attention of the readers of the Journal two papers that were published from this laboratory. The first dealt with the presentation of a technique called "autocerebral cooling," which brought about rapid temperature reductions of brain without requiring pumping or oxygenating units.¹ To date, this method remains the simplest way to cool the human brain. The second paper specifically addressed the problem of cognitive performance following cooling and cerebral circulatory arrest.² These investigations demonstrated that, even in the highly trained subhuman primate, decision-making is preserved.

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References


Response: The work reported by Dr. White and his colleagues in the late 1960's and 1970's forms, at least in part, the pedestal upon which modern methods for establishing profound cerebral hypothermia and circulatory arrest are based.¹² Particularly important is their demonstration that experimental hypothermic cerebral circulatory arrest in subhuman primates was comparable. In addition, they developed techniques by which the brain could be selectively cooled and blood flow arrested while the temperature and blood flow to the remainder of the body was left unaffected. The techniques which they developed are attractive in that they avoid many of the potential difficulties which we addressed in our paper. We have no experience with the techniques described by Dr. White and his colleagues either in the laboratory or in the operating room. Establishment of selective cerebral cooling and circulatory arrest requires surgical exposure and manipulation of both carotid and vertebral arteries as well as cannulation of at least some of these vessels along with the femoral artery. Application of these techniques in humans who are simultaneously undergoing intracranial aneurysm surgery, however, may be logistically difficult and subject the carotid and vertebral arteries to an unnecessary risk of iatrogenic damage. The techniques for establishing profound hypothermia and complete circulatory arrest using cardiopulmonary bypass are well designed, readily available, and well tolerated by most patients. In our institution, at least, we plan to continue using cardiopulmonary bypass in these difficult situations.

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References


Transmission of Creutzfeldt-Jakob Disease by Dural Cadaveric Graft

To The Editor: We have recently read about the probable association between Creutzfeldt-Jakob disease (CJD) and cadaveric dura mater grafts (Thadani V, Penar PL, Partington J, et al: Creutzfeldt-Jakob disease probably acquired from a cadaveric dura mater graft. Case report. J Neurosurg 69:766-769, November, 1988). We have just cared for a young patient, a 27-year-old white man, who had received a dura mater graft of Lyodura in May, 1985, in the course of neurosurgical treatment for fibrous dysplasia of the right temporal bone tissue. In January, 1989, he rapidly became confused and developed gait disturbances, left-sided hemiparesis, left hemianopsia associated with hemispatial neglect, and myoclonus. In April, 1989, a brain biopsy showed diffuse spongiosis scattered along all layers of the cortex, neuronal shrinkage, and reactive hypertrophic gliosis; these neuropathological findings were diagnostic of CJD.

This is the third case of CJD probably acquired through a cadaveric dura mater graft. A second case has very recently been reported from New Zealand.¹ These observations call attention to the still-existing potential risk of the development of CJD in patients who received Lyodura cadaveric dura mater grafts manufactured by B. Braun Melsungen AG, Federal Republic of Germany, prior to May 1, 1987; after that date, according to representatives of B. Braun Melsungen AG, their procedures for collection and processing of dura mater were revised to reduce the risk of CJD transmission, including in the manufacturing process a 1-hour exposure of 1 N sodium hydroxide (NaOH),² a treatment known to inactivate the CJD virus in brain tissue.¹ Notwithstanding that, at the moment neurosurgeons should still seriously evaluate the alternative use of autologous material (such as tensor fascia lata) in cases requiring dura mater replacements.

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References

1. Brown P, Rohwer RG, Gajdusek DC: Newer data on the inactivation of scrapie virus or Creutzfeldt-Jakob disease
RESPONSE: We thank Drs. Rovin and Cybulski for their comments on our management algorithm. As mentioned in our treatment protocol (page 885), surgical fusion was performed without preoperative fixation in patients with partial spinal cord injuries producing continuing spinal compression as documented by myelography. As our algorithm was designed in response to our experience in management of our particular subset of patients, we did not incorporate the treatment 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 management in the presence of continued or progressive neurological deficit. However, as this group of patients was specifically excluded from our patient population, based on our current study we cannot comment on the appropriate surgical management for these difficult cases.

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Surgical Management of Spinal Fractures

To THE EDITOR: We would like to compliment Dr. Buchholz and Dr. Cheung on their careful and thorough review of the conservative management of traumatic spine injuries (Buchholz 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 compression 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 hyperextension 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 Buchholz and Cheung we would simply add a pathway for the treatment of patients with anterior instability and compression injuries.

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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 two cases. J Neurosurg 71:128–132, July, 1989). My patient was a 38-year-old white woman who presented with vascular headaches and depression. Magnetic resonance imaging revealed a low-signal area in the right paraclinoid region, which we thought might be an aneurysm. However, prior to performing arteriography we obtained plain films with localized views of the sella turcica. On that study it was obvious that there was a pneumatized anterior clinoid process. This case illustrates 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 positional oculogyric crises (Heimburger RF: Positional oculogyric crises. Case report. J Neurosurg 69:951–953, 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 studies and incorrectly states that “Even though no specific nucleus...