Anatomic Landmarks of the Glossopharyngeal Nerve: A Microsurgical Anatomic Study

To the Editor:

We read with great interest the article by Özveren et al. (3). This study examined 10 cadaveric heads with microsurgical dissection and identified landmarks for the glossopharyngeal nerve (GPhN). This was a helpful study; however, several issues need clarification and adjustment. The authors state in the Results section that the tympanic nerve (Jacobson’s nerve) forms the tympanic plexus. This is partially true, because the nerve contributes to this plexus, as do the caroticotympanic branches of the internal carotid sympathetic plexus and a small branch from the geniculate ganglion (2, 7).

Not described is that this nerve may supply the posterior portion of the external auditory meatus and a small area of skin on the posteromedial aspect of the auricle and adjacent mastoid region that may be separate from fibers traveling in the auricular branch (Arnold’s nerve) of the vagus nerve (2). General somatic afferent fibers stemming from the dura mater of the posterior cranial fossa may also be derived from the GPhN (2). The authors state that the nerve of the carotid sinus (Hering’s sinus nerve) is formed by both the GPhN and vagus nerve. This is the exception and not the rule, because the IXth cranial nerve (Jacobson’s nerve) is beyond the aim of our article. We think that the reason for the controversy was that we had different references than those of Tubbs et al. regarding the functional anatomy of the GPhN, which is beyond the aim of our article. We think that the information of this study will be a helpful guide for every surgeon dealing with this region. However, the majority of their comments are regarding the functional anatomy of the GPhN, which is not clear at several points.

In our article, we mentioned that the tympanic (Jacobson’s) nerve forms the tympanic plexus because it is the major component of the tympanic plexus. The tympanic nerve enters the tympanic cavity and crosses the medial wall of the middle ear as the tympanic plexus. These fibers reenter the cranial vault as the lesser petrosal nerve, only to exit the cranium again and terminate in the otic ganglion (3). In the literature, it has also been stated that the tympanic nerve divides into six branches in the tympanic cavity: two posterior branches for the mucous membrane surrounding the vestibular and cochlear windows; two anterior branches, the caroticotympanic nerve and a tubal branch to the mucosa of the auditory tube; and two superior branches, the deep greater and lesser petrosal nerves (6). Thus, we have stated that the tympanic nerve forms the tympanic plexus.

The descending branch of the hypoglossal nerve (upper root of the ansa cervicalis) leaves the hypoglossal nerve where the latter turns around the occipital artery and descends anterior to or in the sheath of the internal and common carotid arteries. It contains no fibers from the hypoglossal nucleus but only fibers from C1, which constitute the upper root of the ansa cervicalis (10). This nerve runs anterior branches, the caroticotympanic nerve and a tubal branch to the mucosa of the auditory tube; and two superior branches, the deep greater and lesser petrosal nerves (6). Thus, we have stated that the tympanic nerve forms the tympanic plexus.

R. Shane Tubbs
E. George Salter
W. Jerry Oakes
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the practical point of view, we preferred the use of “ansa cervicalis branch of the hypoglossal nerve” in our article.

The infratemporal fossa is an irregular space behind the maxilla (10). However, there is some controversy as to the exact description of the infratemporal fossa. Some authors refer to the infratemporal fossa as the region below the greater wing of the sphenoid bone, lateral to the medial pterygoid muscle and the lateral pterygoid plate (4, 9), as Tubbs et al. pointed out. However, another group of authors describes the infratemporal region as the area under the floor of the middle fossa (1, 5, 8). In this more inclusive definition, the lower cranial nerves, sympathetic plexus, internal carotid artery, and internal jugular vein are included. Considering the major infratemporal surgical approaches, targeting the structures located in this area, we preferred to use the term “infratemporal fossa” for the location of the extracranial part of the GPhN.

Tubbs et al. mentioned in their comment that the somatic afferent fibers from the auricular branch of the GPhN terminate into the spinal trigeminal nucleus. However, connection of the GPhN with the nucleus of the spinal tract of the trigeminal nerve was expressed as a possibility in the classic textbook (10). Therefore, we did not show this nucleus in Figure 12 in our article (7).

In conclusion, we again thank Tubbs et al. for their interest in and thorough analysis of our article. Their critique will motivate the reader to discuss the obscure and controversial subjects regarding the functional anatomy of the GPhN.

M. Faik Özveren
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Elazığ, Turkey

Correspondence

Proton Magnetic Resonance Spectroscopic Imaging Can Predict Length of Survival in Patients with Supratentorial Gliomas

To the Editor:

We read with great interest the article by Kuznetsov et al. (4), which describes their brilliant work in using proton magnetic resonance spectroscopic (1H MRS) characteristics in predicting the length of survival in patients with supratentorial gliomas. It was apparent that their pioneering and well-designed retrospective study has significant clinical impact, because a noninvasive diagnostic modality could potentially predict the patient’s life expectancy in cases of supratentorial gliomas. We strongly believe that 1H MRS will not substitute for the “gold standard” of histopathological examination but will definitely add important preoperative information regarding the biochemical profile of an intracranial glioma. This preoperative information will enable the involved neurosurgeon to be well prepared for the tumor resection by having planned preoperatively any adjunctive treatment modalities.

We would like to add to these authors’ extensive experience some thoughts and observations from our limited experience with regard to the infracranial glioma 1H MRS analysis. Although metabolite ratios have been used extensively by different investigators in the spectroscopic analysis of intracranial tumors, the fact that the concentrations of both metabolites (numerator and denominator of the metabolite ratio used) could change concomitantly makes the interpretation of these changes less accurate, and sometimes drawing any conclusion on the basis of this change may be erratic. We think that the calculation of the absolute concentration of the measured metabolites could eliminate any potential for error and significantly increase the accuracy of the methodology of the present study. There are already commercially available, user-friendly, software packages that could easily be used for such calculations.

Regarding the use of the calculated metabolite ratios, we have found in our series that the choline/creatinine ratio was an accurate and reproducible malignancy marker, whereas the choline/N-acetylaspartate metabolite ratio was a nonreproducible one (1). We have also found that the choline/N-acetylaspartate ratio is nonspecific for histological grading of supratentorial solid astrocytomas (1). On the contrary, we have found that a strong correlation exists between the value of the choline/creatinine ratio and the histological grade of a solid astrocytoma, the higher the ratio, the higher the grade of the studied astrocytoma (1).

Kuznetsov et al. reported that they noticed that in all of their patients with the diagnosis of glioblastoma multiforme, lipid groups were consistently detected (4). This finding is different from our results based on a large prospective clinical study, as well as the results of two other previous investigational groups (2, 3, 5). In our series, lipid groups were detected in only 29% of anaplastic astrocytomas and in 60% of glioblastomas (2). We believe that lipids might represent a marker of malignancy, because in the same series, lipids were detected in
none of the low-grade astrocytomas, pilocytic astrocytomas, and benign tumors such as meningiomas and pituitary adenomas (2); however, we are still far from stating that lipids are detected exclusively in glioblastoma multiforme. On the basis of the current experience, the presence of lipids could confirm malignancy, but their absence cannot rule it out. Another interesting finding in our series was related to the fact that gliomas with no detectable lipids were more vascular and tended to be more hemorrhagic during resection (2). It would be interesting to hear the thoughts of the Montreal Neurological Institute and Hospital group on this issue. We agree with the authors that this interesting observation needs to be investigated further in the future; the lipid concentration of a glioma might provide significant information regarding the behavior of this group of primary intracranial tumors.

The authors reported that the presence of lactate and the calculated lactate/creatine metabolite ratio did not correlate with the patient’s survival (4). They implied that the increased concentration of lactate might represent a highly aggressive glioma using nonaerobic metabolic pathways. This thought seems to be biochemically reasonable, but what would the case be in highly malignant, very aggressive tumor with significant amounts of neovascularization? Could this pathophysiological mechanism potentially alter the concentration of the measured lactate? It is apparent from this excellent retrospective study and other clinical series that a large multi-institutional, prospective clinical study is mandatory for the evaluation of the 1H MRS in the preoperative evaluation of solid, supratentorial tumors.

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Ioannis Karampelas
Macon, Georgia


In Reply:

We agree that there are limitations to the information content of individual MRS resonance intensities or resonance intensity ratios, which is why we developed the approach described, which uses the information available in the pattern of changes across multiple features (1). When relying on “pattern recognition,” it is not important whether one uses ratios or absolute quantification, provided that the pattern is discriminatory.

Absolute quantification might or might not be more discriminatory. It would certainly be more difficult. With the usual sequences used for spectroscopic imaging, absolute quantification would be confounded by heterogeneous relaxation time changes in tumoral tissue that would not be feasible to measure in individual patients.

Our experience with detection of lipids and lactate in brain tumors is based on spectra obtained at a TE of 272 ms using 90-180-180-degree pulses for volume selection (3). Such spectra show a resonance attributed to lipids, which have a short T2 relaxation time, only in the presence of large concentrations of mobile lipids. In our experience, this sequence is more reliable for detection of lactate than stimulated echo sequences, in which quantum coherences can interfere with detection of the lactate signal. In such T2-weighted spectra, detection of mobile lipids is an indication of malignancy, because the lipids reflect necrosis, which is a hallmark of glioblastoma (4). The pathological significance of lactate is less clear. Lactate may be elevated because it accumulates with tissue infarction as tumors outgrow their blood supply. Lactate also may be elevated because of aerobic hyperglycolysis that is not associated with ischemia (2).

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An Easy-to-use Intraoperative Digital Videography, Still Photography, and X-ray-capture System

To the Editor:

We read with interest the recent article by Ogilvy et al. (1). The authors describe a digital videography and still photography system obtained with Macintosh software and hardware connected either to an operative microscope or to any other device with S-video output. The authors describe this system to be of low expense, easy to use, and useful for storing digital data. According to the authors, the total price of the described system is approximately $10,000.
In our department, a similar system has been used for 2 years; a personal computer (PC) AMD Athlon 1300 with DDR 512 Mb random-access memory (RAM), two hard disk drives of 40 Gb, a Matrox RT2500 video card, and a digital video disk (DVD) read-rewriter are used instead of the Macintosh system proposed by Ogilvy. The Matrox RT2500 is a professional video card that captures and edits video in real time with an excellent resolution of 720 × 576 pixels by Adobe Premiere software and Plug Ins, which costs are included in the price of the graphic card.

We propose an implementation of this system. Such implementation has been successfully attempted during the past few months in our department for didactic purposes and for editing professional video: it consists of integrating the above-mentioned system with two DVD Panasonic DMR-HS2 video recorders (Matsushita Electric Industrial Co., Ltd., Tokyo, Japan). One DVD recorder, placed in the operating room, is connected via an S-video switch to either the operating microscope or an external camera (Fig. C1A). The DVD recorder can create a DVD video by recording sequences of surgical procedures onto DVD-R disk (4.7 or 9.4 Gb in DVD-RAM) conforming to DVD video standards. The DVD recorder can also store up to 20 hours of recording (SP mode) on its hard drive. A recorder remote control, sealed into a sterile bag, is used by the surgeon.

All data stored in the operating room are easily transported on a DVD into a separate postprocessing room where, using a second DVD player-recorder connected to the Athlon PC, procedures of professional video editing and capturing of digital images can later be performed (Fig. C1B). Photographs are stored with the PowerDFVD program as standard JPEG files. An example is reported in Figure C2. Definitive video and images are then explored to a final digital support.

In our opinion, this system is very easy to use and is able to store data on DVD, which represents the best digital data support today. Moreover, the presence of a FireWire port on both the Panasonic DVD recorder and the Matrox RT2500 video card allows the proposed system to be adaptable to the digital camera.

Today, the cost of this system is approximately $4200. This system is less expensive than the one proposed by Ogilvy, is stable, and gives excellent output results.

Nicola Di Lorenzo
Antonio Scollato
Paolo Perrini
Florence, Italy


In Reply:

We thank Di Lorenzo et al. for their thoughtful comments regarding our article (1). There is clearly more than one way to skin the digital videography cat. The authors describe a system using less RAM, with a personal computer instead of the Macintosh system we described. They quote the cost of their system to be far less than the cost of the system we described; however, the cost of our system includes a large-screen display panel as well as an additional laptop computer. One of our colleagues was able to achieve a system very similar to ours using simply a laptop computer and a dazzle bridge. This an area in the process of great evolution in terms of how data are collected, edited, and captured for ultimate storage. The main point, as illustrated by Di Lorenzo et al., is that neurosurgeons can become very facile at using off-the-shelf systems...
to perform the task of digital videography and capture of still photography as well as x-rays. We thank them for their comments and commend them for putting together the system they described.

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Benjamin W. Ogilvy
Peter Sztramski
Boston, Massachusetts


Concussion in Professional Football: Reconstruction of Game Impacts and Injuries

To the Editor:
I read with interest the article by Pellman et al. (2). The authors have classified the striking object in this study as the ground or another player’s helmet, shoulder or arm, or leg, knee, or hip. Table 1 of the article gives the relative frequency with which these striking objects were responsible for causing concussion in the struck players. Surprisingly, in each case, only one of the above-mentioned striking objects is considered responsible for the injury. However, it is possible for some of the players who were struck by one of the striking objects, e.g., a helmet, to have consequently lost their balance and fallen to the ground, which, in this case, would become the second striking object. Concussion in such cases would obviously be caused by the additive effects of the two striking objects.

The authors have encouraged measurement of rotational acceleration for experimental research on concussion and helmet development. The measurement of rotational acceleration in the present study has been largely indirect through reconstruction of game impacts (as recorded on conventional video recordings, which give only a side-on view) in laboratory tests with instrumented dummies. It would probably help researchers if a “top” or “axial” view of the rotating head could be obtained by appropriately placed video cameras mounted on cranes or through information provided by satellite imagery. Measurement of rotational acceleration through such an “end-on” view in addition to the side-on views of the helmeted head is likely to be more accurate.

Future studies on the subject would probably also include gathering of real-time data through incorporation of accelerometers in the helmets of the players on the field. As mentioned by Cantu (1) in his comments on the article, earpieces of race car drivers’ helmets already come custom fitted with accelerometers.

The need to protect players of professional football against repeated concussions has been rightly stressed in the article. Research into this aspect of mild traumatic brain injuries can also be aided by information yielded from the helmets fitted with accelerometers. The additive harmful effect of repeated concussions as well as other significant impacts not sufficient to cause concussion may be studied by maintaining a record, over several years, of all the impacts that a study group of players sustain while wearing helmets fitted with accelerometers. By following up the performance of these players, threshold values of the sum total of impacts above which a decline in the player’s performance at the professional level follows may be quantified.

In Reply:
The authors thank Dr. Kundra for his thoughtful input on our reconstruction of National Football League concussions (1). First, he is correct that some of the collisions involve multiple hits. Our analysis focused on the most severe collision if there was a sequence of helmet impacts. In a subsequent publication, we discuss the possible cumulative effects of successive impacts in a collision involving concussion. Second, the camera views of game impacts involve a downward angle on the field, so the motion of the player is seen from a top and side view. This allows a three-dimensional reconstruction of the player’s movement before impact and the helmet’s response in the collision. Third, we are aware of ongoing efforts to instrument race car drivers and the helmets of college football players. The National Football League Committee is looking at these approaches and considering whatever opportunity they might provide in further defining concussion biomechanics. We thank Dr. Kundra for his comments.

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To the Editor:
Pellman et al. (26) link concussion with translational acceleration. Because there has been much speculation about the role of rotational acceleration, it would help if they could establish statistically whether, once translational effects are accounted for, there is any independent relation with rotational acceleration (Fig. 9 suggests that this is not the case).
They stated that their study could not determine the underlying cause of concussion. This theoretical vacuum is unhelpful. Biology has advanced via concepts, not data (24). Sherrington, a Nobel Prize recipient for work on spinal reflexes, above all provided a set of concepts by which the nervous system could be understood (6). In a throwaway sentence in 1906, he stated that knockout punches on the jaw acted on the labyrinthine proprioceptors, abolishing gravity reflexes. Curiously, few (17) have followed up on this idea. Noncerebral labyrinthine concussion systematizes and clarifies previous research, resolving paradoxes. A small amount of this evidence will now be given in the hope that this idea will now be further pursued.

• “It would be logical to assume that, since the ear contains the accelerometers of the body, changes in acceleration produced by head injuries would be most likely to directly damage those structures that are specifically designed to be most affected by this type of energy” (20).
• A previous review (12) found much to support a labyrinthine origin for the postconcussion syndrome (PCS).
• A video study of facial blows in Australian footballers found short-latency concussive convulsions at brainstem level causing transient functional decerebration consistent with a labyrinthine origin for early seizures (13).
• In an experimental mouse model of repetitive head injury (22), the most prominent deficits were vestibular, not neurological motor function in general.
• Concussion in the undrugged rat was always manifested by a convulsion, loss of labyrinthine righting reflexes (17), and probable inner ear damage (29). Seizures commonly arise from the inner ear in rodents (13) and humans (15, 27).
• The vestibular linear acceleration sensory organs drive homeostasis (body metabolism, temperature, cardiovascular regulation, adiposity, circadian rhythms), influence emotional lability, and are implicated in anxiety disorders (11). Physiological effects of centrifugation are caused largely by the increased gravitational, not the rotational or angular, components.
• Diffuse axonal injury was related most reliably to peak changes in velocity (3), the most plausible correlate for brain damage.
• “Concussion is always an acute syndrome with a chronic aftermath” (25). Traumatic brain damage may take a while to develop.
• There is a peripheral “supravestibular syndrome” (25): intense dizziness from specific head or body movements, nystagmus, motion sickness, sudden intolerance to alcohol (as in Ménière’s disease [19]), and sedatives (as in deaf persons with musical hallucinations [14]), associated with tinnitus and not neurological symptoms. Tinnitus after head injury is associated with objective evidence of cochlear hyperactivity (8, 9) and audiosensitivity that correlates with abnormal middle ear muscle reflexes (12) and occurs in decerebrate mammals (28). Vomiting is peripheral (20), not cerebral (25).
• Concussion or PCS also occurs in several conditions with ear damage but absent, trivial, or dubious brain damage. There is no consistent physiological mechanism for brain damage common to head injuries and the following conditions.
   • Explosive blast from air or water (12, 25). Schwab thought that pure blast would not injure the brain but found naval gunfire exceedingly unpleasant to his ears. Yakovlev added that peripheral acoustic vestibular overload could alter brain synapses (28).
   • Electrical injury. Bruyn (7) saw a man in whom lightning had entered the ear via a telephone, hurling him over a desk and causing “cerebral concussion.” Aural but not vestibular symptoms are prominent. This might show that concussion, like motion sickness, depends on a hyperefficient vestibular system.
   • Whiplash. PCS and vestibular test abnormalities are found in most individuals with whiplash or closed-head injuries even when referring physicians fail to show abnormal otoneurological findings, or hearing is normal, or vertigo is absent (29). The incidence of vestibular and disturbed psychic function were the same after neck injury without a head blow and after major brain injury.
   • Nonrotatory axial jarring, as after jumping from a great height (25).
   • Perilymph fistula (12) and motion sickness (23).
   • Spontaneous intracranial hypotension. There are many PCS symptoms (27). Labyrinthine pressure drops, causing endolymphatic hydrops (audiosensitivity, fluctuant deafness, vertigo, vomiting, tinnitus, “blocked ear” [19]).
   • Studies on the causes and structure of the PCS support a labyrinthine origin.
   • There is a prominent inner ear imbalance factor in Gulf War veterans (15), for which there is a plausible cause, labyrinthine hypotension. A similar factor in PCS must surely have a similar cause (not mechanical brain damage).
   • The seven indices of trauma severity that discriminated those with unequivocal brain damage from the recovered group missed those with PCS (21), but disturbances of special senses were more common in the PCS group, implicating head or neck but not brain injury. “Intolerance” but not “impairment” complaints are unrelated to injury severity (5, 18). Auditory hyposensitivity or hypersensitivity (9) or vestibular syndromes (29) are unrelated to brain injury severity.
   • Cognitive, behavioral, affective, and physical symptoms were more common in those with mild rather than moderate or severe traumatic brain injury. Dizziness was the only one of 16 physical symptoms to differentiate those with mild head injury from five other groups (16).
   • Although there is evidence for posttraumatic stress disorders, such psychogenic reactions can clearly be distinguished from PCS. After a nightclub fire (2), posttraumatic neuroses were far more common in those who had not lost consciousness or memory. Nightmares were of early onset in fire victims but of late onset in the head-injured, and then associated with dizziness (1). Significantly, headaches and dizziness that persisted in almost all individuals after closed-head injury were practically absent in fire victims.
Posttraumatic mental symptoms were far more common in those with dizziness but were unrelated to bloody cerebrospinal fluid, cranial fracture, electroencephalographic changes, coma, and amnesia.

- Psychological disturbances were more common in those with vertigo, most likely of peripheral origin in the absence of brainstem pathology and signs (20). Neither correlated significantly with degree of brain injury.
- Audiosensitivity (1), nausea, and dizziness in PCS are often considered of psychological origin, but in a large medical sample (4), they loaded onto an inner ear factor that also included tinnitus and headaches, and not onto the psychological/neurotic or cognitive factors.
- Symptom-based grading systems after sport concussion are better than neurologically derived guidelines. The only one out of 21 symptoms to correlate with three potential indicators of concussion severity was dizziness (10).
- Inner ear symptoms such as audiosensitivity, dizziness, and headache (12, 19, 21) loaded onto the postconcussion factor, not the emotional/vegetative factor, as would be expected if they were psychogenetic (6). These same symptoms, plus trouble following conversation (8, 29), loaded onto the postconcussion (ear) factor and not the factor indicating severity of neurological injury (18).
- Every concussion patient is emotionally labile, with a characteristic but temporary “disturbance of the central vegetative centers in the mid-brain,” mimicking anxiety neurosis (25). The common trigger has now been identified in mice. Head injury selectively damages vestibulomotor functions (22), the same ones defective in anxiety-prone mice (23). When vestibular hair cells were knocked out, autonomic chaos ensued (11).

Surprisingly, Pellman et al. ignored boxing, in which video analyses, especially with overhead cameras, would be very informative. Boxing authorities would welcome the chance of a major positive contribution to neuroscience, rather than simply supplying patients, as they do now. Knockout blows are more likely semisideways punches (12) than uppercuts, supporting Pellman’s highlighting of lateral translational acceleration. Knockouts in boxers and cattle are associated with intense reflex postural overreaction to the blow, suggesting (asymmetrical?) labyrinthine overload (17, 19). Vertigo after head injury is also more common with lateral than frontooccipital blows (29). One would expect broken noses and hands to be more common at the start of fights, in line with stronger punching and greater resistance, and to correlate with velocity. The pattern for concussion is quite different. After subconcussive blows, boxers become unsteady and more susceptible to a knockout. Referees often stop fights in this case. Protective reflexes have fatigued, so when punched, head acceleration is greater.

Labyrinthine concussion is a simple concept vital for biological progress (24). It systematizes and clarifies a wide body of research and is readily falsifiable. The only pre-Darwin British evolutionist was a layman! Leading zoologists did not see the forest for the trees, being distracted by seeming discrepancies. Evolution is amazingly obvious in retrospect. I suspect that the same applies to labyrinthine concussion. 

The first English usage of “stunned” was in 1762: “My stunned ear tangles to the whizzing tide.” The original meaning of stun was “to daze with noise.”

A. G. Gordon
London, England

In Reply:

The authors greatly appreciate the interest shown in our work (1) by Dr. Gordon. His comments regarding possible vestibular mechanisms of concussion certainly position the vestibular system as affected in closed-head injury. However, we believe that the brain itself, not the peripheral vestibular system alone, is significantly affected by the forces documented in this article and that it is the effects on the brain that are the primary cause of concussion. Later articles in this series will document the severe forces acting on the central nervous system in National Football League concussions. We agree with Dr. Gordon that physicians need to be aware of the effects of trauma on the peripheral vestibular system. We also agree with Dr. Gordon that studies on head injuries in boxers are relevant to studies in American football.

Conservative Neurosurgical Management of Intracranial Epidural Abscesses in Children

To the Editor:

We read with great interest the article by Heran et al. (2) in the October 2004 issue of Neurosurgery. Unlike the situation in developing countries, intracranial supplicative disorders secondary to chronic otorhinogenic infections have become exceedingly rare in the developed regions of the world. In the modern era of neurosurgery, with improved antimicrobial therapy and advanced techniques in minimally invasive surgery, it is not unreasonable to treat Third-World diseases with First-World technology and expertise. Furthermore, these authors should be complimented for their report to show that conservative management of a small, focal intracranial pus collection is possible. We have known for a long time that concurrent drainage, at the time of drainage of the sinuses, of small extradural empyemas located in either the frontal (the posterior wall of the frontal sinus is half as thick as its anterior wall) or mastoid (erosion of the tegmen tympani) regions is possible. Therefore, it is logical to contemplate simultaneous drainage of small extradural pus collections by the attending otolaruminal surgeon using endoscopic techniques to treat the primary source of infection (sinuses) and also obtain bacteriological identification of the offending organism for appropriate antimicrobial therapy.

Heran et al. describe a small cohort of children with uncomplicated rhinogenic and otogenic extradural empyemas (cerebrospinal fluid). We think that additional information about patient sex, previous history of chronic otorhinogenic infection, immune status, location of these patients in relation to the base hospital (urban versus rural), infecting organisms, antibiotics used, number of computed tomographic (CT) investigations per patient, and more importantly, duration of hospital stay has not been provided and in our opinion would have improved the value of this article.

In our experience, increasing headaches should be evaluated promptly by neuroimaging to determine an increasing pus collection, development of subdural pus, or more rarely, a bleed into the pus collection because of erosion of a dural vessel. As a rule, we have been able to manage the vast majority of these extradural collections with burr holes, because, in our experience, unlike their subdural counterpart, they generally consist of thin pus and are devoid of loculations. The authors point to the use of CT scanning for these pus collections when, in our opinion, magnetic resonance imaging is a preferable imaging modality to rule out a concurrent small loculated subdural empyema often missed by conventional contrast CT and will prevent an unnecessary durotomy when evacuated surgically.

Finally, we wish to draw the authors’ attention to our work (4), which to date is the largest single institutional experience published on extradural empyemas. Sixty-four of 69 patients (92.8%) with otorhinogenic pathogenesis were less than 20 years old. More important, however, are the 12 patients (15%) who had nonoperative management of their pus collections after surgical eradication of their primary sinus infection. A single patient experienced unique spontaneous drainage of his infratentorial empyema (5), whereas five patients had their small pus collections (three otogenic and two frontal) drained by the attending otolarineological surgeon. The remaining six patients were neurologically intact and apyrexial, such that they were managed on an outpatient basis with oral antibiotics and weekly scans. We think that additional information about patient sex, previous history of chronic otorhinogenic infection, immune status, location of these patients in relation to the base hospital (urban versus rural), infecting organisms, antibiotics used, number of computed tomographic (CT) investigations per patient, and more importantly, duration of hospital stay has not been provided and in our opinion would have improved the value of this article.

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CORRESPONDENCE

In Reply:

We welcome Nathoo and colleagues’ comments regarding our recent article (1), particularly in relation to their seminal work published in Neurosurgery (2). We apologize for not recognizing those patients managed conservatively in their earlier article.

Although the two articles are different in the patient populations reviewed, in a highly selected population of pediatric patients, we have confirmed the conclusions reached by Nadvi and van Dellen in their review of a general population of patients of all ages harboring epidural abscess. Both reports emphasize the importance of eradication of the primary source of infection by medical and surgical, usually otolaryngological, means, obtaining suitable material for microbial identification, and antibiotic administration in a form and for a period of time appropriate to the infecting organism(s). Clinical and radiological monitoring of the abscess/empyema mass effect should be used to determine whether neurosurgical drainage of the epidural space is necessary.

Our series differ with respect to the proposed role of sinus drainage. In none of our Group 2 patients was there preoperative CT evidence of bone destruction that would have implied direct continuity between the infected sinus and the extradural collection. CT scans performed early in the course of treatment revealed the persistence of mass effect without extradural air. We think it unlikely that sinus drainage, endoscopic (in one patient) or open (in one patient) or for aspiration of pus for culture (two patients), resulted in concurrent evacuation of the epidural pus. In contrast, in the Durban series, one patient drained spontaneously, and the collections in five patients were drained by the otorhinological surgeons. The remaining six patients, treated nonoperatively, would seem to have been similar to those described in our article. This suggests that although in some patients, the sinus procedure may evacuate the extradural collection via a bony dehiscence, a sinus wall dehiscence is not necessary for successful conservative neurosurgical management.

Our article addressed only patients with epidural abscess formation, and those with intradural collections or brain abscess were excluded. We did not screen the reported patients with magnetic resonance imaging looking for intradural suppurative, and as reported, no patient developed this complication while receiving treatment or after treatment. Had it been the case that patients were not responding to treatment and repeat CT imaging suggested intradural spread, brain magnetic resonance imaging would have been performed for further evaluation.

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In their recent article on the treatment of poor-grade aneurysmal subarachnoid hemorrhage (SAH) patients (i.e., World Federation of Neurosurgical Societies [WFNS] Grades IV and V), Laidlaw and Siu (1) have made some very important points. They showed that a favorable outcome may be obtained by aggressive urgent surgery, independent of the age of the patient. These data should encourage neurosurgeons to treat all patients with aneurysmal SAH regardless of clinical grade and not to deny treatment to patients but rather to give them the benefit of any doubt regarding the ability to recover from SAH. This study confirms the observations of previous investigations that surgery is more demanding in poor-grade patients than in good-grade patients and that despite ultra-early surgery, rebleeding remains a frequent and often fatal complication. Therefore, it becomes difficult to justify simple observation of patients with severe SAH, as is often advocated (5). The results of this study are slightly favorable compared with previous surgical series in which an aggressive treatment was advocated (2).

We adopted a management protocol consisting of early occlusion of the aneurysm and aggressive treatment of intra-
cranial hypertension and vasospasm, and we published the results of a prospective study with early endovascular treatment of very-poor-grade patients (i.e., WFNS Grade 5 only) (4). During a 12-month period, 20 patients were admitted after aneurysmal SAH in very poor neurological condition, classified as WFNS Grade 5 after resuscitation. Nine patients were excluded from the study, either because of an important intracerebral hematoma that was evacuated surgically (n = 4) or because of absent brainstem reflexes (n = 5). After a mean follow-up of 18 months, more than half of the patients had a favorable outcome (Glasgow Outcome Scale score of good or moderate disability), and the mortality rate was 18%. These figures compare favorably with the results of the present study (favorable outcome in 40% of patients and a mortality rate of 45%). In both series, the incidence of survivors in a persistent vegetative state is low.

There are some important remarks to be made. First, the number of patients is much larger in the series of Laidlaw and Siu, making their results more reliable. However, the number of patients actually treated per year is comparable in both series (15 patients), as is the proportion of patients excluded from treatment for different reasons (25%). We believe, therefore, that it is justified to compare the two studies. Next, the follow-up period in the series of Laidlaw and Siu is only 3 months, whereas in our series, the follow-up was at least 12 months. A better outcome can be expected at a later follow-up, because patients treated for SAH continue to improve after 3 months. Conversely, we reported on the results of WFNS Grade V patients only, whereas a significant number of patients in the series of Laidlaw and Siu were in Grade IV. It is well known that the outcome of patients with SAH is inversely correlated to clinical grade, so the subgroup of patients in Grade IV may have influenced their results in a positive way.

We agree with the authors that the International Subarachnoid Aneurysm Trial does not provide enough evidence to support a potential benefit of coiling over surgery in poor-grade patients, because most patients included in the study were in good clinical grade (3). However, compared with endovascular treatment, surgery in poor-grade patients is likely to cause further damage to the already edematous brain by retraction and manipulation of cerebral vessels, even with the use of modern neuroanesthesia and microsurgical techniques to minimize the need for retraction. We therefore prefer endovascular treatment for all very-poor-grade patients when the configuration of the aneurysm allows endovascular coil placement, and we are encouraged by the results we obtained (4). Endovascular treatment of the aneurysm has several other advantages. It often allows earlier occlusion of the aneurysm; in our series, it could be performed immediately after completion and review of diagnostic angiography. The technique seems both logistically easier and faster than ultraearly surgery. A further advantage is the possibility of performing balloon angioplasty and intra-arterial infusion of papaverine to treat early vasospasm immediately after occlusion of the aneurysm. Of course, further studies are needed, and we agree that this group of patients would be an interesting population to study in a prospective, randomized trial to compare the use of microsurgical versus endovascular techniques.

In Reply:

We thank Drs van Loon et al. for their thoughtful comments on our article (1). As they have pointed out, their publication demonstrated good results with endovascular treatment for Grade 5 SAH patients (5). We agree that there is now a growing body of published evidence that provides a strong rational basis for the aggressive management of these poor-grade patients (1, 3, 5).

However, we must emphasize again that our series was a surgical series; this was because at the time of treatment, we did not have endovascular facilities. We now have outstanding neuroradiology endovascular support, and we are strong advocates of endovascular coiling of suitable aneurysms, regardless of the grade.

Although we have some reservations regarding the findings of the International Subarachnoid Aneurysm Trial (4), our recent practice has been to discuss all aneurysm cases with the endovascular radiologists and to use endovascular coiling techniques on all aneurysms in which the anatomic configuration of the aneurysm would seem to be suitable for this technique. However, in cases in which the anatomy of the aneurysm does not seem optimal for good endovascular obliteration, we perform ultraearly surgery wherever possible.

We consider that the decision as to whether surgery or endovascular techniques should be used should be based primarily on the anatomy of the aneurysm and the feeding vessels, not on the age or clinical grade of the patient. The risks of surgery, as pointed out by van Loon et al., are well known, and we agree that surgery for poor-grade patients may be more demanding than for good-grade patients. However, our personal observations with ultraearly surgery is that the edematous brain is not commonly a problem, even in poor-grade patients, and good brain relaxation can usually be achieved with cerebrospinal fluid drainage at the time of surgery. Our anecdotal impression is that the edematous brain
is much more common in poor-grade patients who present late and therefore are not managed with ultraearly surgery. This suggests to us that this brain swelling is most likely a result of ischemia caused by prolonged untreated intracranial hypertension. Therefore, with ultraearly surgery, good anesthesia, cerebrospinal fluid drainage, and meticulous microsurgical technique, the vast majority of poor-grade patients treated with microsurgery experience minimal brain retraction and should not demonstrate any postoperative contusions or local injury. It is important to note that Le Roux et al., when comparing good-grade and poor-grade patients treated surgically (early surgery, <3 days, not ultraearly), did note more brain swelling (often secondary to intracerebral hemorrhage) in the poor-grade group but did not note any higher incidence of contusions or other surgical complications (2).

The results of our series support our premise that ultraearly surgery for poor-grade patients can provide good results that can be favorably compared with current endovascular series. Although it is definitely not our purpose to suggest that surgery is better than coiling for these patients, we would point out that the widely held opinion that all poor-grade (and elderly) patients are better treated with endovascular techniques similarly cannot be supported by the current published data. In conjunction with our endovascular radiologists, we decide whether to use endovascular or microsurgical treatment primarily on the basis of anatomic obliteration of the aneurysm without arterial compromise. Such a decision must be made according to the vascular anatomy and aneurysm conformation, and the grade and age of the patient would seem to us not to be significant selection criteria. We would hope that the results from our study would encourage both cerebrovascular surgeons and endovascular surgeons to consider microsurgical techniques in poor-grade patients in whom the anatomy of the aneurysm does not seem optimal for endovascular coiling. We also look forward to further studies on this group of patients and stress the need for close cooperation between endovascular and cerebrovascular surgeons.

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Neurosurgery appears to be more widespread than that for Parkinson DBS and tissue destruction. The genesis of essential tremor again speaks to a difference in potential mechanism between subpopulations. The effectiveness of lesioning in these cases (5, 7). If heterogeneous, some of the variability may be identified by subpopulations. The effectiveness of lesioning in these cases again speaks to a difference in potential mechanism between DBS and tissue destruction. The genesis of essential tremor appears to be more widespread than that for Parkinson’s disease (8), and as such there may be more circuity involved and hence a lower likelihood of successful tremor control. The therapeutic target area appears to be relatively tight, with only a 2-mm variance before an increased failure rate. Such as a small degree of variance may account for some of the variability in therapeutic effectiveness but certainly not for all of it. Thus, although the anatomic placement may be perfect, the physiological placement may be quite imperfect. The authors are quite correct in cautioning that these results pertain to essential tremor and not necessarily to Parkinson’s disease. Clearly, there are differences in the electrophysiological effects of DBS on parkinsonian tremor versus those of essential tremor (13, 14).

Given this quality of anatomic information, one may question whether there is, in fact, a need for microelectrode recordings. Clearly, there are a number of experts who do not perform microelectrode recordings but simply use the robust electrophysiological information provided by the thalamus to establish correct placement of the DBS lead. Using the same electrophysiology should allow one to make appropriate adjustments should there be a translational error between the intended target and the actual target. I used such techniques early in my career, and I know that these can be extremely successful. Reasons to continue using the microelectrode, of course, relate to determining the target as an electrophysiological target and making any necessary anatomic corrections (it is far better to make two or three microelectrode passes than two or three DBS passes, which can convert a microlesion effect into a macrolesion effect), as well as the continued need for research into the understanding of brain pathology to advance the effectiveness of our surgical therapeutic technology.

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