assumption with regard to the distribution of L.

We should not lose sight of the variability in years of life lost. It can be shown that the variance of the years lost, under the gamma model, is

$$\sigma^2 + \frac{1}{r^2} - \frac{1}{r^2}\left(1 + \frac{\sigma^2 r}{L}\right)^{-2L^2/\sigma^2} - \frac{2L}{r}\left(1 + \frac{\sigma^2 r}{L}\right)^{-L^2/\sigma^2}.$$

For the example that Hutchinson gives with a life expectancy of 15 years, the standard deviation (the square root of the variance) of lifetime loss is 5.8 years. This is far larger than the expected lifetime loss of 2.5 years, due to the highly skewed distribution. Note that the standard deviation of lifetime loss (5.8) with the gamma model is ten times larger than the difference between our formula for the lifetime loss (2.5) and that of Hutchinson (1.9).

In the face of this variance of years lost under the gamma model, should we be focusing just on expected life lost? There are other criteria that might be more important. For instance, the patient might be more interested in living until the year 2000. In this case, comparing the probabilities of surviving 2.5 years might be a more meaningful basis for deciding whether to operate or not. This is quite easy to work out. Suppose P is the probability that the time to a natural death exceeds t years. Then if surgery is carried out, the probability of surviving t years is simply $0.935P$. If surgery is not carried out, the probability is $(e^{-\pi} + 0.27(1 - e^{-\pi}))P$. For the above gamma model, the probability of surviving to the year 2000 is 92.5% if surgery is carried out, and 95.4% if not. In this case, the patient might prefer not to have surgery, whereas working with expected lifetime loss, the patient might prefer surgery (15 x 0.065 = 1 year lost) than not (1.8 years lost from [2]). In general, the decision to operate does not depend on P nor any model for the natural lifetime. It can be shown that if the patient wishes to maximize the chances of living more than $t = 4.66$ years, that is beyond the spring of 2002, then surgery is preferable.

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To the Editor:

Early Seizures After Closed Head Injury

Lee et al.[1] studied seizures within a week of closed head injury, but ignored the likely trigger for these fits, the vestibular labyrinth. They were unrelated to CT scans, Glasgow Coma Score and 6-month neurological status: mortality was actually lower with seizures. This is decisive evidence against their cortical origin.

A recent study[2] on concussive convulsions in rugby players is relevant. Again, there was no evidence of brain damage: outcome was excellent. Fits started within a second, too short for a vascular or reflex cerebral vascular ischemic mechanism. The authors proposed "transient functional decerebration", analogous to convulsive syncope.

If this is the best explanation neurologists can produce, then it is surely time to review a simple otological explanation,[3] only a part of the evidence for which can be quoted here. Sherrington was probably the first to suggest that boxing knockouts were of vestibular origin. The postconcussion syndrome, where otovestibular symptoms are prominent, is unrelated to brain damage, whereas there is much objective evidence for labyrinthine damage from closed head injury. Early seizures were commoner in pedestrians and after falls than in car accidents,[1] suggesting that contact with hard unyielding surfaces was the relevant factor, causing deceleration overload on vestibular transducers. Studies of "cortical blindness" in rugby players also indicate that a labyrinthine reflex is involved, not damage to the occipital cortices.[3] Direct evidence of premonitory vestibular hyperexcitability was found in experimental syncope.[4] EEGs in vertiginous patients clearly correlate with vertigo of peripheral not central origin.[5] In fact it may be irritable or disinhibited vestibular function which generates abnormal EEGs, simulating or even causing "temporal lobe" epilepsy. The only objection to this theory (from Ojala et al.[5]) was that afferent sensory information is not large enough to influence EEG recordings from the cortex. However, the large animal literature on audiogenic seizures clearly refutes this objection. Four quite different cochlear insults from congenital deafness, hypothyroidism, ototoxic drugs and acoustic trauma during a critical period all predispose to sound-induced convulsions. Irritative rather than destructive cochlear lesions seem to be necessary,[6] and the abnormal activity is amplified at the inferior colliculus. Higher parts of the brain are not directly involved.

In summary, the labyrinth is implicated in all the curious phenomena after closed head injury not attributable to brain damage – unconsciousness, early fits, EEG spiking, transient blindness, postconcussional syndrome. Occam, for one, should approve this theory.

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Reply:

Dr. Gordon has long posited that labyrinthine dysfunction is the cause of many incompletely understood neurologic, neuropsychiatric, and neurophysiologic phenomena, including epilepsy.[1] His suggestion that so-called "concussive convulsions" may be otovestibular in origin is certainly tenable. However, the latter events have been shown to occur invariably within 2 seconds of impact,[2] and thus represent a nosologic entity different from the early post-traumatic seizures described by Lee et al.,[3] which did not occur until more than 24 hours after head injury in a majority (65%) of patients. Dr. Gordon misleadingly links the universal findings of no structural brain damage and excellent outcome after concussive convulsions[2] to the lack of correlation between CT abnormalities, 6-month neurological outcome and occurrence of early seizures in the study by Lee et al.[3] as evidence against a cortical origin for early post-traumatic seizures. In fact 66% of patients with early seizures

had CT evidence of cerebral parenchymal damage and fully 63% had a 6-month outcome of severe disability, vegetative state or death.[3]

Some partial epileptic disorders may indeed be related to peripheral vestibular or cochlear insults, presumably through aberrant cortical reorganization, but would need to develop over a much longer period than the early seizures under discussion, and would (like their associated EEG abnormalities) represent, at that time, direct cortical involvement. This idea remains largely hypothetical. In contrast, focal cortical epileptic discharges causing symptomatic vertigo (or auditory hallucinations) have been demonstrated numerous times by direct brain recording and/or stimulation.[4-6]

The labyrinth may well be implicated in some curious neurologic phenomena. There is, however, no reason to invoke a labyrinthine etiology for the majority of early post-traumatic seizures.

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To the Editor:

Musical Hallucinosis With Brainstem Lesions

In 1994[1] I stated that there was no known cause of musical hallucinations (MHs) due to a neurological as opposed to otological lesion, and challenged neurologists to find such a case. Douen & Bourque[2] report a man with MHs of allegedly brainstem origin. Far from being a pure example of this, their case has 3 of the 4 features disqualifying a non-otological neurological case, i.e.: Drugs were not excluded – the man was alcoholic. Alcohol withdrawal triggers MHs from normal ears, even more so if abnormal. He presented with Meniere symptoms (nausea, vomiting, imbalance).

Deafness of unproven brainstem origin occurred. A more peripheral lesion was far more likely, since: 1) There was vestibular involvement, with nystagmus to the right; 2) Four adjacent cranial nerves were also involved; 3) Latent ear infection was a more plausible cause for the homolateral cerebellar abscess and circumscribed meningitis than bloodborne listeria; 4) The limited audiological testing showed speech discrimination apparently poorer than expected from the audiogram. This indicates a neural deafness, the usual cause for which is eighth nerve disorder; 5) As noted,[2] all ten prior cases of "brainstem" auditory hallucinations (AHs) had hearing loss ipsilateral to the AH. In none had sufficient audiological testing excluded a peripheral cause.

Douen & Bourque stated:[2] "Clearly, AHs which include verbal content or recognition of a specific melodic line must involve activation of cerebral cortical areas." If it is implied that AHs must be generated in those areas, then this needs critical discussion. Just because AHs end up in the brain, it doesn't mean they started there.

Douen & Bourque wrongly state[2] there is no evidence for a peripheral generator of AHs. Supposedly spontaneous or random cochlear nucleus activity is of carotid origin[3] – the noise of blood flow being transduced into cochlear electrical activity. Artistic creativity in composers[4] and in some poets seems to depend on MHs from hypersensitive ears. Even if one rejects this specific mechanism, it is clear that they really transcribed rather than composed, as music, even whole pieces, rose unbidden from the unconscious, consistent with a subcortical generator. A Heartsongs CD is commercially available on which real electrocardiograms generated melodies;[5] musical composition may involve "the recreation by the mind of the body's own naturally complex rhythms and frequencies". Even cochleas may be superfluous, as a temporal code in the auditory nerve alone is capable of defining musical pitch.[6] Variation in pulse rate on single bipolar electrodes can in totally deaf subjects result in pitch changes sufficiently salient to support musical interval perception.

The topic of insight shows how muddle and illogical current formulations of AHs are. Douen & Bourque[2] say their patient's "preserved insight and clear sensorium establish that these are hallucinations, not... tinnitus". Some definitions of AH use insight for the exact opposite purpose: if patients say they hear noises in their ears they are said to have tinnitus, whereas if they hear similar noises from non-existent environmental objects, they have AHs. Also, given that people with normal minds and brains but defective ears can have AHs that they falsely believe are real, it is not odd that in their case of alleged brainstem origin, insight was retained? This is not an isolated occurrence. Berrios[7] found that insight into MHs correlated with brain disease, not ear disease. In other words, the more MHs were attributed to brain disease, the more insight the patients had. Incidentally, Berrios was the main source for Douen & Bourque's assertion of a direct cerebral role for AHs. However, Berrios, despite his historical expertise, only found 46 cases of MH to review, 31 of whom were definitely deaf, strongly indicating otherwise. If brain lesions cause MHs, where are these cases? Douen & Bourque suspect psychiatric disorder for AHs with impaired insight. All the evidence points to insightlessness, in psychotics or others, being of otological not neurological origin.[4]

Douen & Bourque propose[2] that AHs of aural or brainstem origin result from deafferentation. This ignores tinnitus, of which many with MHs complain bitterly (e.g., Margery Kempe, Luther, Rousseau, Schumann).[4] They do not say if their patient heard tinnitus (e.g., buzzing or humming). Confusingly, Berrios[7] labeled all such sounds (as occur in ENT patients) hallucinations, consistent with most psychiatric definitions (perceptions without objects).

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