whether other branches of complementary medicine should be authorised.

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1 Goldbeck-Wood S, Dorozynski A, Lie LG, Zinn C, Josefson D, Ingram M. Complementary medicine is booming worldwide. *BMJ* 1996;313:131-3. (20 July.)

Single dose of oral dexamethasone for outpatient croup

Failure to follow up all patients is a concern

EDITOR,—G C Geelhoed and colleagues conclude from their study that oral dexamethasone at a dose of 0.15 mg/kg is effective in reducing return to medical care in children with mild croup.[1] The sample size seems adequate, though on the borderline for detecting a 90% reduction, but the fact that four children were not followed up is of concern. If the two children lost to follow up in the treatment group had in fact sought some medical care (or even died) this would have meant that the difference in outcome between the two groups was not significant by Fisher's exact test. We wonder why the calculation of sample size assumed such a large reduction as 90%. Was this a retrospective calculation?

The final key message states that all children presenting with croup should be considered for steroids. Before the authors' findings can be generalised we need to know what proportion of children who presented with mild croup were excluded from the trial and what the rate of parental refusal was.

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1 Geelhoed GC, Turner J, Macdonald WBG. Efficacy of a small single dose of oral dexamethasone for outpatient croup: a double blind placebo controlled clinical trial. *BMJ* 1996;313:140-2. (20 July.)

Authors should have stated how drug was given

EDITOR,—The results of G C Geelhoed and colleagues' trial of a single dose of oral dexamethasone for outpatient croup would be easier to apply in practice if we knew how the authors gave the dexamethasone.[1] Did they use the 500 μg tablets or a solution, and did they round the dose to the nearest 250 or 500 μg? Was the dose given in the emergency department immediately on diagnosis, or did a parent give it later after getting it dispensed?

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1 Geelhoed GC, Turner J, Macdonald WBG. Efficacy of a small single dose of oral dexamethasone for outpatient croup: a double blind placebo controlled clinical trial. *BMJ* 1996;313:140-2. (20 July.)

Authors' reply

EDITOR,—Rosie Helowicz and Jill Walton express concern about the children who were lost to follow up in our study. Our follow up rate of 96% is extremely good when compared with follow up rates in other clinical studies of outpatient treatment. Although we could not follow up four children directly, we were able to ascertain that none of them presented to any hospital in the Perth metropolitan area in the 10 days after their original episode of croup.

Helowicz and Walton also point out that if the two children lost to follow up in the treatment group had relapsed then the results would no longer be significant ($P = 0.08$ by our calculations). On the basis of the outcome in the 96 other children in the study, however, it seems more logical to assume that the two children lost to follow up in the treatment group would not have relapsed, which would give $P = 0.003$. Rather than indulge in more "what if" scenarios, we think it worth noting that we saw 935 children with croup in our emergency department in the first six months of 1996, when it was standard practice to offer these children a dose of oral dexamethasone. Of the 668 children who were sent home, only 19 (2.8%) reattended, which is a much smaller proportion than the 15% we would have expected. In answer to Andrew Herxheimer's questions, children in the study were given a solution of oral dexamethasone BP (manufactured in our hospital pharmacy[1]) in the hospital emergency department. The dose given was exactly 0.15 mg/kg.

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1 Accordino A, Chambers R, Thompson B. A short term stability study of an oral solution of dexamethasone. *Aust J Hosp Pharm* 1994;24:312-6.

QTc dispersion and risk of cardiac death in peripheral vascular disease

Dispersion of 60 $ms^{1/2}$ is insufficient to change clinical management

EDITOR,—Dawood Darbar and colleagues investigated the usefulness of QTc dispersion as a predictor of cardiac death in patients with peripheral vascular disease.[1] They compared QTc dispersion in a routine electrocardiogram in patients with peripheral vascular disease who had probably died of cardiac causes with that in those who survived. The mean QTc dispersion in these patients was 86.3 $ms^{1/2}$ and 56.5 $ms^{1/2}$ respectively. The authors claim that a QTc dispersion of 60 $ms^{1/2}$ is highly predictive of sudden cardiac death. However, QTc is a continuous variable, so the normal distributions of the data for each group would be as shown in figure 1.

Although the figure of 60 $ms^{1/2}$ is significant, in reality only patients with a QTc dispersion of >86 $ms^{1/2}$ may confidently be considered to be at increased risk. Given that the mean QTc dispersion of the survivors was 56.5 $ms^{1/2}$ with a standard deviation of 25.4, it is difficult to see how, in clinical practice, a QTc dispersion of 60 $ms^{1/2}$ would be sufficient to alter the management of the patient.

There are also methodological problems with this qualitative measurement of QT dispersion. For example, estimating the end of the T wave as being at the nadir between a T and U wave[2] may lead to an underestimate. Determining the adjusted QTc dispersion by dividing the QTc dispersion by the square root of the number of leads measured[3] may compound qualitative errors that might be expected to arise from a high interobserver variability in QT measurement.

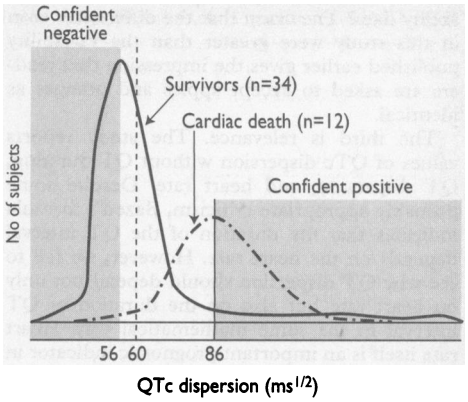

Fig 1—*Diagrammatic representation of data of Darwood and colleagues*[1]

Although promising, the evaluation of dispersion of repolarisation from surface electrocardiography requires validation and would be much more useful clinically if qualitative bias could be removed. It is at present rather optimistic for QT dispersion to be considered the "electrophysiological Holy Grail."[1]

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1 Darbar D, Luck J, Davidson N, Pringle T, Main G, McNeill G, *et al.* Sensitivity and specificity of QTc dispersion for identification of risk of cardiac death in patients with peripheral vascular disease. *BMJ* 1996;312:874-9. [With commentary by R Campbell.] (6 April.)
2 Day CP, McComb JM, Campbell RWF. QT dispersion: an indication of arrhythmia risk in patients with long QT intervals. *Br Heart J* 1990;63:342-4.
3 Day CP, McComb JM, Matthews J, Campbell RWF. Reduction in QT dispersion by sotalol following myocardial infarction. *Eur Heart J* 1991;12:423-7.

Three facets of the study need attention

EDITOR,—Darwood Darbar and colleagues showed the predictive value of QTc dispersion (≥60 ms) in patients with peripheral vascular disease,[1] but we think that three parts of this paper need attention.

The first is the patient population. This small, retrospective study was not controlled for risk factors, which introduces significant selection bias. Patients with peripheral arterial disease have a higher incidence of coronary artery disease and its complications. Ischaemic burden (symptomatic or asymptomatic) and the severity of coronary stenosis influences prognosis.[2] Mean ejection fraction is useful, but in individual patients the above events clearly alter the prognosis, with only a small change in mean ejection fraction. The paper fails to correct for these influences. Several earlier studies have found QTc not to be a predictor of ventricular tachycardia or ventricular fibrillation even in patients after a myocardial infarction.[3] Therefore, we wonder whether this test would still be predictive in a multivariate analysis.

The second is that the measurement of QT and QTc dispersion is unstandardised[4] and shows wide variability even with digitised boards.[4] Darbar and colleagues do not mention the exact method of measurement, but the impression is that it was manual. With three observers measuring QTc, we find it surprising that interobserver or intraobserver variability was not evaluated. Why do the authors cite studies observing large errors in QT dispersion assess-