French complaint that Anglo-Americans seem to be ignorant of French research. He appeared not to see that research reported in the way he recommends is condemned to oblivion irrespective of the language in which it is presented. He concluded by appealing to French authors to defend their language by rejecting Anglo-American methods (*les normes*) of writing.

It is true that scientific medical writing happens to have originated in America and Britain—every advance has to begin somewhere—but has not the time come to “deanglo-saxonise” it, and make it known and available to other language groups? So I should like to suggest that teaching seminars on medical writing, similar to those held by the *BMJ* in London last November, in Edinburgh in January, and in Dublin in April should now be made available in the international field. This teaching is an important advance—perhaps the first step was the publication of Sir Clifford Allbutt’s *Notes on the Composition of Scientific Papers*³—towards a general realisation that preparing medical research reports is a technique that has to be learnt.

Since the recent seminars were intended mainly for English-speaking doctors, they were oriented naturally towards composing papers in this language. In international courses the theory and principles of medical writing (which, of course, must be applied in compiling papers in any language) would be taught to all the participants, problems specific to different languages being dealt with in appropriate language groups. What better means of disseminating good scientific medical writing than a multilingual course, widely publicised in the medical journals of the EEC countries, and organised at BMA House, the seat of the EEC Medical Secretariat?

A universal technique? Yes. A universal language? No.

That the standard of research reporting in some countries is low is admitted in the countries themselves. Dtrie has

written of the contempt in which French surgeons hold French medical journals.⁴ But is publication in English a logical or a practicable remedy? If researchers have never learned (for the simple reason that as yet no one has provided them with facilities for learning) how to prepare reports in their own language, why should they (or, more realistically, the tiny minority who can write in English at all) prepare them any better in a foreign one?

The adoption of the principles and practices of scientific medical writing by research workers all over the world is a logical corollary to the adoption of an international system of units. Reports prepared according to a uniform international pattern and in a simple basic vocabulary will (a) be easier for researchers with a limited knowledge of a given language than the literary productions of writers with a taste for linguistic subtleties,* and (b) lend themselves to straight translation, even by non-medical translators (generally the only kind available), without the radical editing and rewriting needed at present.

In short, that all research should be reported in the same way is feasible and desirable; that it all be reported in the same language is not feasible, and therefore the question of its desirability is academic.

*To forestall protests from the advocates of a “personal style” I should make clear that in this letter I am talking about research reports. Other kinds of medical writing—editorials, review articles, didactic material—afford scope for the stylists.

References

¹ Veylon, R, *Nouvelle Presse Mdicale*, 1975, 4, 435.
² Vargues, R, *Nouvelle Presse Mdicale*, 1975, 4, 1131.
³ Allbutt, T C, *Notes on the Composition of Scientific Papers*, London, Macmillan, 1923.
Dtrie, P, *Nouvelle Presse Mdicale*, 1975, 4, 675.

Statistics at Square One

X—Paired alternatives

T D V SWINSCOW

British Medical Journal, 1976, 2, 225-226

Sometimes it is possible to record the results of treatment or some sort of test or investigation as one of two alternatives. For instance, two treatments or tests might be carried out on pairs obtained by matching individuals chosen by random sampling. Or the pairs might consist of successive treatments of the same individual (see Part XIII for comparison of pairs by the *t* test). The results might then be recorded as “responded or did not

respond,” “improved or did not improve,” “positive or negative,” and so on.

This type of study yields results that can be set out as follows:

Member of pair receiving treatment A	Member of pair receiving treatment B
Responded	Responded (1)
Responded	Did not respond (2)
Did not respond	Responded (3)
Did not respond	Did not respond (4)

The significance of the results can then be simply tested by McNemar’s test in the following way.

Ignore rows (1) and (4), and examine rows (2) and (3). Let the larger number of pairs in either of rows (2) or (3) be called *n*₁

and the smaller number of pairs in either of those two rows be n_2 . We may then, as in Part IX, calculate $\frac{n_1 - n_2}{\sqrt{n_1 + n_2}}$. The result is normally distributed, and its probability can be read from table 7.1.

However, in practice, the fairly small numbers that form the subject of this type of investigation make a correction advisable. We therefore diminish the difference between n_1 and n_2 by 1, using the following formula: $\frac{(n_1 - n_2) - 1}{\sqrt{n_1 + n_2}}$.

Again, the result is normally distributed, and its probability can be read from table 7.1.

For example, Dr Katherine Grey is a registrar in the gastroenterological unit of a large hospital in an industrial city. She sees a considerable number of patients with severe recurrent aphthous ulcer of the mouth. Claims have been made that a recently introduced preparation stops the pain of these ulcers and promotes quicker healing than existing preparations.

Over a period of six months she therefore selected every patient with this disorder and paired them off so far as possible by reference to age, sex, and frequency of ulceration. Finally she had 108 patients in 54 pairs. To one member of each pair she gave treatment A, which she and her colleagues in the unit had hitherto regarded as the best; to the other member she gave a new treatment, B. Both forms of treatment are local applications, and they cannot be made to look alike. Consequently to avoid bias in the assessment of the results a colleague recorded the results of treatment without knowing which patient in each

pair had which treatment. The results are shown in table 10.1.

TABLE 10.1—Results of treating aphthous ulcer in 54 pairs of patients

Member of pair receiving treatment A	Member of pair receiving treatment B	Pairs of patients
Responded	Responded	16
Responded	Did not respond	23
Did not respond	Responded	10
Did not respond	Did not respond	5
Total		54

Here $n_1 = 23$, $n_2 = 10$. Entering these values in the formula given above we have $\frac{(23 - 10) - 1}{\sqrt{23 + 10}} = \frac{12}{\sqrt{33}} = 2.089$.

The 5% level of probability is at 1.96 standard deviations (table 7.1). Here 2.089 exceeds that. Therefore we may conclude that treatment A, the traditional treatment, gave significantly better results than treatment B, the new one.

Exercise 10. A dermatologist tested a new topical application for the treatment of psoriasis on 47 patients. He applied it to the lesions on one part of the patient's body and what he considered to be the best traditional remedy to the lesions on another but comparable part of the body. In three patients both areas of psoriasis responded; in 28 patients the disease responded to the traditional remedy but hardly or not at all to the new one; in 13 it responded to the new one but hardly or not at all to the traditional remedy; and in four cases neither remedy caused an appreciable response. Did either remedy cause a significantly better response than the other? *Answer:* Yes, the traditional remedy; $0.05 > P > 0.01$.

What links are there between 5-HT, depression, and pain. 5-HT is well known as a chemical cause of local pain and I have noticed that in depressed subjects the pain threshold is much reduced. Is this why depressives complain so bitterly about their pain?

In animals decreasing brain 5-HT leads to increased pain sensitivity.¹ But relatively little research has been done. Clinically, there have been a few reports of increased pain sensitivity in depressed patients, though obviously it is difficult to know whether this is due to some primary neurobiological abnormality or to secondary "psychological" factors. This has been discussed by Walters² and Bradley.³ Bradley found that in the depressives he studied who had localised pain without organic cause he could identify two groups. One group had chronic pain followed by depression, the depression alone responding to treatment but with increased pain tolerance. The other group had both pain and depression appearing together and both were relieved by treating the depression. To invoke a link between the possible decrease in brain 5-HT concentrations in depression and an increase in pain sensitivity would clearly therefore be quite unwarranted at present.

¹ Tenen, S S, *Psychopharmacologia*, 1967, **10**, 204.

² Walters, A, *Brain*, 1961, **84**, 1.

³ Bradley, J J, *British Journal of Psychiatry*, 1963, **109**, 741.

How would you treat (a) falciparum malaria, (b) benign tertian malaria (Plasmodium vivax), or (c) chloroquine-resistant malaria in a patient who is also suffering from G6PD deficiency? What drug prophylaxis for malaria would you advise for such a patient?

In patients with G6PD deficiency most antimalarial drugs can be administered in the usual dosage except for primaquine, which should not be used in chemoprophylaxis because of the risk of haemolysis. In a chloroquine-sensitive area, I would advise chloroquine weekly (300 mg base in adults) and in a chloroquine-resistant area pyrimethamine 75 mg with sulfadoxine 1500 mg every four weeks. The standard treatment for vivax malaria is a course of chloroquine (1.5 g base in adults), followed by a 14-day course of primaquine

(15 mg base daily in adults) to prevent a relapse. In patients with G6PD deficiency the primaquine should be given under supervision at full or half dosage and stopped if haemolysis or haemoglobinuria occurs; alternatively the primaquine could be omitted and used only in a relapse. Chloroquine-sensitive falciparum malaria can be treated with a 1.5 g course of chloroquine. In seriously ill patients the initial 200 mg doses of chloroquine should be given by slow intravenous infusion or quinine used instead. Chloroquine-resistant falciparum malaria can be treated with a short course (at least four doses) of quinine followed by a single dose of sulfadoxine-pyrimethamine.¹ In patients whose clinical condition is causing concern the initial doses of quinine should be administered as a slow intravenous infusion.

¹ Hall, A P, et al, *British Medical Journal*, 1975, **2**, 15.

What risk is there in working in an atmosphere containing calcium silicate dust?

It is not clear whether the question refers to the inhalation of calcium silicate as a pure chemical or to the inhalation of a more complex material which might loosely be referred to as "calcium silicate." There is little information on the toxicity of the chemical compound calcium silicate. Many dusts of calcium are considered soluble in lung tissue and cannot be held responsible for a pneumoconiosis.¹ Cement is a compound containing calcium, silicon, and oxygen and is differently described as a mixture of calcium oxide and silica (generally in small amounts) or as a mixture of calcium silicates and aluminates with added gypsum.² Cement dust does not cause pneumoconiosis unless more than 2% "free silica" is present as a contaminant. Pneumoconiosis among cement workers is remarkably rare, although a hazard may exist for workers at the early stages of the cement-making process. It should be remembered, however, that cement dust is alkaline and may be irritant. Also, it may contain chromium and sometimes other metals which could produce a contact dermatitis.³

¹ Hamilton, A, and Hardy, H, *Industrial Toxicology*, 3rd edn, p 446. Massachusetts, Publishing Sciences Group Inc, 1974.

² Parkes, W R, *Occupational Lung Disorders*, p 158. London, Butterworths, 1974.

³ Fisher, A A, *Contact Dermatitis*, 2nd edn, p 104. Philadelphia, Lea and Febiger, 1973.