Neurosurgical forum

2. Diringer H, Braig HR: Infectivity of unconventional vi-
3. Nisbet TJ, MacDonaldson I, Bishara SN: Creuzfeldt-
Jakob disease in a second patient who received a cadaveric
dura mater graft. JAMA 261:1118, 1989 (Letter)

RESPONSE: The patient described by Dr. Masullo and
his colleagues appears to be the third to have acquired
Creuzfeldt-Jakob disease (CJD) from a cadaveric dura
mater graft. Transmission of CJD by that route should
now be considered established. While the CJD agent
is unusually difficult to eradicate and has caused disease
in only a small number of patients, the lessons from
these three cases are clear. Alternatives to using cadav-
eric dura mater grafts should certainly be sought. Ster-
ilization procedures for reusable instruments and trans-
planted human tissues should be adequate to destroy
the infectivity of the CJD agent whenever that is pos-
sible. When it is not (blood, whole organs), screening
of donors to eliminate those with dementing neurolo-
cal illness should be especially diligent. As recom-
mded in our report in the Journal of Neurosurgery,
accepting as donors only persons under 50 years of age
would be likely to make such screening simpler and
more effective.

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Surgical Management of Spinal Fractures

TO THE EDITOR: We would like to compliment Dr.
Bucholz and Dr. Cheung on their careful and thorough
review of the conservative management of traumatic
spine injuries (Bucholz RD, Cheung KC: Halo vest
versus spinal fusion for cervical injury: evidence from
an outcome study. J Neurosurg 70:884–892, June,
1989). The nonsurgical management of spine fractures
is gaining support and has proved to be an effective
alternative to spinal fusion in select cases.

There are instances, in our opinion, that require
surgical intervention. 1) When the spinal canal and
neural elements are compromised following compres-
sion injuries, we recommend anterior decompression
of retropulsed bone and disc fragments followed by
interbody fusion and A-O (ASIF — Association for
Study of Internal Fixation) plating. 2) In hyperexten-
sion injuries where the anterior ligamentous complex
and anulus are disrupted, we perform discectomy and
interbody fusion followed by A-O plating. Thus, to
the already useful management algorithm presented in
the paper by Bucholz and Cheung we would simply add a
pathway for the treatment of patients with anterior
instability and compression injuries.

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RESPONSE: We thank Drs. Rovin and Cybulski for
their comments on our management algorithm. As
mentioned in our treatment protocol (page 885), sur-
gical fusion was performed without preoperative fixa-
tion in patients with partial spinal cord injuries produc-
ing continuing spinal compression as documented by
myelography. As our algorithm was designed in re-
spose to our experience in management of our partic-
ular subset of patients, we did not incorporate the
management of patients with partial spinal cord injury
into the flow chart published for this paper.

We certainly agree that anterior compression requires
prompt evaluation and appropriate surgical manage-
ment in the presence of continued or progressive neu-
rological deficit. However, as this group of patients was
specifically excluded from our patient population, based
on our current study we cannot comment on the ap-
propriate surgical management for these difficult cases.

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Pneumatized Anterior Clinoid Mimicking an
Aneurysm

TO THE EDITOR: I would like to add a third case to
the two cases reported by Gean, et al. (Gean AD, Pile-
Spellman J, Heros RC: A pneumatized anterior clinoid
mimicking an aneurysm on MR imaging. Report of
patient was a 38-year-old white woman who presented
with vascular headaches and depression. Magnetic res-
one imaging revealed a low-signal area in the right
paracilind region, which we thought might be an
eurysm. However, prior to performing arteriography we
obtainedplainfilmswithlocalizedviewsofthesella
turcica. On that study it was obvious that there was a
pneumatized anterior clinoid process. This case illus-
trates that plain skull x-ray films are still valuable, and
I would also recommend a computerized tomography
scan of the area as an investigation of the anterior
clinoids.

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Cystic Glioma With Positional Oculogyric Crisis

TO THE EDITOR: Dr. Heimburger has described an
interesting case of a cystic glioma presenting with po-
tional oculogyric crises (Heimburger RF: Positional
December, 1988). The author has attempted to explain
anatomically the supine-positionally induced conjugate
upward eye deviation and neck extension in this patient.
He quotes some fine but rather old studies1,4,8 and
incorrectly states that “Even though no specific nucleus

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or tract that controls vertical eye movements has been identified, the mesencephalon at the level of the quadrigeminal plate is implicated in the reports of changes in vertical eye positioning."

Over the 22 years since Dr. Heimburger's latest quoted reference on the topic, there has been a considerable amount of work, in both anatomy and physiology, which has led to the refinement of our knowledge of vertical ocular motor control. The excitatory short-latency burst neurons for vertical saccades are situated in the medial rostral midbrain tegmentum in the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF).3,9 There is a topographic arrangement with the upgaze burst neurons situated caudal, ventral, and medial in the riMLF, and the downgaze burst neurons situated rostral, dorsal, and lateral.7 There is a functional segregation in the efferent projections from the riMLF, with the upgaze fibers traversing the posterior commissure en route to the extraocular motor nuclei,2 while the exact course of the downgaze fibers is less clear. A slightly more caudal deep medial mesencephalic structure that is relevant to this case is the interstitial nucleus of Cajal, which carries vertical eye velocity and position signals to vertical ocular motor neurons5 and serves as a mathematical integrator of vertical eye velocity commands to eye position commands.6 The interstitial nucleus of Cajal plays a role in neck and axial motor control through the interstitial-spiral tract, hence offering an explanation for the associated neck extensor spasms in this case. It also receives bilateral vestibular nuclear projections. It is possible that the positional triggering of the crises in this patient was initiated by a change in activity in the ascending central vestibular projections to a rostral deep mesencephalon that is both "irritated" and distorted by the cystic tumor.

Dr. Heimburger alludes to the patient's paralysis of downward gaze in his discussion. I believe this is a mistake, as there is no mention of this in his case report, only the oculogyric crises. During these tonic spasms of upgaze, by definition, downgaze must of course be temporarily absent.

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References

Response: Dr. Stechison should be commended for bringing findings on the nuclei and tracts which control vertical eye movements to the attention of the Journal's readers. His correction of my statement about paralysis of downward gaze is well taken, and I thank him. It should have read "temporary paralysis of downward gaze" since the patient was unable to look downward sufficiently to bring his irises into view only while she was in the supine (oculogyric) position.

Dr. Stechison's strongly stated views confirm that not all readers reach the same conclusions after reading the same report. It would be nice if the authors of the papers Dr. Stechison cites were as positive as he is in transferring data from monkey and cat brains to the human brain. As an example, Dr. Stechison relies strongly on the uncellular recordings by Büttner, et al.,7 to make his point. The authors of that paper state in their section entitled Anatomy: "The location of eye movements related to neurons is also in good agreement with available clinical data, but is not sufficient to explain all clinical phenomena like occurrences of isolated upward or downward paresis. However, most of our units were located relatively rostrally, in the area which from the few clinical reports ... appears to be concerned with downward eye movements." Careful observation of more than 100 human patients after bilateral stereotactic lesions made in the pulvinar near the posterior commissure did not reveal any changes in ocular motility, even though spasticity and intractable pain were decreased. It is probable that nuclei thought to control vertical eye movements were damaged in some of these patients, although there is no autopsy proof. Additionally, chronic stimulation of electrodes with five to seven outlets placed stereotactically into the periaqueductal gray matter to treat intractable pain have not produced changes in eye motility, in spite of the fact that the nuclei which are thought to control vertical eye movements were undoubtedly involved by spread of the electrical current. This emphasizes the differences between species stated in the 37-year-old paper by Bucher and Bürgi,1 cited by Dr. Stechison. The lack of changes in eye motility from relatively
discrete stereotactic lesions in humans suggests that a larger lesion, which incidentally includes the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) and rostral pole of the interstitial nucleus of Cajal, is necessary to produce changes in vertical eye motility in the human.

The advances made through imaginative use of electrical measuring devices introduced stereotactically into animal brains is truly admirable. Ranalli, et al., make a striking comparison between these 10-year-old electrophysiological animal studies and human pathological findings. Since all of the pathologies studied by Ranalli, et al., were the result of infarcts, it is safe to assume that the changes in ocular motility came from widespread complete or incomplete damage in the part of the brain which includes the riMLF. The location of the lesions found by Ranalli, et al., encompass the fairly diffuse midbrain area implicated in the patient whom I reported. Until more stereotactically controlled electrophysiological studies can be made in man, the 22-year-old paper by Nashold and Gills remains the authority on the location of stimuli that affect vertical eye movements in the human brain.

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References

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