that supplied both frontal lobes. The circle of Willis as an
anatomic structure can be defined as a network of blood vessels
affording communication between the carotid system and the
basilar system, encompassing an anterior and two posterior com-
municating arteries and proximal portions of the anterior and
posterior cerebral arteries. However, this description is still not
fully satisfying because part of the middle cerebral artery and the
top of the basilar artery also contribute to this communicating
circle. It was our deliberate choice to list the anomaly among
anomalies of the circle of Willis to avoid confusing the reader, who
will find virtually all other previously reported variations of the
postcommunicating ACA in articles about anomalies of the circle
of Willis, as is illustrated in several important papers considering
large populations.1,4 Moreover, De Smet illustrates the ambiguous
use of the term "circle of Willis" even by Willis himself when De
Smet refers to "the circle depicted in Willis' Cerebri Anatomicus"2
showing "the ACAs proceeding medially and coming together in a
confluence, a possible but most unlikely anomaly..." Furthermore,
moreover, the statement that the unique anomaly we described could
in fact have first been reported by Willis himself in 1664 is
extremely speculative, as is also illustrated by the use of the
conditional tense by De Smet in his letter. Even if, as he suggests,
the draftsman who contributed to Willis' 1664 paper included "the
unknown anterior communicating artery" in the precommunicat-
ing part of the right ACA, the anomaly in our patient still differs
from this would-be variant because of the existence of a plain
communicating ACA.

In the second part of his letter De Smet addresses the clinical
features of our case. Erroneously, however, he writes that our
patient had ophthalmoplegia. This was the case for one of the
two patients discussed by Ferbert and Thron,4 who rightly conclude
that bilateral ACA territory infarction should be considered in the
differential diagnosis of basilar artery occlusion, even when it is
accompanied by vertical gaze palsy. De Smet proposes that an
association of quadriplegia with akinetic mutism and ophthal-
moplegia corresponds to the tetraparetic mutism he described,6,10
which combines a locked-in-like deafferentation with an akinetic
mutism-like deafferentation. For our patient, we prefer to use only
the term "akinetic" mutism and agree with Ferbert and Thron 8
that variations of a classical akinetic mutistic syndrome may exist.
Communication by means of a code of eye blinking or vertical eye
movements, as in the locked-in syndrome, was not possible for our
patient. We further suggest an association with receptive aphasia,
because of the state of akinetic mutism we were unable to
prove this hypothesis.

Peter P. De Deyn, MD, PhD
Fons Borgherve, MD
Department of Neurology
General Hospital Middelheim
and Department of Neurochemistry and Behavior
Born Bunge Foundation
University of Antwerp
Antwerp, Belgium

References

1. Fawcett E, Blachford JV. The circle of Willis: an examination of

2. Von Mitterwallner F. Variationsstatistische Untersuchungen an

3. Baptista AG. Studies on the arteries of the brain, II: the anterior
cerebral artery—some anatomic features and their clinical

1887;22:289-293.

5. Hillen B. The variability of the circle of Willis: univariate and

6. Hillen B. The variability of the circle of Willis (Willisii): order

7. Hughes JT. Willi's contribution to neuroanatomy and neurophys-
ology. In: Hughes JT, ed. Thomas Willis 1621-1675: His Life and
Work. London, United Kingdom: Eponymists in Medicine, Royal
Society of Medicine Ltd; 1991;8:60-73.

8. Ferbert A, Thron A. Bilateral anterior cerebral artery territory
infarction in the differential diagnosis of basilar artery occlusion.

9. De Smet Y, Rousseau JJ, Brucher JM. Infarcuts putamino-
capsulo-caudés bilateraux, synétyques et simultanés. Rev Neurol.
1990;146:415-419.

J Neurol 1993;240:258-259.

Vertebral Flow Void and Lateral Medullary Syndrome

To the Editor:
The outstanding descriptions by Kim et al1 of magnetic reso-
nance (MR) imaging findings in the lateral medullary syndrome
include three fabulous MR imaging sections through the medulla
(Fig 2). The figure legend does not provide details of the
neuroradiological findings. One "pearl" overlooked in the text is
that all three images showed absence of the normal left vertebral
flow void (FV) phenomenon. This finding has been previously
described 1,4 and is worth reiterating.

Joseph S. Jeret, MD
Rockville Centre, NY

References

1. Kim JS, Lee JH, Suh DC, Lee MC. Spectrum of lateral medullary
syndrome: correlation between clinical findings and magnetic res-


diagnosis of basilar artery occlusion using magnetic resonance

findings in a case of locked-in syndrome. J Neuromaging. 1993;3:
139-141.

Response

Dr Jeret noticed the abnormal vertebral FV pattern in the left
vertebral artery presented in Fig 2 of our article.1 In fact, the right
vertebral artery showed enhanced FV signal on this T1-weighted
MR image as well. The enhanced signal intensities were most
intense at the level of the lower medulla (bottom panel), which
diminished as the section planes went up. Unfortunately this
figure, presented to demonstrate the MR imaging findings of three
different cuts of the medulla, was not from a patient with lateral
medullary syndrome but from one with a supratentorial lacunar
infarct. In this patient, the T1-weighted MR image showed a
normal vertebral FV pattern, arguing strongly against the possi-
bility of thomboembolic formation.

The FV pattern in MR imaging has usually been assessed using
a T1-weighted image,2,4 where altered FV signal is more reliably
detected. In our daily practice, we occasionally observe the
abnormal vertebral FV pattern on a T1-weighted image unaccom-
panied by corresponding changes on the T2-weighted image in
patients with various types of stroke. This phenomenon might
suggest reduced blood flow velocity in vertebral arteries. However,
this almost always occurs bilaterally, and exclusively in vertebral
arteries (but not the basilar artery) seen in less than three
consecutive cuts from the lowest image. Therefore, we think that
this phenomenon is more likely to be caused by tilted cutting of
the vertebral artery or entry slice phenomenon, and that a decisive
conclusion should not be made unless the T2-weighted MR image
shows compatible findings.
Nevertheless, after receiving this interesting correspondence, we reviewed the vertebral FV pattern seen on MR imaging in the 26 patients with lateral medullary syndrome included in our study. In five patients we noted the absence of normal vertebral FV on the T2-weighted image, all on the same side of the lesion. Enhanced FV signal in the same vertebral artery was noted on the T1-weighted image in all five patients. We also examined the vertebral FV pattern in a control group of 20 patients with unilateral pontine basis infarction who were of comparable mean age and had vascular risk factors similar to those of the patients with lateral medullary syndrome. We found that none showed the absence of vertebral FV. Therefore, although it is infrequently seen, the altered vertebral FV pattern judged from both T2- and T1-weighted MR images appears to be of value in the evaluation of the vascular status of patients with lateral medullary syndrome.

References

Vertebral flow void and lateral medullary syndrome.

J S Jeret

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