose integrity. (In contrast, ted edema is primarily vasogenic and preferen- he white matter—see Chapter 5.)

spection of CT and MR images done within few hours after vessel occlusion can give clues to y, even before gross tissue edema or mass effect se "hyperacute" signs primarily relate to morpho- in the vessels or physiology of perfusion rather or signal changes in the parenchyma. On CT, the ous may occasionally be seen in larger intracranial ulting in the "hyperdense artery sign" (Fig. 4.1). normal black signal of flowing blood within the



ion, high density indicative of thrombus is seen in the eady present. The 10 regions scored by ASPECTS are ow attenuation in the right insula, posterior lentiform CA territory and ASPECTS is much lower than 7, both e: www.aspectsinstroke.com.

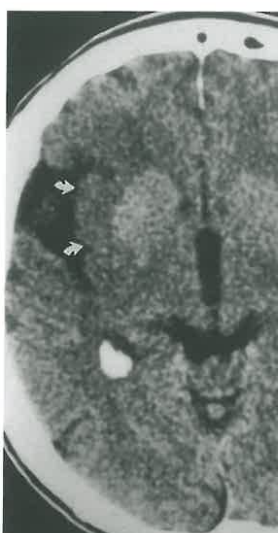


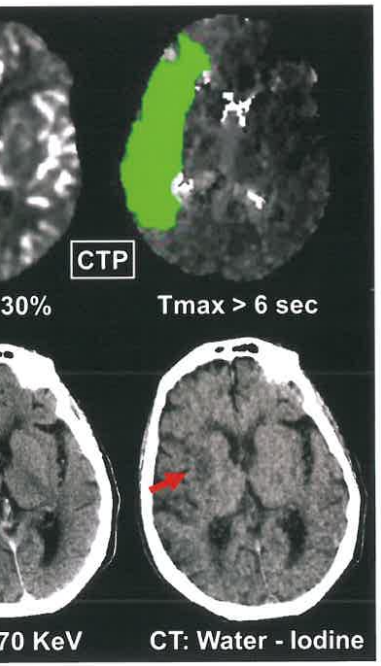
FIGURE 4.2. **Insular Ribbon Sign. A:** A
and loss of gray-white borders in the ri
The insular cortex, claustrum, and extr
ticulostriate vessels. (From Truwit CL, E
early CT sign of acute middle cerebral a

lumen ("flow void") is immediately lost an
by abnormal signal representing clot or slow

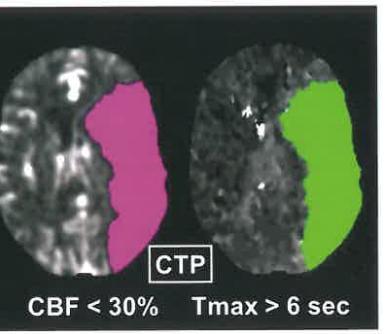
***Acute MCA Ischemia on CT: Insular Ri
form Nucleus Edema.*** CT scans done with
dle cerebral artery (MCA) occlusion will con
"insular ribbon sign," a subtle but importa
gray-white layers of the insula due to early
Early edema may also be most conspicuou
in proximal MCA occlusions (lentiform nu
MR examinations in the first few hours m
loss of gray-white borders and slight crowdi
destined to undergo infarction. However,
imaging sequence for detection of brain isch
weighted MR imaging, which may turn pos
infarction begins, well before the CT shows
Hyperintense signal on diffusion-weighted
("light-bulb sign") precedes T2 hyperintensi
develops at 6 to 12 hours post ictus (Fig. 4.

Imaging Triage for Emerg
Stroke Intervention

Careful but rapid interpretation of CT sca
important in patients who are candidates
lytic drug treatment (e.g., tissue plasminogen
intra-arterial (IA) endovascular intervention,
ment. IV t-PA is front line therapy for patient
of symptom onset, and patients with favora
also be selected for endovascular thrombecto
24 hours, sometimes using a bridging app
lowed by IA therapy. The screening CT is exa
patients with brain hemorrhage, masses, o



78-year-old woman developed abrupt left hemiplegia rokes, but her usual oral anticoagulants were withheld of internal capsule (*arrow*), with ASPECTS = 8. CTA processed with RAPID software (iSchemaView) shows n significantly prolonged Tmax transit times = 92 mL the favorable ASPECTS, a small "core" infarct, large At angiography the M1 occlusion was confirmed (AP Postprocedure dual-energy CT using low KeV shows retention ("stain") in the ischemic core (*arrow*), with nsity subtracted.



. This 62-year-old woman was transferred with stroke on (*arrows*; ASPECTS = 1), a small old right parietal A occlusion (*arrow*) with poor distal collateral filling. rct, with low CBF core = 135 mL (*shaded pink*) and technically accessible, this is a poor candidate for inter- ge volume of low CBF), matching severe Tmax region

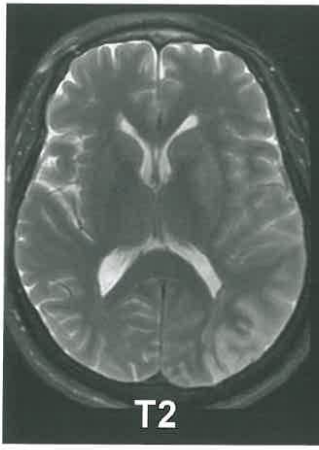


FIGURE 4.5. **Edema in Stroke of Unknown** Edema is detected as high signal intensity an there is a loss of flow voids in the left sylvian of cytotoxic edema in acute ischemia. These show that if T2/FLAIR is negative, with only candidate for IV TPA (based on the "tissue cl

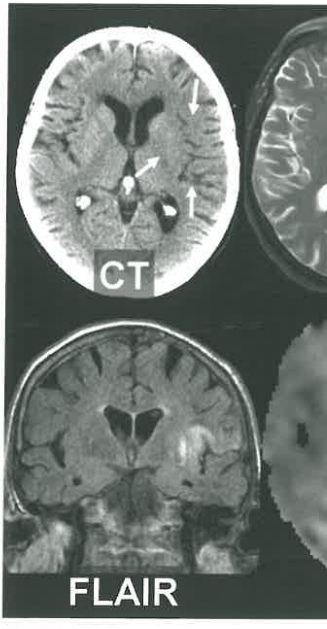
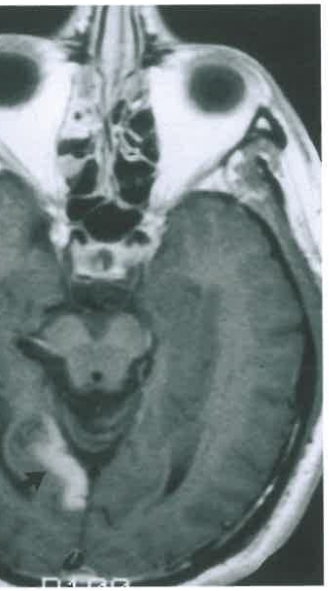


FIGURE 4.6. **Diffusion–Perfusion Mismatch** sudden right hemiplegia and aphasia. A: The tex (*arrows*). On T2WI, the cortical gray matt shows cortical edema and stasis in the left MC show a larger area at risk extending into the p hypoperfused tissue not yet infarcted is conside surrounding perfusion lesions if untreated. Foll



lema and mass effect subside, but before devel-
s. A: T2WI is essentially normal in the occipital
nium shows enhancement of the infarcted deep

ks and months following infarction, macrophages
tissue, leaving a small amount of gliotic scar and
lacia behind. CSF takes up the space previously
brain. The affected corticospinal tract atrophies
generation) leading to a shrunken appearance of
cerebral peduncle. If hemorrhage accompanied
emosiderin may be seen grossly or detected as sig-
nsity by T2-weighted images (T2WI). Widening
ulci and "ex-vacuo" dilatation of the ventricle
nt to the infarcted area (Fig. 4.8).

hagic Transformation of Infarction

into infarcted capillary beds may secondarily
or microscopic hemorrhage, seen in up to half
n most cases this takes the form of microscopic
edesis) of red blood cells, but on rare occasions a
ma will form. Physical disruption of the capillary
lls, loss of vascular autoregulation, and anticoag-
rombolytic use may all contribute to the develop-
hemorrhages. Patients may develop headaches at
leeding, but commonly have no new symptoms,
ecause the hemorrhage occurs within brain areas
dy dead or dysfunctional. Hemorrhagic infarction
the territory of the infarcted vessel, whereas pri-
hage does not necessarily respect vascular bound-
entricular extension is uncommonly seen with
transformation and should raise the possibility of
ess (such as hypertensive bleed or a ruptured arte-
lformation [AVM]).
time for hemorrhagic transformation is at
weeks post infarction. It is usually manifest as

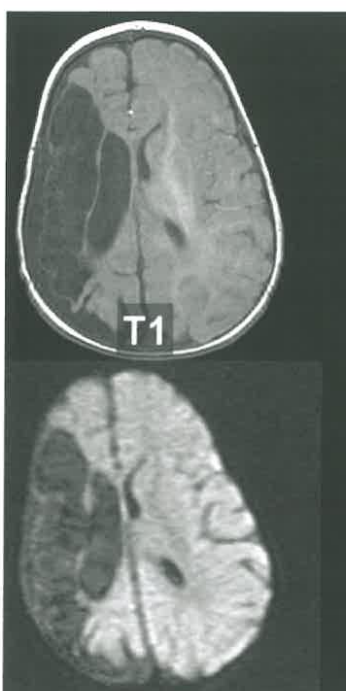
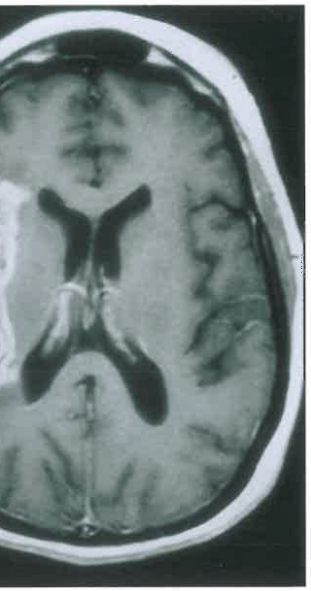


FIGURE 4.8. **Chronic Infarction.** Cystic encepha
tion. Note cystic changes approach CSF on all se
the ipsilateral ventricle (ex-vacuo dilatation).

a serpiginous line of petechial blood follow
tours of the infarcted cortex. These dots o
often patchy and discontinuous. On CT a
attenuation is observed, and on MR bright s
the affected gyrus on the unenhanced T1W
hemoglobin (Fig. 4.9). (Alternate explanati
signal have been offered, including laminar
cation related to infarction; the practical po
this appearance as a feature of ischemia.) T
pattern is not seen in primary brain hemor
helpful in confirming the underlying ische
suspicious lesion. This is considered a norm
lution of an infarct. Management in the pre
hemorrhage is controversial, but many neu
anticoagulation if there is a well-documente

More extensive hemorrhagic transfo
infarcted tissue may lead to the formation
chymal hematoma. Here, the blood does
gyrus and may form a clot indistinguishabl
hematoma. Large cortical infarcts are at
risk for this type of change, compared with
subcortical lesions. Catastrophic hemorrhag
can also follow thrombolysis, particularly
delayed or the baseline CT shows extensive e
to the petechial gyral transformation descr
parenchymal hematomas tend to occur ear
commonly associated with clinical deterio



farction. A: Precontrast T1WI shows mild
intensity scattered along the cortex indicate
/I demonstrates marked gyral enhancement,

information not readily available from the non-
Stasis of gadolinium within vessels or leakage
nrough an abnormal blood–brain barrier will
laxation of adjacent protons, leading to hyper-
ancement) on T1WI. As with CT, a noncontrast
is mandatory before contrast is given since
and subacute blood both appear hyperintense
his will be discussed in the "Hemorrhage" sec-
bolus of contrast may also be captured dynam-
apid imaging techniques to produce a family of
ghted images to help identify ischemic regions.
lar enhancement on MR is commonly seen
ed territory during the 1st week. This may be
low or vasodilatation leading to stasis of gad-
y in both arteries and veins. The intravascular
pattern may be detected within minutes of ves-
is seen in a majority of cortical infarcts at 1 to
solves by 10 days. The proximal trunks of more
ded arteries and leptomeningeal cortical chan-
prominently involved (Fig. 4.10). The area of
ancement may extend beyond the T2 hyperin-
ly indicating recruitment of collateral supply at
border. Meningeal enhancement which attends
nd dural enhancement seen postoperatively can
resemble intravascular enhancement, but the
ould be obvious on clinical grounds. MR intra-
ancement helps identify early strokes, indicates
flow, and has no obvious CT counterpart.
chymal enhancement occurs in a similar pattern
on CT (and with the same time course seen by
cine infarct scans of the past). It may occur as
, but more typically begins after the 1st week, a
ravascular enhancement is waning. Reperfusion

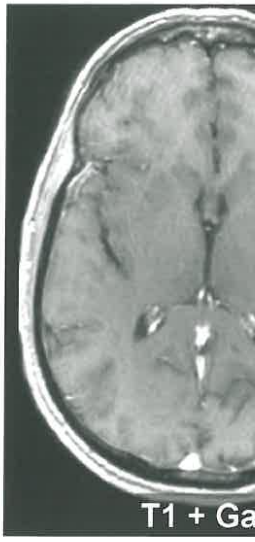


FIGURE 4.10. Intravascular Stasis left MCA infarction. Mild sulcal effa evident on T1. As seen here, FLAIR lar enhancement is typically seen onl

after thrombolysis can lead to early enhance cortical infarcts enhance by MR at 2 week marized this in his Rule of 3s: MR parench peaks at 3 days to 3 weeks and resolves by

The imaging time course for CT and M brain infarction are summarized in Table 4.

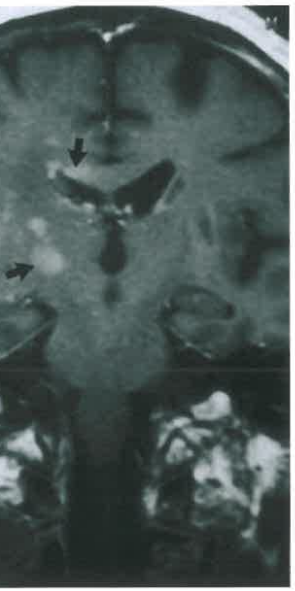
Pattern Recognition in Ischer

Familiarity with the major vascular territor guish between infarction and other patholo

TABLE 4.2

IMAGING TIME COURS

TIME	CT
Minutes	No ch
2–6 hours	Hyper Insula
6–12 hours	Sulcal +/– De
12–24 hours	Decre
3–7 days	Maxir
3–21 days	Gyral (pea
30–90 days	Encep Loss o Resol



shows edema primarily in the right MCA
us, and periatrial regions. **B:** Postcontrast
glia and periventricular regions (*arrows*).
bution and atypical enhancement pattern
udies will usually clarify the diagnosis.

emia, and available collateral supply. Complete
ions are occasionally found in asymptomatic
weil-developed collateral supply.
rotic disease near the carotid bifurcation is
the majority of ischemic events in the ICA ter-
l dissection, trauma, fibromuscular dysplasia,
nent, prior neck radiotherapy, and connective
may also cause significant carotid narrowing
emodynamic effects begin to be seen when there
ction in area or >60% decrease in diameter.
g less severe narrowing may nonetheless become
when they serve as a nidus for thrombus forma-
masked by hypotension. Studies have shown a
f endarterectomy in symptomatic patients with
s but not for those with <30% narrowing. In
carotid stents are now used in place of surgery,
high-risk patients.
ve screening of the carotid arteries may be
either US, MR angiography (MRA), or CT angi-
). The choice of modality depends on the abilities
e personnel and equipment. Sensitivity and spec-
gh as 85% to 90% for each of these techniques.
s noninvasively identify patients with hemody-
ificant disease who might then be referred for
ngiography or directly to intervention. US is the
ly employed screening examination in most cen-
advantage of portability, generally lower costs,
erformed in patients with contraindications to
is more operator dependent than MRA and is
ably assess portions of the distal ICA near the
A provides excellent visualization from the arch
l circulation, but at the small risk of contrast

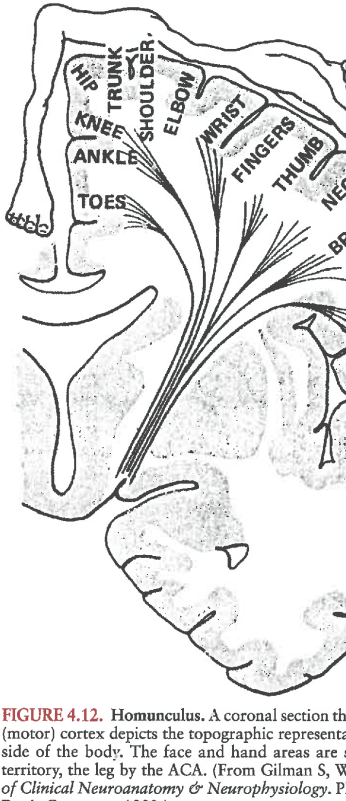


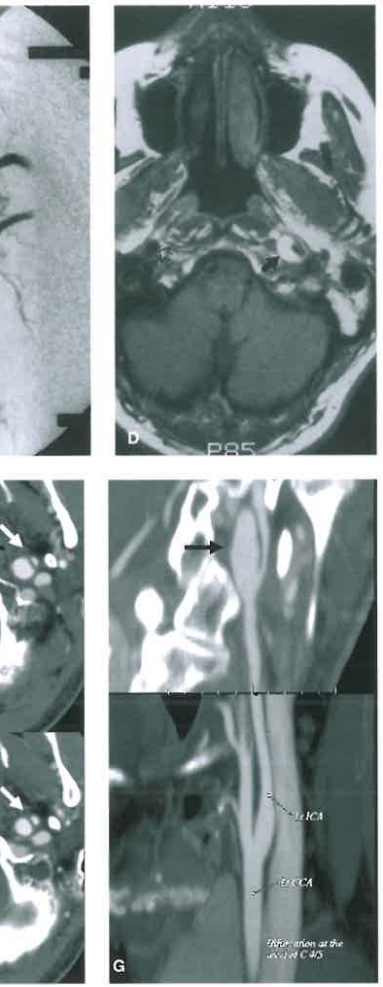
FIGURE 4.12. **Homunculus.** A coronal section th
(motor) cortex depicts the topographic representa
side of the body. The face and hand areas are
territory, the leg by the ACA. (From Gilman S, W
of Clinical Neuroanatomy & Neurophysiology. P
Davis Company; 1982.)

toxicity and radiation exposure. MRA can e
course of the carotid and may be quickly p
junction with the patient's brain MR stud
larly good method for screening pediatric c
in whom conventional angiography may be
difficult.

Selective common carotid angiography
standard for preprocedure carotid artery e
being replaced by noninvasive studies in m
study should cover the entire ICA, including
nial portions. Evaluation of the surgically in
segments (petrous, cavernous, and supracli
to exclude high-grade intracranial stenos
lesions which might contraindicate endartere

Anterior Cerebral Artery. The terminal bifu
is into the anterior and middle cerebral ar
The ACA is divided into three subgroups:
striates serve the rostral portions of the basa
losal branches supply the corpus callosum;
branches serve the medial aspects of the fro
lobes (Fig. 4.15). About 5% of infarcts invol

The medial lenticulostriates penetrate th
rating substance to give variable supply the
aspect of the internal capsule, putamen, glob
date head, and portions of the hypothalamus



by conventional digital subtraction angiography. The normal caliber above. CCA, common carotid artery; lerosis. Lateral maximum intensity projection from a imal narrowing (*arrow*). C: Carotid dissection with a patient shows the "mural crescent sign" indicative of er flow void and scant amounts of fat surrounding the tic shadowing (*arrows*), vessel narrowing, and spectral section with pseudoaneurysm. Source images (F) show oaneurysm. Thick slab 2D reconstructions (G) show a .

dy. Complete occlusion of the MCA beyond the es causes a combination of these deficits: con- e and arm hemiparesis, field defect, and either bal aphasia, depending on which hemisphere is weakness may also be seen when the MCA stem because of internal capsule involvement. These are summarized in Table 4.3.

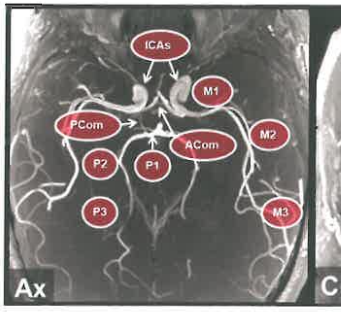


FIGURE 4.14. **MR Angiography of the Norma**
maximum intensity projections (MIPs) in three
Ax, Axial/submentovertex projection outlines th
the internal carotid arteries (ICA) and basilar
artery (ACom) are present, giving potential coll
anterior projection depicts the normal internal c
sequentially for distal branching order (e.g., M1,
basilar (B) which terminates in paired posterior c
inferior cerebellar artery; and PICA, posterior in
single right PCom extending posteriorly and the

Posterior (Vertebrobasilar) C

Vertebral Arteries. The vertebral arteries
from the subclavian arteries, ascend straig
transverse foramina of C6–C3, turn sharpl
C1-foramen magnum levels and unite ar

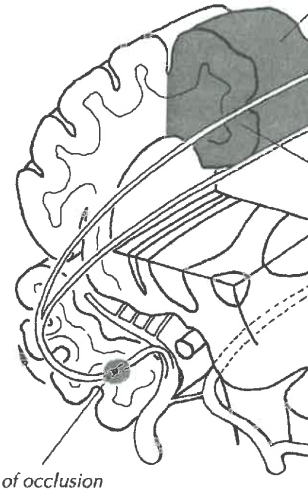


FIGURE 4.15. **Anterior Cerebral Artery Occlus**
sion causes infarction of the paramedian fronta
for motor and sensory function of the opposite l
bilateral, incontinence and akinetic mutism may
indicate direction of blood flow. (From Patten J.
ential Diagnosis. New York: Springer Verlag; 199

FICIT/SYNDROME
eakness
tinence, akinetic mutism
weakness
thria; ± motor aphasia
and arm > leg weakness
r aphasia (anterior lesion)
otive aphasia(posterior lesion)
l aphasia (total MCA)
ct syndromes
spatial dysfunction
ble lacunar syndromes
anopsia
cal blindness
ory deficits
olence
ry disturbances
a, vertigo, vomiting
if mass effect
instem deficits
in a barrel syndrome
e memory problems

r artery; SCA, superior cerebellar
posterior cerebral artery.

branches to the cerebellum and smaller perforat-
he brainstem. The basilar ends at its bifurcation
rior cerebral arteries just above the tentorium
usion of the basilar artery itself is usually rap-
to infarction of respiratory and cardiac cen-
dulla. Occlusion of the perforating end-arteries
ar artery causes focal brainstem infarction, usu-
s cranial nerve dysfunction, ataxia, somnolence,
otor or sensory deficits. These lesions character-
t the midline of the brainstem and often extend
surface (Fig. 4.19). Metabolic disturbances (e.g.,
e myelinolysis) and hypertensive hemorrhages
nly in the pons) tend to be more centrally or dif-
Large or multiple lesions in the pons can cause
syndrome of quadriparesis with intact cogni-
ed in" state.

ebral Artery (PCA). The basilar artery ends at
into the PCAs at the midbrain level, just above
iatus. The major branches of the PCA include
thalamic *perforating vessels*, *posterior choroi-*
nd *cortical branches* to the medial temporal and
(Fig. 4.20). Ten to 15% of infarcts occur in the

nal segments of the PCAs sweep posterolaterally
idbrain, giving off small perforating branches
ephalon and thalamus along the way. Midbrain
ses loss of the pupillary light responses, impaired
omnolence due to damage of the quadrigeminal
anial nerve nuclei, and reticular activating for-
ctively. Proximal PCA perforators also supply
of the thalamus and sometimes portions of the
of the internal capsule. Thalamic infarction
ariety of disturbances, but contralateral sensory
st common problem.

FIGURE 4.18. **Vertebrobasilar Arteries. A:** Late
ilar; *5*, anterior inferior cerebellar artery (AICA)
9, branches of the SCA and AICA in the horizo
B: Anterior view. 1, right vertebral; 2, left vertel
communicating; 11, ICA. (From Osborn AG. *In*

The posterior choroidal arteries arise f
PCA to supply the choroid plexus of the thi
tricles, pineal gland, and regions contigu
ventricle. Isolated posterior choroid infarc
to rich collateral supply through the cho
cortical branches supply the inferomed
(inferior temporal arteries), superior occip
occipital artery), and visual cortex of t
(calcarine artery) (Fig. 4.21). Hemispheri
are usually from an embolic source. Inf
ral infarction may cause memory deficits,

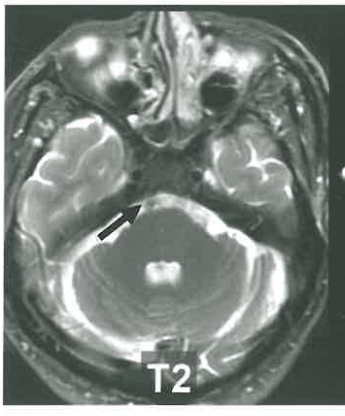


FIGURE 4.19. **Acute Brainstem Infarction.** Alth
(*white arrows*) which respects the midline. Note
due to focal atherosclerosis.

al visual field

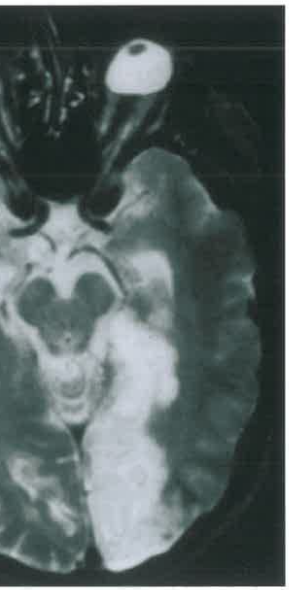
al visual field

ed

on

FIGURE 4.20. **Posterior Cerebral Artery Occlusion.** A PCA occlusion results in syndromes of memory impairment, opposite visual field loss, and sometimes hemisensory deficits. (From Patten J. *Neurological Differential Diagnosis*. New York: Springer Verlag; 1996.)

Because of clinical urgency, acute evaluation of bellar strokes should be performed by CT. Cer-rhages and any infarctions with significant mass rosurgical emergencies requiring posterior fossa n. Multiplanar MR is preferred for evaluation



nvolvement of the left occipital lobe and visual field defect.

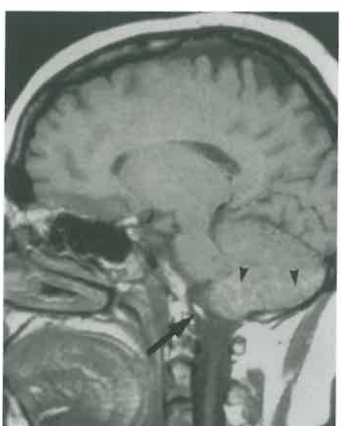


FIGURE 4.22. **Vertebral Dissection With PICA I**
and transverse (B) T1WI without contrast show l
left vertebral artery (*open arrows*). Hemorrhagic

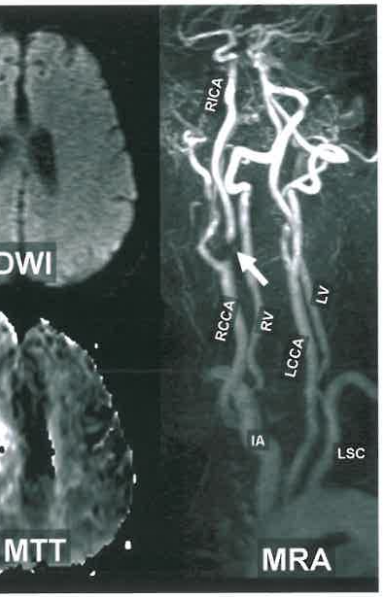
beyond the acute phase, since beam-hardenin
posterior fossa images on CT.

Even though deficits related to the cer
are hard to distinguish clinically, it is impo
characteristic distributions in order to eluci
anisms. Luckily, only a *SAP* would forget
of cerebellar branches going from top to l
rior, *a*nterior inferior, and *p*osterior inferior
(AICAs) (Fig. 4.18).

Superior Cerebellar Arteries (SCAs). The
cerebellum are supplied by the SCA. These
tal basilar as the last large branches beneath
ebelli. The SCA territory includes the superi
and superior cerebellar peduncles, and super
the cerebellar hemispheres (i.e., the "roof" c
Most SCA infarcts are embolic.

Anterior Inferior Cerebellar Arteries. Th
from the proximal basilar to supply the a
bellum and sometimes part of the middle
cle. AICA is usually the smallest of the thre
hemisphere branches. Occlusion commonly
limb ataxia, nausea, vomiting, dizziness, and

Posterior Inferior Cerebellar Arteries. T
cerebellum is supplied by the PICA. The
major intracranial branch of the vertebroba
ally arising from the distal vertebral artery
the basilar origin. Its territory is variable b
the dorsolateral medulla, inferior vermis, a
cerebellar hemisphere. PICA maintains a r
with AICA above it. If the PICA is large th
AICA is usually small, and vice versa. Thi
sometimes referred to as the AICA–PICA lo
ally the largest cerebellar hemispheric bran
commonly infarcted. Occlusions may occu
of a vertebral dissection which began at the
4.22). If only the cerebellar hemisphere is af
limb ataxia, nausea, vomiting, dizziness, a
seen, just as for AICA infarcts. Involvemen



Watershed Infarctions. This patient presented with ing-limb" TIAs. DWI shows a cluster of lesions in the diata (*black arrow*). MTT maps indicate long transit tire hemisphere, particularly the deep watershed zones = longer times). Gadolinium-enhanced MRA shows reat vessel origins, but a critical stenosis of the prox- with a flow gap (*white arrow*). Mechanisms leading emia are debated, but may include distal emboli, local to slow flow, and hemodynamic causes.

ssociated neurologic deficit or other clinical rele- nition, Virchow–Robin spaces should follow CSF ll MR sequences, have no associated mass effect, ong the path of a penetrating vessel. Common ude the medial temporal lobes and inferior one- utamen and thalamus. Occasionally they may be e course of small medullary veins near the vertex, n T2 images at 3 T. Most perivascular spaces seen etween 1 and 3 mm in diameter, but some may be er. Enlarged perivascular spaces are observed as ant in all age groups (Fig. 4.28). Both increasing ency are noted with increasing age.

Ischemic Changes. Small foci of T2 hyperin- ommonly seen scattered throughout the brains ents, with or without clinical symptoms. These dentified bright objects) can cause considerable . They are commonly associated with patchy or perintensity in the centrum semiovale (Fig. 4.28). e filled with different authors' terms for related ite matter hyperintensities, small vessel ischemic scent change, Binswanger disease, multi-infarct d leukoariosis, to name a few. There is no con- en these imaging changes should be considered d when they simply represent a normal part of cess. At one end of the spectrum are patients who d enough tiny infarcts over the years to impair n. Individually or in small numbers these were

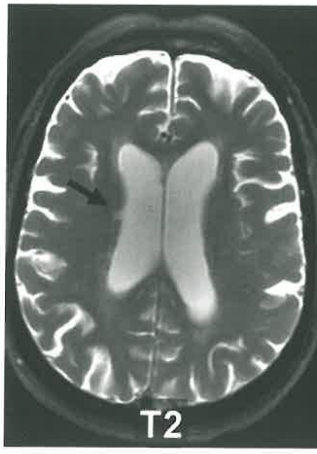


FIGURE 4.25. Old Versus New Lacunes Di
small old lacune in the right periventricular
edema of the acute infarct is seen only on DW
a month, and then evolves toward more wate

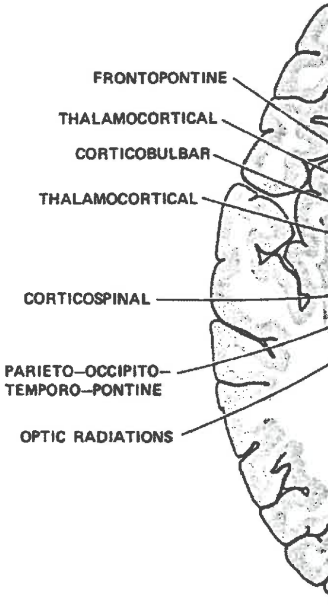
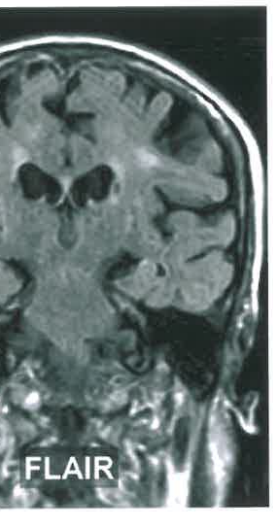


FIGURE 4.26. Somatotopy of the Internal Ca
right) and major fiber tracts passing through i
callosum; C(h), caudate head; C(t), caudate tai
ventricle; SP, septum pellucidum; Th, thalamus
Neuroanatomy & Neurophysiology. Philadelp

FIGURE 4.27. **Virchow–Robin Spaces.** All sequences show enlarged but normal perivascular spaces (*white arrows*) which exactly follow CSF intensity. There is no mass effect, and the patient had no symptoms referable to this region. These spaces are commonly seen at the ends of the anterior commissure ("black mustache," *black arrows*) in the anterior inferior basal ganglia. They should not be mistaken for lacunes, which typically show DWI hyperintensity acutely and signs of gliosis on FLAIR chronically.



Aging. Transverse T2WI at the level of
r areas likely represent prominent CSF
ws hyperintensity indicative of gliosis
ivascular spaces.

presumably asymptomatic, but in aggrega
vascular dementia picture. At the other er
are perfectly healthy patients who have pre
a speck of gliosis or occlusion of an inconse
as a normal part of aging. The clinical findi
which of these patients with small vessel
needs further workup.

*Vasculitis and Reversible Cerebral Vaso
drome.* Patchy inflammatory changes in arte
to either large or small vessel stroke. Vasculit
by autoimmune disorders, polyarteritis nod
processes (e.g., giant cell arteritis). Drug
amphetamines, serotonin reuptake inhibito
postictal states have been linked to revers
constriction syndrome (RCVS), which can
RCVS causes irregular beading of vessels, b
the inflammatory infiltration of the vessel
of vasculitis. Vasculitic infarcts are often sca
tiple vascular territories and therefore may
patterns of damage. Varying stages of inflar
fibrosis, and aneurysms may be seen simulta

Cases of suspected vasculitis are eval
tional angiography which provides the hig
olution. Views of the intracranial circulatio
carotid artery are reviewed in search of ir
rowing. High-resolution gadolinium-enha
MR images with fat saturation can also som
arterial wall thickening. Positive sites may
for biopsy confirmation. Sometimes the ve
so small the angiogram is normal. In these
muscle, or random temporal artery biopsy
to make the diagnosis. Diagnostic confirma
since many of the vasculitides respond to ste
drugs, or in the case of RCVS, potential tre
dilators and removal of the underlying trigg

Venous Infarction

Venous occlusion is an uncommon but im
stroke. Characteristically, venous infarcts
patients who present with headache, sudd
and often seizures. Predisposing factors in
uable states, pregnancy, infection (spread
scalp, face, middle ear, or sinus), dehydratio

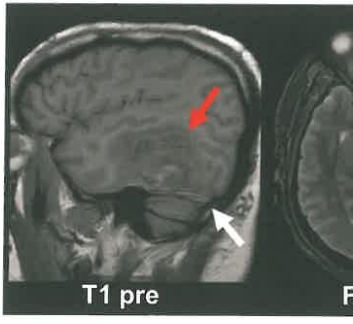
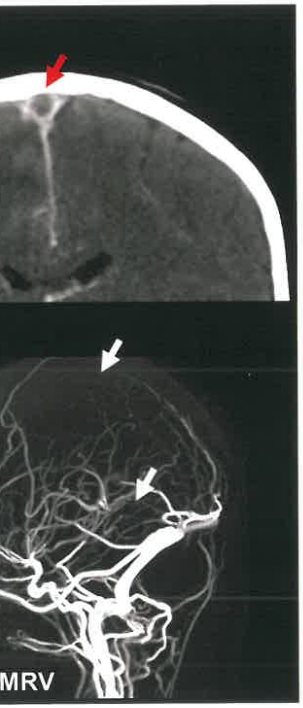


FIGURE 4.29. **Transverse Sinus Occlusion With**
occlusions. This previously healthy man presented
early hemorrhage (consistent with intracellular deo
This location and appearance is classic for venous i
transverse sinus (*white arrows*) becomes thrombos



us Infarction. This 4-year-old girl recently
d left hemiparesis. CT images (above) show
Filling defects (clot) outlined by enhancing
nus thrombosis (*arrows*) in both axial and
bin clot in the superior sagittal sinus (*white*
ck of flow in these sinuses.

embranes. Dominant among these mechanisms
on state and location of iron species related to
Oxygenated hemoglobin is sequentially con-
xyhemoglobin, methemoglobin, and then hemo-
time. The magnetic properties of the resultant
products change the MR relaxation rates of adja-
allowing the hemorrhage to be detected. A small
unding edema is common in the subacute phase
nal bleeds, sometimes making interpretation of
s quite complex. High-field scanners and gradi-
uences tend to improve conspicuity of subacute
lood products. The general pattern of MR signal
over time on a 1.5 T magnet is summarized in
Figure 4.32. Individual cases may of course vary
om these simplified guidelines due to the multiple
ed.

roll down physical chemistry lane will help us
e complicated signal changes seen during the evo-
norrhage. In order to change the signal character-
ue, hemorrhage must affect T1 or T2 relaxation.
l oxidation products of hemoglobin accomplish
anges in both magnetic properties and in molec-
ation. Iron within hemorrhage breakdown prod-
he effective local magnetic field, a process known
susceptibility. This change in field is translated

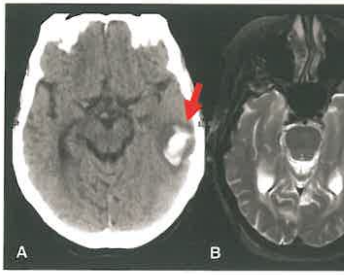


FIGURE 4.31. Amyloid Versus Hypertensive Mi
hematoma (*red arrows*) which blooms darker on
old hemorrhages (aka "microbleeds") on T2*, di
sive hemorrhage (T2, D, and T2*, E) more typic
often keeping company with lacunes. Although C
weighted sequences are required to detect older r

into an alteration in signal intensity because
slowing of T1 and T2 relaxation rates. Ch
ation occur only within a very short range
stroms) while T2 effects can be seen millime

Under normal conditions, circulating re
tain a mixture of both oxy- and deoxyh
During transit through the capillary bed, ti
gen according to metabolic needs, convertir
to deoxyhemoglobin in the process.

Neither of these forms have much detec
signal intensity in clinical images, but th
guished due to their opposite effects on T2
bin is a diamagnetic compound containing
detected as high signal intensity on T2WI
echo). Deoxyhemoglobin also contains Fe^{+2}
magnetic substance. The magnetic suscep
hemoglobin causes accelerated dephasing o
T2*-weighted images (e.g., gradient-recalle
which results in signal loss. *Deoxyhemog*
hypointense on heavily T2WI. These patte
signals are occasionally encountered on c
acute hemorrhage. These same magnetic su
related to the balance of blood oxygenation
clinical functional MR mapping methods (b
vated by a task recruit more blood flow an
detected as a focal *increase* in T2* MR signa

When hemorrhage occurs, oxyhemoglob
deoxyhemoglobin at a rate dependent on loc
tension. This takes place over hours for pare
mas but can be considerably delayed when c

TABLE 4.4

EVOLUTION OF HEMOR

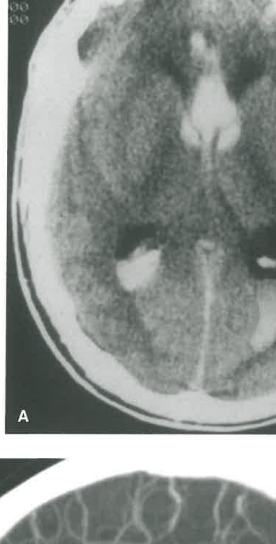
TIME	RBC[a]
<1 day	Intact
0–2 days	Intact
2–14 days	Intact
10–21 days	Lysed
≥21 days	Lysed

[a]Red blood cells.

FIGURE 4.32. **Biochemical Evolution of Hemorrhage.** Within minutes of hemorrhage, a hematoma consists of intact red blood cells containing oxyhemoglobin. Over several hours, the clot begins to retract and the hemoglobin is oxidized from oxy- to deoxy- to methemoglobin. Methemoglobin tends to form in a ring which converges from the periphery to the center over time. Red cells lyse, releasing methemoglobin into the surrounding fluid. Macrophages break down the iron products into hemosiderin and ferritin, leaving a stain at the periphery of older hematomas. (From Atlas SW. *Magnetic Resonance Imaging of the Brain and Spine*. New York: Raven Press; 1991.)

who presents with SAH is very likely to harbor a genital (berry) aneurysm (Fig. 4.33). One to 2% neurysms, thought to occur due to a congenital e arterial media. Probably many of these aneu- asymptomatic, but those greater than 3 to 5 creased risk for rupture. Berry aneurysms often anch points of the circle of Willis. About 85% the anterior part of the circle of Willis, while the vertebrobasilar territory. Common loca- branch points near the anterior communicat- iddle cerebral (30%), posterior communicating asilar (10%) arteries. Less commonly the oph- , cavernous ICA, or PICA are to blame. When aneurysms are seen, an episode of prior trauma nfection should be considered (e.g., bacterial with "mycotic" aneurysm). Other conditions th aneurysms include atherosclerosis, fibromus- , and polycystic kidney disease. Management he clinical situation, location, and size of the eatment options include surgical clipping, inter- lovascular coil embolization, and combinations ig. 4.34).

e acute SAHs easily seen by CT may be entirely utine spin-echo MR. CT is over 90% sensitive ion of acute SAH, probably due to the increased tted blood. Use of FLAIR sequences on MR can picuity of acute blood, but CT is still considered method of choice when clinical findings suggest of SAH (Fig. 4.34). SAHs may be quite difficult by CT when the patient's hematocrit is low, the morrhage small, or there is a delay in scanning.



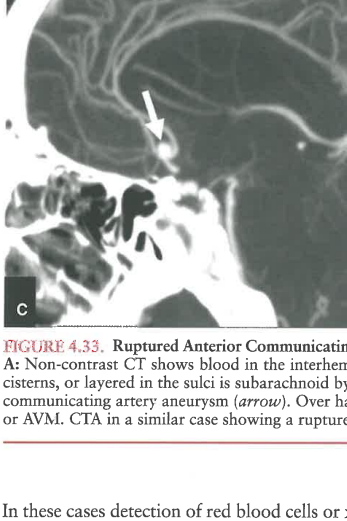
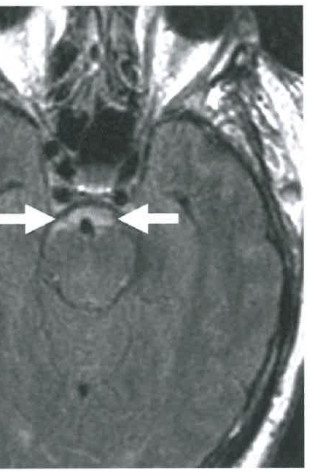
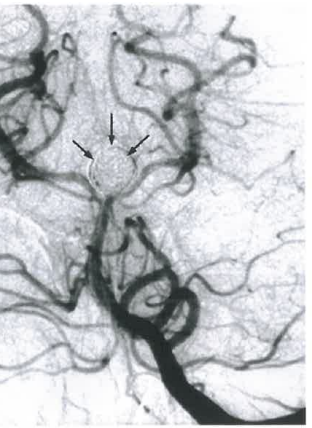


FIGURE 4.33. **Ruptured Anterior Communicatin**
A: Non-contrast CT shows blood in the interhem
cisterns, or layered in the sulci is subarachnoid by
communicating artery aneurysm (*arrow*). Over ha
or AVM. CTA in a similar case showing a rupture

In these cases detection of red blood cells or
lumbar puncture may be the only way to co
SAH. The most sensitive places to look for S
dependent portions of the subarachnoid spa
causes the blood to settle—the interpeduncul
sylvian fissure, and the far posterior aspect
horns (Fig. 4.35). Prompt scanning is impo
lution of subarachnoid blood reduces CT s
by day 3.

About 15% to 20% of patients with sul
ing will have multiple aneurysms. Due to t
CTA or "four-vessel" angiogram is needed c
uation. When multiple aneurysms are presen
largest or more irregular, has focal mass effe
mal clot, or shows a change on serial exam
to be the culprit. CTA has become an imp





36-year-old patient presented with a severe
in the interpeduncular fossa (*arrows*) and
utine MR sequences, but is easily visible on
ection shows a basilar tip aneurysm (*arrow*).
atinum coils shows obliteration of the aneu-

ntion. Two- and three-dimensional CTA recon-
aneurysms can help select and plan either open
dovascular procedures.
studies are an integral part of SAH evalua-
al or subsequent CT may show communicating
is requiring a ventriculostomy or shunt. Epi-
ible rebleeding are evaluated with noncontrast
nay also be seen in patients with elevated intra-
re or vasospasm, and are the main pathologic
ients whose condition continues to deteriorate
ial SAH. Posttreatment angiography is used
quacy of clip placement and to rule out vaso-
graphy or MRA can be used to follow coiled

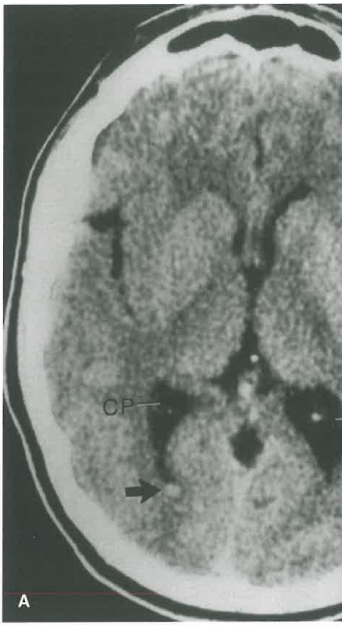


FIGURE 4.35. **Subtle Subarachnoid Hemorrhag**
horns (A, *arrow*) and the interpeduncular fossa
dense due to calcification or enhancement. The n

Parenchymal Hemorrh

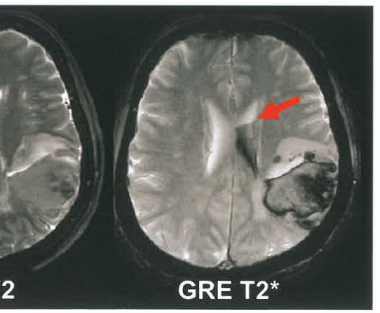
Primary intraparenchymal hemorrhage oc
bleeding directly into the brain substance.
rhages are not included in this section; the
Chapter 3. Parenchymal bleeds generally
tial mortality than infarcts, but on recover
cits than a similar-sized infarct. This is be
tends to tear through and displace brain
resorbed. A similar-sized infarct is made
than just displaced neurons. The main dif
ations are hypertensive hemorrhage, vascul
drug effects, amyloid angiopathy, and blooc

Hypertensive hemorrhages are seen in th
to 50%), the subcortical white matter (3
lum (15%), thalamus (10% to 15%), a
10%) (Fig. 4.36). As with lacunes, lipohy
is thought to be the primary predisposing
although miliary aneurysms in the vessel w
a role. Small hypertensive hemorrhages
few deficits. Bleeds in the posterior fossa,
amount of mass effect, or hemorrhages e
ventricular system have a relatively poor p
tern of microbleeds sometimes seen in a pa
rhage can help distinguish hypertension fro
bleeding (Fig. 4.31). Focal contrast extrav
acute hematoma on CTA or routine contras
("spot sign") predicts a high risk of clot e
first several hours after admission, compare
a spot sign (Figs. 4.36 and 4.37).

FIGURE 4.36. **Hypertensive Hemorrhage, "Spot Sign" Predicts Clot Expansion.** This patient with a history of hypertension presented with abrupt left hemiparesis. Noncontrast CT (**A**) shows a focal parenchymal hematoma centered in the right putamen. CTA source images (**B**) show a tiny focus contrast extravasation (*arrow*) contained within the hematoma which is even more conspicuous on routine postcontrast images (**C**) obtained 4 minutes after the CTA. Focal spot signs on either CTA or contrast CT suggest active bleeding and therefore high risk of hematoma expansion over the next several hours. (**D**) Follow-up noncontrast CT at 24 hours confirms marked enlargement of the hematoma as well as worsening mass effect.

ally by contrast-enhanced MR, most often in the ment is necessary.

rmations (aka developmental venous anomalies, nas) are congenitally variant veins which drain They are seen in about 5% of patients studied by but may easily be missed on CT or noncontrast



ntense on T1 and T2, with heterogeneous dark signal , "blooming" due to susceptibility). Focal puddles of RI "spot sign" indicative of active bleeding with risk n (*arrow*, T2*), and clot volume >30 mL (5 × 5 × 4 cm

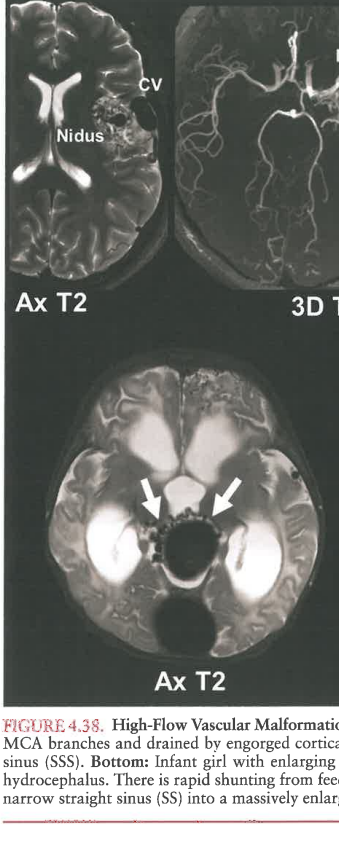
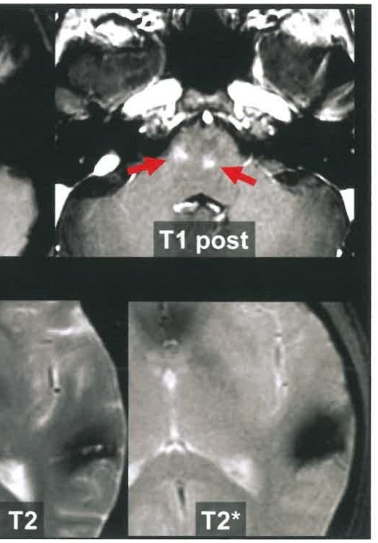


FIGURE 4.38. **High-Flow Vascular Malformatio** MCA branches and drained by engorged cortica sinus (SSS). **Bottom:** Infant girl with enlarging hydrocephalus. There is rapid shunting from fee narrow straight sinus (SS) into a massively enlar

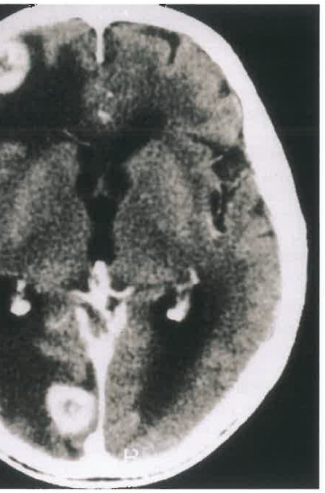
MR. The classic appearance is of an enlarge venous complex extending to the ventricular The contrast-enhanced MR appearance is and most will show a dark signal on T2* d venous blood, such that angiography is ra for confirmation. Although these may rarely somewhat controversial since they are very asymptomatic patients and are often the on for a brain region.

Cavernous malformations are thin-walle (neither arteries nor veins) which may prese small parenchymal hemorrhages. These lesi tomatic and can occur on a familial basis. ography are usually normal. MR will show enhancing lesion with dark rim (hemosiderin Venous malformations may provide drain malformations, but no arterialized feeding seen. Unless recently ruptured, a cavern should show no mass effect or edema. If a met, conventional angiography may be unn

Hemorrhage due to Coagulopathies. I rhage may also occur because of blood d oral anticoagulation increases by eightfol cranial hemorrhage. The association is part



ngiectasias. No changes are visible on T2, but focal
small veins. These are visible as lacy, focal enhance-
that should not be confused with pathology such as
) with associated cavernous malformation (aka, cav-
centrally, with dark periphery (*red arrows*). A small
ward the periatrial ependymal veins (*white arrows*).
products including hemosiderin. DVAs are usually
leed or cause focal symptoms.



e lung presented with new onset seizures. The
matocrit" layer (*arrow*). Marked white matter
scan (B) shows irregular ring enhancement of
egree of surrounding edema, focal and irregu-

TABLE 4.5

FEATURES OF BENIGI

SIGN	
Evolution of blood prod	
Hemosiderin rim	
Surrounding edema	
Acute enhancement patte	

related to cerebral amyloid angiopathy. W
cal involvement can be seen in some cases
T2*-weighted MR sequences are used to ma
more conspicuous (Fig. 4.31). Amyloid angi
to mind when an elderly or demented pa
new or recurrent superficial hemorrhages. F
"microbleeds" may also be an underlying so
of postthrombolytic hemorrhage.

Primary Hemorrhage V
Hemorrhagic Neopla

Intracranial tumors are an uncommon b
cause of intracranial hemorrhage. They a
2% of bleeds in autopsy series and as high
clinical radiologic series. Tumor necrosis,
and neovascularity may contribute to th
hemorrhagic neoplasms. Glioblastomas are
primary brain tumors to hemorrhage, whil
category bronchogenic carcinoma, thyroid
riocarcinoma, and renal cell carcinoma ofte

It may be possible to distinguish between
plasm and a primary (benign) intracranial
on the MR findings. Intratumoral bleeds ten
plex and heterogeneous than benign hemato
evolution of blood products is commonly de
possibly due to profound intratumoral hyp
scanned in the acute phase, lack of enhanc
hematoma strongly supports a primary in
rhage. If there is an enhancing component
as tumor or AVM must be considered. In t
however, a resolving hematoma may develop
enhancement of its own. Both acute hemo
rhagic neoplasms may cause an edematous
in the tumors edema is more predominant. In
nial hypertensive bleed, the edema should be
resolve within a week, while in the presenc
should persist. With a resolving benign hem
cumferential hemosiderin ring begins to dev
3 weeks' time on MR. In the hematoma asso
this hemosiderin ring may be absent or inco
ful differential features are summarized in Ta
when the findings are ambiguous, a follow-u
to 6 weeks will clarify the diagnosis, avoidin

Suggested Reading

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