time lost from aneurysm rupture is evidently $x - t$ (provided $x > t$). Thus the expected lifetime lost is

$$\int_{x=0}^{x=\infty}\int_{t=0}^{t=x}(x-t)\,re^{-rt}f(x)dt\,dx$$
$$=\int_0^\infty\left(x-\frac{1}{r}+\frac{1}{r}e^{-rx}\right)f(x)dx.$$

Unfortunately, therefore, we need to know the whole distribution of lifetimes, not just a few parameters like the mean and standard deviation, in order to calculate the answer.

We can, if we wish, write the answer in a compact form. Define

$$\ell(s) \text{ by } \ell(s)=\int_0^\infty e^{-sx}f(x)\,dx.$$

This is called the Laplace transform of $f(x)$. Then the expected lifetime lost is

$$L-\frac{1}{r}+\frac{1}{r}\ell(r). \qquad (1)$$

Gamma Distribution of Lifetimes

To obtain something that is usable, we might be willing to assume the distribution has some convenient mathematical form. The gamma distribution may be familiar to readers. It is a skewed distribution having probability density proportional to $x^{\alpha-1}e^{-x/\beta}$. The Laplace transform takes a simple form, $(1+\beta r)^{-\alpha}$. Hence the expected lifetime lost is $L - r^{-1} + r^{-1}(1+\beta r)^{-\alpha}$. The parameters α and β of the gamma distribution may be written in terms of its mean L and s.d. α as follows: $\beta = \sigma^2/L$ and $\alpha = L^2/\sigma^2$. Consequently, the expected lifetime lost is

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

This is simple enough for use on a hand-held calculator.

A special case of the gamma distribution is the exponential. In this case, $\alpha = 1$ and $\sigma = L$. Beck et al.[2,3] have argued for its usefulness in the present context. For this special case, [2] simplifies to

$$\frac{rL^2}{1+rL}. \qquad (3)$$

To take $\sigma = L$ may be appropriate for patients whose life expectancy is short (because of age or conditions other than the aneurysm), but otherwise [3] will overstate the loss of lifetime.

Leblanc and Worsley

Here, I will make explicit why Leblanc and Worsley's method overcorrects for rL not being small compared to unity. Suppose that anyone not suffering an aneurysm rupture lives for exactly time L. Further, suppose aneurysm rupture is extremely rare. (That is, we are assuming that both σ/L and rL are close to zero.) Then the proportion of people suffering aneurysm rupture is rL, on average this happens midway through their life so they lose $L/2$ years of life each, and the average over the whole population of years of life lost is $rL^2/2$. Now, if rL is not small, the proportion of people suffering aneurysm rupture is not rL, but $1 - e^{-rL}$. Leblanc and Worsley have this. (They actually write $1 - (1 - r)^L$ instead, but the difference is unimportant.) But it is not now correct to say the average over the whole population of years of life lost is $(1 - e^{-rL})L/2$.

For a proportion $r\,e^{-rt}\,dt$ of people, their aneurysm ruptures between time t and time $t+dt$ (for small dt). The length of life they lose is $L-t$. The average for the population is $\int_0^L re^{-rt}(L-t)\,dt$, which works out to be $L - r^{-1} + r^{-1}e^{-rL}$. Expanding the exponential as an infinite series in rL, we get $\frac{1}{2}rL^2(1-\frac{1}{2}rL + \ldots)$. In contrast, Leblanc and Worsley's expression $\frac{1}{2}L(1 - e^{-rL})$ becomes $\frac{1}{2}rL^2(1 - \frac{1}{2}rL + \ldots)$. Because the term that multiplies $\frac{1}{2}rL^2$ is approximately $1 - \frac{1}{2}rL$ rather than $1 - \frac{1}{2}rL$, the correction is greater in magnitude than it should be.

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Reply from the Authors:

Hutchinson challenges our assumption that aneurysm "rupture will occur, on the average, when half the life expectancy has expired". As we stated in our paper this is true if "the annual rate of rupture is constant over the patient's life expectancy so that rupture will occur, on average, when half the life expectancy has expired if 'r' (the annual risk of rupture) is small".[1] This point was previously made in our paper in *Neurosurgery* addressing angiographic screening and elective surgery of familial cerebral aneurysms, and by Levey et al. in their paper addressing *Occult Intracranial Aneurysms and Polycystic Kidney Disease.*[2,3] The detailed derivation and the implications of this assumption are detailed in the Appendix to our paper in *Neurosurgery* and were referenced in the paper in question in the *Canadian Journal of Neurological Sciences.*[1,2]

The reader should note that our analysis was made conditional on the observed natural lifetime L. We made no assumptions about the distribution of L. What our results tell the patient and the surgeon is the expected years of life lost *if* the patient's lifetime is L. The patient and the surgeon are then free to average these results over whatever distribution the lifetimes might have, be it a gamma distribution (as Hutchinson has assumed, to get [2]), or a more realistic distribution taken from life tables that might depend on age, sex, health, smoking habits, weight, cholesterol level, daily exercises, medical history, etc. (to get [1]). The patient and the surgeon are free to choose this. The *expected* years of life lost, under the gamma distribution, is usually greater than the *conditional* years of life lost evaluated at L - *life expectancy* because the conditional years of life lost is concave in L. This is the essence of Hutchinson's comment. Whether one chooses to use our formula or Hutchinson's depends on his or her choice of assumption with regard to the distribution of L.

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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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1. Leblanc R, Worsley KJ. Surgery of unruptured, asymptomatic aneurysms: a decision analysis. Can J Neurol Sci 1995; 22: 30-35.
2. Leblanc R, Worsley KJ, Melanson D, Tampieri D. Aniographic screening and elective surgery of familial cerebral aneurysms: a decision analysis. Neurosurgery 1994; 5: 9-14.
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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.

A.G. Gordon
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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