

The law allows a temporary solution to the problem of providing professional liability insurance coverage to physicians throughout the state, but far more important the law insures uninterrupted delivery of medical treatment and patient care for all residents of the Commonwealth.

The Massachusetts Medical Society stands ready to co-operate, as we have in the recent past, with all interested organizations and individuals to work for a more permanent solution to a most difficult problem. The welfare of all of our patients is foremost.

The Society is hopeful that the new law will deal stringently with incompetent medical practitioners and that frivolous and non-meritorious malpractice lawsuits will be reduced through the new screening tribunal.

As President of the Society, I can assure all people of this Commonwealth that the officers, members, staff and resources of this organization will be made available to the new study commission that will conduct an ongoing evaluation of this new legislation. We will assist the members of this Commission in whatever way or means possible.

$P(D|T)$  is the probability that the patient has the disease after the test result.  $P(T|D)$  is the probability of the test result if the patient has the disease, and  $P(T|\bar{D})$  is the probability of the test result if the patient does not have the disease. With this terminology the usefulness of both positive and negative test results can be assessed. A line drawn from  $P(D)$  on the right of Figure 1 through the ratio of  $P(T|D)$  to  $P(T|\bar{D})$  in the center of Figure 1 gives  $P(D|T)$  on the left of Figure 1.

With Dr. Katz's renovascular hypertension example:  $P(D) = 10\%$ ,  $P(\text{Positive IVP}|D) = 100\%$ , and  $P(\text{Positive IVP}|\bar{D}) = 10\%$ . The ratio of  $P(\text{Positive IVP}|D)$  to  $P(\text{Positive IVP}|\bar{D})$  is 10. A line drawn from  $P(D) = 10\%$  through the ratio of 10 gives  $P(D|\text{Positive IVP}) = 53\%$ .

The above approach can also be used to examine the usefulness of a negative test result. The ratio of  $P(\text{negative test}|D)$  to  $P(\text{negative test}|\bar{D})$  is calculated and marked on the center line of Figure 1. A line drawn from  $P(D)$  through this new ratio gives  $P(D|\text{negative test})$  on the left of Figure 1.

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Letters to the Editor are welcome and will be published, if found suitable, as space permits. Like other material submitted for publication, they must be typewritten double spaced (including references), submitted in duplicate, must not exceed 1½ pages in length and will be subject to editing and possible abridgment.

NOMOGRAM FOR BAYES'S THEOREM

To the Editor: The interest in Dr. Katz's probability graph (N Engl J Med 291:1115, 1974) causes me to offer a solution to the Bayes's rule in the form of a nomogram (Fig. 1).  $P(D)$  is the probability that the patient has the disease before the test.

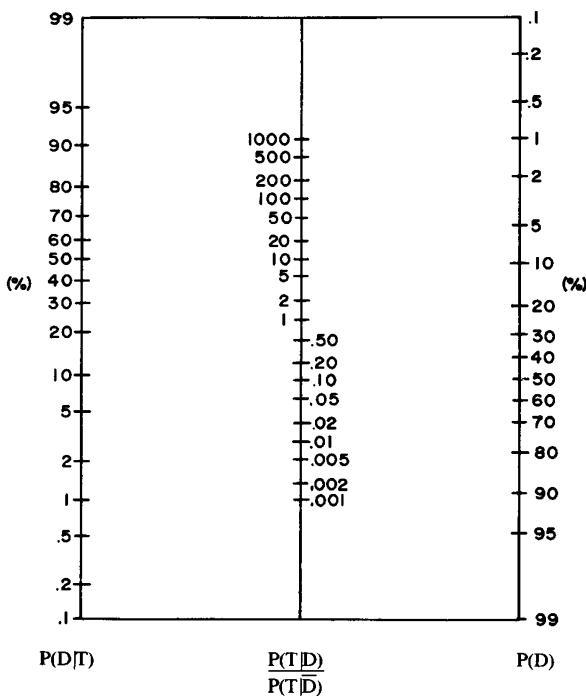


Figure 1. Nomogram for Bayes's Theorem.

BOOK REVIEW

**The Nature and Treatment of Depression.** Edited by Frederic F. Flach, M.D.; and Suzanne C. Draghi, M.D. 422 pp., illustrated. New York: John Wiley and Sons, 1975. \$19.75.

In the past 15 years work on the major depressive (and manic) illnesses has undergone several changes; advances have been made in a number of areas — in their treatment (with several classes of drugs), in attempts to test the biogenic amine hypothesis of their pathogenesis, in studies of their hormonal correlates, in a beginning recognition of their heterogeneity, and in their genetics. Much of this work has resulted in a series of books, of which this is the latest. Like many multi-authored volumes, this one varies in the quality of its contributions. The highlight of this book is a thoughtful and critical review by Baldessarini of the biogenic amine hypothesis, which when it was first proposed broke the stranglehold of purely psychologic and social hypotheses about predisposition to and initiation of the manic and depressive psychoses. It was soon apparent that the design of direct, critical experiments of this hypothesis was in fact very complicated. As Baldessarini points out, there is no direct evidence that profound depressive mood states are the consequence of a relative insufficiency either of catecholamines or of indoleamines in the synaptic cleft and, presumably, therefore, at their post-synaptic receptors. The technics for the assay of these putative transmitters are inadequate, as are the animal models for the study of depressive psychoses in man. If technics for the estimation of levels and turnover rates of the biogenic amines do not suffice, why should the estimation of pituitary polypeptides or adrenocortical hormones, which are regulated ultimately by hypothalamic releasing and inhibiting hormones and factors, be a better test of the biogenic amine hypothesis? Granted that the biogenic amines have been implicated in the regulation of these hypothalamic factors, the fact remains that a number of other hormonal and neural inputs are involved in their regulation. It is therefore confusing to the reader that one contributor asserts that this hypothesis is not adequate or correct, and another author, basing his argument on much remoter, unverified or poorly controlled evidence, summons up arguments to state that it is.

Several such discrepancies can be found in this book — to take other examples, the role of hatred or anger in the pathogenesis of severe depression, or the evidence that environmental factors do or do not play a part in the onset of severe depression. To state that a depressive or manic illness is "endogenous" is to make a mysterious statement concerning some putative brain process that sets it off. On the other hand, there are major problems of method in verifying the social setting in which the illness may begin, not only because manic and depressed people are notori-