BRITISH MEDICAL JOURNAL

SATURDAY 29 JUNE 1985

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Because we receive many more letters than we have room to publish we may shorten those that we do publish to allow readers as wide a selection as possible. In particular, when we receive several letters on the same topic we reserve the right to abridge individual letters. Our usual policy is to reserve our correspondence columns for letters commenting on issues discussed recently (within six weeks) in the BMJ.

Letters critical of a paper may be sent to the authors of the paper so that their reply may appear in the same issue. We may also forward letters that we decide not to publish to the authors of the paper on which they comment.

Letters should not exceed 400 words and should be typed double spaced and signed by all authors, who should include their main degree.

Peer review at work

SIR,—The recent article and correspondence (25 May, p 1555) highlight the problems of specialist referees for general interest journals. Clearly the specialist referee should indicate to the editor which papers draw erroneous conclusions from the data, but often authors give insufficient information or evidence to enable the referees to judge whether the conclusions are valid or not and the referees request modifications or amplifications to the paper.

In the paper by Drs J W Dean and P B S Fowler a difference in response to thyrotrophin releasing hormone was observed in two groups of women. This could have been due to many reasons, of which perhaps four are relevant: (*a*) there was severe coronary artery disease in one group, (*b*) there were more smokers in one group than in the other, (*c*) some other, unmeasured, factor could account for the difference—for example, alcohol consumption, or (*d*) the method of computing the statistics was incorrect.

Surely it is the task of Drs Dean and Fowler to convince the reader that explanation *a* is more likely than *b* or *c* and that the method of computing the statistics is valid? The evidence concerning cigarette smoking and alcohol consumption is clearly of relevance to thyroid stimulating hormone and was mentioned by the referees, so why was it not included in the final version of the paper?

In addition, why did they not justify the use of a response of more than 15 mU/l as an exaggerated response for TSH in the paper? In the analysis of the exaggerated response to divide a continuous variable into "good" and "bad" categories is artificial. A better method of comparing the groups might have been to compare the changes between TSH at baseline and that at 20 minutes using a two sample *t* test.

Concerning the correspondence on the statistical points in the table of their paper the authors quote significance levels for the comparison of two continuous variables. They clearly could not use the unmodified χ^2 test referred to in the methods. Why do they not say which test they did use for they could have used the *t* test, or one of a number of non-parametric tests? Each would have given different p values.

It is always dangerous to quote statistical "authorities"—for example, Upton 1982 referred to in the letter from Dr Fowler on 10 December 1984. Indeed a more recent paper has criticised Upton and advocates the use of Yates's correction in the situation discussed here.[1]

It is not true for Dr Fowler to say that greater subject numbers will not change the significance of the result. Presumably what he means is that with increased numbers the difference in groups is still likely to be significant at the 5% level. The null hypothesis in this case is that the TSH response is the same in the two groups of women. The statistical significance is the probability of rejecting this null hypothesis when it is in fact true. If the null hypothesis were in fact true, which with the type 1 error probability of 0·0056 quoted in the correspondence is unlikely, then increased numbers will reduce the probability of making this error. However, if the null hypothesis is in fact false, for a given significance level increased numbers will reduce the probability of a type 2 error.

The referees' suggested modifications are unlikely to have changed the conclusion—that is, that women with coronary artery disease have an increased response to thyrotrophin releasing hormone—but they would have rendered it more plausible. One of the jobs of a referee is to suggest amendments to a paper to make it acceptable for publication. Few people are in a position to repeat this study, and rather than saying "Here is my hypothesis, now someone confirm it" Drs Dean and Fowler could have improved their paper by making their arguments more convincing. If the results are as important as the authors claim, then surely they could take more note of the referees' comments and not be in such a rush to get into print?

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1 Yates F. Tests of significance for 2 × 2 tables. *Journal of the Royal Statistical Society A* 1984;**147**:426-63.

SIR,—Thank you for allowing us to see the peer review system in action (25 May, p 1555). The paper by Drs J W Dean and P B Fowler does seem to make some unjustified assumptions and dismisses the most important cause of the difference between the two groups.

The two groups compared by this study differ greatly in two respects. Firstly, an abnormal response to thyrotrophin releasing hormone may or may not be a risk factor but there is little evidence from elsewhere to support this view. The other difference between these groups is the difference in cigarette smoking. Surely this is more than enough to account for the different incidence of coronary disease in the two groups. Perhaps in those patients who smoke and develop coronary disease smoking also causes abnormalities of thyroid function. I cannot see how this study can support the hypothesis that minimally impaired thyroid function is an independent risk factor for the development of coronary artery disease.

The study that needs to be done is to compare the response to thyrotrophin releasing hormone in patients with normal coronary arteries (smokers