ly, the harmful effects of indiscriminate application of medical science.

All experienced and thoughtful physicians are or should be aware that medical intervention can kill as well as cure, disable as well as benefit. The likelihood of rib fractures from chest compression, barotrauma from positive-pressure ventilation, and renal failure from drug use increases markedly as age and chronic disability increase. If 30% of a mixed population survive CPR, 70% do not, and these 70% are forced to die more miserably because of well intentioned resuscitative measures. The percentage who are subjected in their last moments to physical assault is even higher among older individuals.

Indiscriminate resuscitation of aged patients creates needless suffering. It is not for this that we are physicians.

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Dysphagia due to cervical spine osteophytes

Yee and colleagues' paper on dysphagia due to cervical spine osteophytes (*Can Med Assoc J* 1985; 132: 810–812) has prompted me to describe a similar case.

My patient, a 55-year-old white man, presented with an unusual symptom, difficulty breathing while he was lying down, as well as dysphagia. The dysphagia had been worsening gradually for 5 years, but the respiratory difficulty had begun only 1 month before his presentation. Yee and colleagues include stridor as a possible symptom, but they do not mention the danger that stridor presents to adequate breathing during sleep.

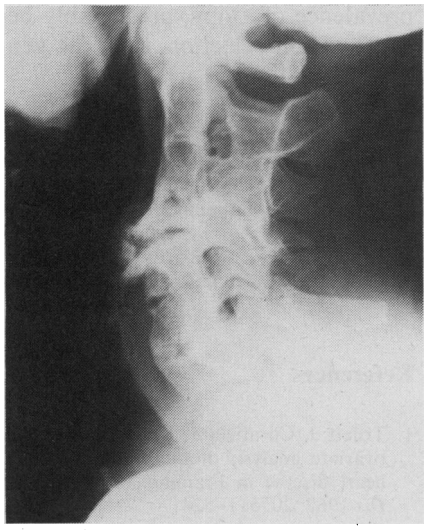

Fig. 1—Osteophyte anterior to disc space between fourth and fifth cervical vertebrae.

In my patient plain x-ray films demonstrated multiple bony congenital anomalies in the cervicobrachial region, including "blocking" of the cervical vertebral bodies from the odontoid process of the axis to C4 inclusive and from C5 to C7 inclusive, and a large osteophyte anterior to a disc space between C4 and C5 (Fig. 1). The cervical spinal canal was enlarged. A myelogram confirmed the existence of a large thecal sac and showed only mild anterior identation of the contrast column at the level of the C4–5 disc. In addition, there were clinically less significant bony abnormalities in the left cervicoclavicular area, which suggests a variant of Klippel–Feil syndrome. I presume that the restriction of the cervical spine's mobility to the C4–5 level resulted in slowly progressive, reactive anterior hyperostosis, which eventually produced symptoms.

The osteophyte was excised, and an anterior C4–5 discectomy, without fusion, was performed. The operation relieved the patient's symptoms. I am aware of Yee and colleagues' warning that the problem may recur in my patient, particularly because the C4–5 interspace is still the only area that is mobile in this patient's cervical spine.

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Family history of allergy and skin test reactivity

The study by Vedal and colleagues (*Can Med Assoc J* 1985; 132: 34–37) was of interest to me as an occupational physician.

The authors stress the importance of a family history of allergy in predicting atopy in workers, when in fact this history provides little additional information. The odds of skin test reactivity found by the authors (4.76 given a personal history of allergy and 2.06 given a family history of allergy) are larger than the relative risks that could have been derived from the same data (3.3 and 1.8 respectively). Moreover, it should be recognized that a high relative risk cannot be equated with a high positive predictive value.[1] In fact, knowledge of a family history of allergy increased the likelihood of atopy only from 39% to 47%, an increase to which I would attach little clinical significance.

As it is often more important to rule out atopy than to diagnose it, the authors also examined the negative predictive value of a history of allergy. They found that there was an 88% likelihood of nonatopy when there was no personal history of allergy and a 90% likelihood of nonatopy when there was also no family history of allergy. Knowledge of the absence of a family history of allergy thus does not add to the negative predictive value to any significant degree.

The physician or employer wishing to predict atopy in workers should be aware that knowledge of a personal history of allergy is likely to be misleading more often than it is helpful. When there is a family history of allergy as well, prediction of atopy is still likely to be incorrect about 50% of the time.

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Vedal and colleagues' study appears to have been inappropriately analysed.

The title and substance of the article make predictive inferences. In a prospective study such inferences re-

quire calculation of relative risk. If the authors had used a case-control design and the outcome was rare (e.g., a prevalence rate of less than 5%), calculation of an odds ratio might have been an acceptable estimate of relative risk. However, the study was a cross-sectional prevalence survey of a population in which immediate skin test reactivity was common. Because the factor examined was a family history of allergy, a predictive relation was assumed between this and atopy. In this sense the study could be considered to have a temporal component.

The authors chose to analyse their results as though the study had a case-control design. The odds ratio gives an overestimate of the true relative risk in this instance because the prevalence of atopy was so high. This point can be illustrated by a two-by-two table (Table I). Only in rare diseases can a and c be ignored in the relative risk denominator. Ignoring a and c in the investigation of a common disorder, as in this study, artificially deflates the denominator and produces spuriously high relative risk estimates (odds ratios). In addition, because the data were gathered in a cross-sectional survey, calculation of relative risk can be made directly and should perhaps be termed "relative probability". All the odds ratios given by Vidal and colleagues are, accordingly, inflated estimates of relative risk.

Application of more appropriate calculations to the data reveals that the relative probability of skin test reactivity was 1.8 (not 2.06) times higher in the workers with a family history of allergy than in those without and 3.30 (not 4.76) times higher in those with a personal history of allergy than in those without. The workers with a family history of allergy were 1.65 (not 1.91) times more likely than those without to have a personal history of allergy. These revised calculations are still of some practical interest, although the fact that the hypothesis was assessed among workers in an allergy-prone population does not permit extrapolation to the general population.

Table I—Two-by-two table

Predictor	Outcome +	Outcome −
+	a	b
−	c	d

Odds ratio = $\frac{a \times d}{b \times c}$ or $\frac{a}{b} \div \frac{c}{d}$

Relative risk = $\frac{a}{a+b} \div \frac{c}{c+d}$

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[Vedal and colleagues reply:]

We appreciate the interest in our paper. In response to Campin, we presented a table of predictive values in our article (Table V). In a worker with a personal history of allergy, knowledge of a family history of allergy distinguishes a risk of atopy of 1/3 from a risk of 1/2. Also, absence of a personal history of allergy, regardless of a family history, makes atopy unlikely (probability of 10% to 16%). This hardly makes knowledge of a personal history "misleading". It only shows that its negative predictive value is much better than its positive predictive value. If there is any usefulness, then, to knowing of a family history of allergy in predicting atopy it is within the setting of a personal history of allergy. Even then, with the presence of both a personal and a family history of allergy, the probability of being correct is only 50%.

We did not "stress" the importance of a family history of allergy but stated that "the presence or absence of a family history of allergy should not be used to rule in or rule out atopy".

In response to White, it is well known that the odds ratio gives an overestimate of the prevalence ratio (relative risk), especially when the outcome of interest is not rare. This is important when one is interested primarily in estimating the prevalence ratio. However, the odds ratio is itself a measure of association. White mistakenly implies that use of the odds ratio should be limited to case-control studies, perhaps because the odds ratio is the only measure of association that can be meaningfully estimated from such studies. We agree with White to the extent that the prevalence ratio is probably a more "natural" measure of association than the odds ratio in a cross-sectional study and allows one to speak in terms of the ratio of probabilities rather than of odds. We chose to use the odds ratio, however, because we made use of the multiple logistic regression model in determining whether a family history of allergy was predictive of atopy independent of its associations with both a personal history of allergy and age. Because coefficients from the logistic model give estimates of odds ratios, we chose to present odds ratios throughout the paper to be consistent and to allow a comparison of the association before and after adjusting for the other predictors. Since the raw data were presented in the paper, it is possible, if desired, to calculate the prevalence ratios.

We are in good company in using odds ratios in a non-case-control study. White states that a prospective study requires calculation of relative risk. However, in the well known Framingham study,[1] a prospective study, the logistic model and odds ratios were correctly used as measures of association.

Finally, one might hesitate to extrapolate our findings to a general population since our population consisted of workers and therefore, to some extent, was self-selected. However, it was not "allergy-prone" and may, in fact, have had a lower prevalence of atopy, presumably because of self-selection, than the general population.[2]

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