herpes zoster in which the ganglion itself has been directly damaged.

In our series, a single ganglion was removed in those patients in whom the injury was believed to have occurred at the level of the dorsal root or the ganglion itself, especially in patients with failed back syndromes. Dr. Smith is correct in stating that to denervate a peripheral area of pain generation would require excision of multiple levels, as was done in only a few of our patients. Many of our patients already exhibited sensory loss in a radicular pattern, but following sensory ganglionectomy each patient demonstrated a radicular pattern of partial sensory loss or hypalgesia/hypesthesia. A small central area of dense sensory loss was seen only in patients who had undergone excision of multiple sensory ganglia from contiguous areas.

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Diffuse Axonal Injury

TO THE EDITOR: In the recent research article by He, et al. (He X, Yi S, Zhang X, et al: Diffuse axonal injury due to lateral head rotation in a rat model. *J Neurosurg 93:* 626–633, October, 2000), the authors describe a new model of head injury in the rat by using angular acceleration to produce diffuse axonal injury (DAI).

Abstract

Object. The authors investigated the ramifications of producing diffuse axonal injury (DAI) by lateral head rotation in a rat model.

Methods. Using a special injury-producing device, the rat's head was rapidly rotated 90° in the coronal plane at an angular velocity of at least 753.13 rad/second and an angular acceleration of at least 1.806×10^5 rad/second^2; the rotation was complete within 2.09 msec. There were no statistically significant changes in PO_2, PCO_2, pH, or blood pressure values at 5, 15, or 60 minutes after head rotation compared with their respective preinjury baseline values. The rats exhibited posttraumatic behavior suppression for an average of 12.6 minutes. The mortality rate was 17%. The rats that survived had diffuse subarachnoid hemorrhage around the brainstem and upper cervical cord, but no obvious brain contusion. In sections stained with silver or hematoxylin and eosin, axonal swelling and bulblike protrusions at the axonal axis were observed in the medulla oblongata, midbrain, upper cervical cord, and corpus callosum between 6 hours and 144 hours postinjury. The axonal injuries were most severe in the brainstem and were accompanied by parenchymal bleeding. The density of bulblike axonal protrusions peaked 6 hours postinjury in the medulla oblongata and 24 hours postinjury in the midbrain.

Conclusions. Rapid lateral head rotation can produce DAI characterized by severe damage to the rat brainstem.

We believe that the core of the methods and conclusions are fundamentally unsound, particularly with regard to the biomechanics of injury.

It is well established that rotational acceleration is the predominant mechanism that causes tissue deformations within the brain in humans leading to DAI. Mass effects of the large brain of humans play an essential role in this process, in which the deforming brain can be thought of as literally pulling itself apart during the acceleration–deceleration period. To produce equivalent tissue strains in animals with smaller brain masses, the accelerations must be much greater. The authors failed to mention that injury-producing accelerations across different sized brains has been extensively examined[2,4] and is summarized by the standard known as the Holbourn scaling relationship: $\ddot{\Theta}_s = (M_{ref}/M_s)^{2/3}\ddot{\Theta}_{ref}$, where M_{ref} and M_s denote the reference and scaled brain masses, respectively, and $\ddot{\Theta}_{ref}$ and $\ddot{\Theta}_s$ denote the reference and scaled rotational accelerations, respectively.

This scaling relationship has previously been demonstrated in animal models of rotational acceleration. To replicate the tissue strains leading to DAI in humans, accelerations had to be increased 500% for a 140-g brain of a baboon[1] and 630% for a 90-g brain of a pig.[3,5] By extension, the inertial forces necessary to produce equivalent tissue strains in the less-than-2-g brain of a rat would be unachievable, and the accelerations would have to approach 8000% of the level required for DAI to occur in the brain of a human. The applied forces and accelerations reported by He and colleagues were, however, at least 10 times less than anticipated by the scaling relationship. In addition, insufficient details were provided to determine the accuracy of the accelerations reported.

Finally, it is important to note that the signature anatomical characteristic of DAI in humans is axonal damage distributed throughout the large white matter domains. But the lissencephalic brain of a rodent has a remarkable paucity of white matter, and as such, it could be argued that clinically relevant DAI cannot be produced in rodents by any means. Note that if one's goal is simply to study traumatic axonal injury, this pathology has already been convincingly demonstrated in a number of established rodent brain impact models.

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RESPONSE: We express our gratitude to Drs. Meaney, Margulies, and Smith for their comments on our research.

It has made us review and rethink the reliability and validity of our reported DAI model in rats.

For many years the rotational acceleration of the head has been considered an effective way to induce DAI in the brain. The lateral head rotation leads to more severe tissue deformations within the brain. It has been documented in many animal models that this rotational process, through shearing force, causes a series of behavioral and pathological changes similar to those that occur in humans.[3,7,10] The inertial effect determined by animal brain mass plays an important role in the magnitude of shearing force. It is reatively easier to produce DAI in animals with a larger brain. We have found that a few distinguished researchers have thoroughly studied the biomechanical mechanism and biopathological reactions in DAI by using nonhuman primate models or nonhuman primate and human surrogate brain models, with a combination of complicated physical, mathematical, and morphological methods to further understand DAI in humans.[4–6,8] Their milestone works have encouraged many followers to explore the possibility of inducing DAI in a variety of animals with brains of different sizes and anatomical structures. Ross, et al.,[13] successfully produced the DAI model in miniature swine, although the results in this model did not mirror exactly those of DAI in humans in terms of distribution of injured axons. Indeed, only a very small proportion of proposed animal models provide data of a precisely comparable distribution and time course of axonal damage to that found in models of DAI in humans.[2,7,9,12] It is our view that if a particular model reveals any behavioral and pathological data similar to those occurring in DAI in humans, it should be considered instrumental to understanding the biological mechanisms of axonal injury; which animal brains are used does not matter. Small animals such as rats are not expensive and are easily controlled. Although the smaller mass of the rat brain does not easily lend itself to DAI, we should not use that fact to prevent us from exploring and discover the possibilities of such a model. As was written in our article, the DAI in a rat model by using lateral head rotation produced significant posttraumatic behavior suppression, axonal swelling, and bulbs that were identical to the typical characteristics of DAI in humans. This is enough for us to study the biopathological and biochemical mechanism included in axonal damage. We believe that the rotational acceleration of a rat head is superior to brain impact in simulating the conditions of external forces leading to DAI in humans. In addition, the fact that in our model predominant damages were demonstrated in the brainstem indicated the biomechanical effects of shearing force on the part of the brain located in the atlantooccipital area. Although data in this model do not completely reflect the distribution of injured axons in DAI in humans, it can serve as a useful guide in understanding human brainstem injury caused by sudden displacement between the head and neck, that is, by the scourge-brandishing mechanism. Around this area, the white matter is relatively comparable between rodents and humans. Moreover, we also noticed in our clinical work that there were many head-injured patients who presented with only small bleeding spots in the midaxial area such as the basal ganglia and brainstem, as demonstrated using computerized tomography or magnetic resonance imaging, and a coma of lengthy duration but with relatively better prognosis. Although we had no autopsy data to support our hypothesis, we had reasons to deduce that the neuronal axons within the brainstem were injured. We think our DAI model produced in rats by using lateral head rotation had some similarities to that clinical phenomenon in humans.

The Holbourn scaling relationship is indeed a very scientific formula in scaling rotational acceleration based on the rotational acceleration value found in studies with experimental animals (reference rotational acceleration) and the brain mass of the studied animal (reference brain mass).[5,6,11] But we consider that, because of the anatomical disparities of the head and neck in various animal species, it is more rational to scale the rotational acceleration to animals with similar anatomical structures. In other words, it is more reliable to scale the rotational acceleration of the human brain according to the nonhuman primate (for example, a baboon) brain mass and acceleration. Hence, the mechanical parameters of the DAI model in rats with lateral head rotation are not suitable to be scaled to the DAI in humans by using the Holbourn scaling relationship. We prefer to measure the mechanical parameters for one animal species or one individual within that species and to make scaling across the similar species or across the different individual within the same species, rather than to refer to the mechanical parameters of one species and make scaling to another species with different head and neck structure.

The instruments we used to measure and evaluate the rotational velocity and acceleration mainly included an acceleration transducer (impulsive load transducer), a charge amplifier, and a voltage meter (digital peak volt). Based on the instruction manual of the charge amplifier Type 2635,[1] the output voltage sensitivity, or the peak voltage sensitivity (S_p), is determined by the charge sensitivity of the acceleration transducer. The S_p is expressed as "d" in our article and is a fixed value. The voltage of full-scale deflection (V_{FSD}) is indicated by the voltage meter and is represented by "h" in our article. Assuming that the acceleration/velocity/displacement switch of the charge amplifier Type 2635 is set to a vibration velocity, the signal level corresponding to full-scale deflection (L_{FSD}) is the velocity corresponding to full-scale deflection. The L_{FSD} is expressed as linear velocity (V) in our article. The equation provided in the instruction manual is shown as[1] $L_{FSD} = V_{FSD}/S_p$; that is, $V = h/d$. This is the source formula in our article, on which are derived the formulas for linear acceleration, rotational (angular) velocity, and rotational (angular) acceleration. We assume that the head rotation by an extremely short arc can be thought of as a linear movement, that the velocity and acceleration have a linear correlation to rat body weight, and that the latter is also in a linear correlation to rat brain mass. These approximations are the theoretical basis for the above derivations and make it easier for us to analyze, though not precisely, the complicated mechanical parameters during lateral head rotation.

There may be a lot of aspects regarding the mechanical, behavioral, morphological, and biochemical mechanisms of the DAI model in rats by using lateral head rotation that need to be further studied. We believe that future research will increase understanding of the pathogenesis of DAI. We hope that researchers from different institutions will engage in a cooperative effort to evaluate this new model.

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Pituitary Abscess

TO THE EDITOR: We read with interest the article by Vates, et al. (Vates GE, Berger MS, Wilson CB: Diagnosis and management of pituitary abscess: a review of twenty-four cases. *J Neurosurg 95:*233–241, August, 2001).

Abstract

Object. Pituitary abscess is a rare but serious intrasellar infection. To better determine the salient signs and symptoms that help in making the diagnosis, and to determine the most appropriate treatment, the authors reviewed their experience in a series of 24 patients treated at the University of California at San Francisco.

Methods. Nine of the patients were female and 15 were male, and their mean age was 41.2 years (range 12–71 years). Surprisingly, most patients in our series presented with complaints and physical findings consistent with a pituitary mass, but rarely with evidence of a serious infection. Headache, endocrine abnormalities, and visual changes were the most common clinical indicators; fever, peripheral leukocytosis, and meningismus were present in 33% or fewer of the patients. Imaging tests demonstrated a pituitary mass in all patients, but the features evident on computerized tomography and magnetic resonance studies did not distinguish pituitary abscesses from other, more common intrasellar lesions. Because of the ambiguous clinical features and imaging findings, most abscesses were not diagnosed before treatment; rather, the diagnosis was made during surgical exploration of the sella turcica, when the surgeon encountered a cystic mass containing pus. There were only two deaths in this series (8.3%). Patients presenting with headache and visual changes noted improvement in almost all cases; patients with endocrine dysfunction generally did not recover normal pituitary function, but were easily treated with hormone replacement therapy.

Conclusions. Antibiotic therapy is suggested for patients who have symptoms of sepsis, or for patients in whom specific organisms are identified from cultures obtained during surgery. The transsphenoidal approach is recommended over open craniotomy for surgical drainage.

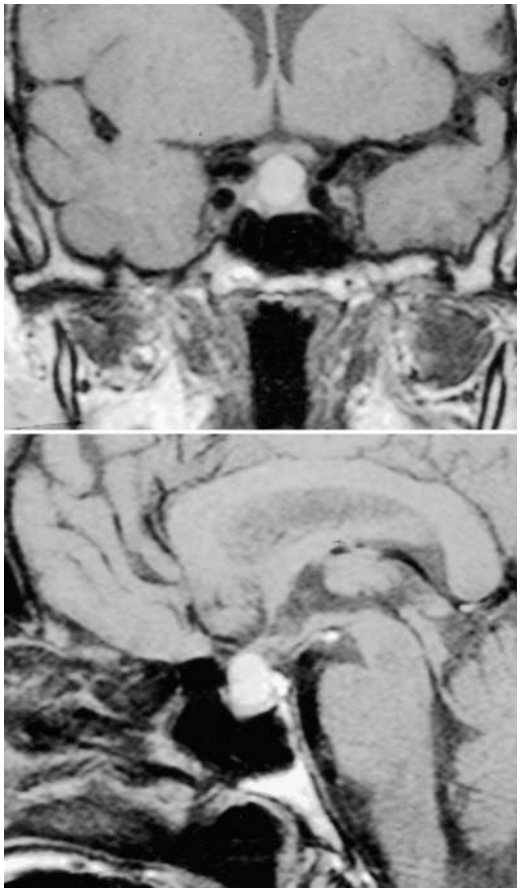

FIG. 1. Sagittal and coronal unenhanced T_1-weighted magnetic resonance images demonstrating the relatively high signal from the Rathke cleft cyst within the sella. Same patient as Figs. 2 and 3.

The authors outline their unit's experience with what constitutes the largest series of pituitary abscesses described in the literature. We would like to query the authors regarding what we consider to be an important technicality; we wish to know how they actually defined and diagnosed pituitary abscess. We believe that up to 10 Rathke cleft cysts were possibly incorporated in their series that did not strictly warrant inclusion. The appearance of this cyst can be misleading when encountered intraoperatively, but in the appropriate clinical context and with histopathological analysis, it can be readily differentiated from a true pituitary abscess of infectious origin. The definition of an ab-