often lead to erroneous results because the basic assumption on which the method is based are not satisfied by the current situation—in other words "a little learning really is a dangerous thing." I would therefore like to make the following points, though they mainly re-echo statements of Dr Swinscow.

(1) Researchers ought not to make the mistake of assuming that *any* set of observations tends to be normally distributed—that is, constitutes a sample from a normally distributed population. Although many medical and biological parameters appear so to behave, other commonly occurring distributions exist. In particular the binomial, multinomial, and Poisson distributions repay study and, given appropriate tables, demand no greater mathematical fluency than the present articles required. It may be mentioned, in passing, that a "goodness of fit" test, based on the χ^2 distribution, exists for determining whether or not a given set of observed frequencies is in accord with the frequencies expected from a specified theoretical distribution. Some care in use is necessary.

(2) Notwithstanding the preceding paragraph, by virtue of a result known as the "central limit theorem," in *large* samples the comparison of means, the comparison of proportions, and the establishment of confidence intervals may happily be carried out using the test statistic based on the normal distribution, and which Dr Swinscow has dealt with in articles VII, VIII, and IX. If, however, the samples are *small*, then the above types of calculation demand the use of the *t* statistic, covered in articles XI, XII, and XIII. These *t* tests are further subject to the assumptions that the parent population from which a sample is drawn be normally distributed and that when sample means are being compared the variances of the parent populations are equal. The hypothesis that sample variances are not significantly different may be investigated using an F test (and considered, for example, in the book by Professor Armitage[1]). The division between "small" and "large" samples is commonly held to occur in the range 25-30, for the *t* distribution with more than 25 degrees of freedom is virtually indistinguishable from the standardised normal variate. Although, as Dr Swinscow has mentioned, the possibility may exist of transforming a non-normal variate to one nearly normal such technique should be treated with caution.

(3) The advent of electronic pocket calculators, whether programmable or not, has made the execution of the majority of the calculations which the articles cover a seemingly trivial matter. However, experience has shown that numerical mistakes are very easy to make—a finger may rest too lightly (or too heavily) on a key, a decimal point be inserted incorrectly, one too few numbers entered in memory, etc, etc. It is therefore wise to record intermediate values wherever practicable, and the final answer should certainly be checked.

Dr Swinscow has given the mathematical identity $\Sigma(x-\bar{x})^2=\Sigma x^2-(\Sigma x)^2/n$, which is useful for calculating purposes. Other results which may be found useful, especially if no calculator is available, are that if x is transformed linearly by $x'=\alpha x+\beta$, where α represents a change of scale and β a change of origin, then $x'=\alpha\bar{x}+\beta$ and $SD(x')=|\alpha|\,SD(x)$. The modulus sign, $|\ |$, is used to ensure that the standard deviation is always positive. Also for the correlation coefficient $|r(x,y)|=|r(x',y)|$.

(4) Wherever practicable data should be plotted in some *appropriate* form—for example, a histogram, frequency polygon, or scatter diagram. Mere "number crunching" of the data plotted in fig 18.1D (18 September, p 681) will yield a sample correlation coefficient in excess of 0·9, yet the graph strongly suggests a curved line (and hence non-linear association). Again, statistical techniques exist to determine the degree of the curve of best fit.

In conclusion, referring to the final article on "Correlation" (2 October, p 802), Dr Swinscow gives the formula for the regression equation $y=a+bx$ when y regresses on x. This allows an estimate of the "average" anatomical dead space (y) to be obtained given the height of a child (x). On occasion it may be asked what height of child would correspond to an anatomical dead space of so many millilitres. This "converse" situation requires the calculation of the (linear) *regression of x on y*. If the equation is written $y=c+dx$, then

$$d=\frac{\Sigma(y-y)^2}{\Sigma(x-\bar{x})\,(y-\bar{y})},\quad c=\bar{y}-d\bar{x}.$$

For the given example, $d=1{\cdot}443$, $c=-141{\cdot}68$.

The closer the points of a scatter diagram lie to a straight line, the closer are these regression lines which interest in $(\bar{x},\bar{y})$. The line of y regressing on x is the less steep.

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[1] Armitage, P, *Statistical Methods in Medical Research*. Oxford, Blackwell Scientific, 1971.

"Vasovagal" syncope

SIR,—Dr P Taggart and others, in their report (2 October, p 787) on the reflex cardiac reaction to fear and pain in healthy young adults, suggest that the bradycardia observed in some of them is of parasympathetic origin and that vagal overactivity may be the basis of the syndrome known as "vasovagal" syncope. This implies that atropine should prevent the syndrome though they do not state that atropine should be used for this purpose. As an anaesthetist I am quite certain that atropine not only fails to prevent syncope but increases the hazards of the other forms of cardiovascular collapse which may complicate the induction of anaesthesia in frightened patients—the ventricular tachycardia-fibrillation sequence and myocardial infarction. In reaching the conclusion that vagal overactivity may be the basis of the bradycardia observed in their patients Dr Taggart and his colleagues did not consider the role of the reaction of the peripheral blood vessels to adrenergic stress in conscious subjects.

Fear and pain activate the sympathetic nervous system to cause reflex constriction of the alpha-adrenoceptor blood vessels, especially the veins, and reflex dilatation of the beta-adrenoceptor blood vessels of the skeletal muscles. Emotional overactivity of sudden onset causes a temporary sequestration of relatively large amounts of blood in the dilated blood vessels of the muscles when the venous return pathways are blocked by the venoconstriction of fear. A failure of the venous return to the heart is precipitated and is increased by gravity when the subjects are standing or sitting in the upright position. As explained by Starling many years ago, the sudden loss of venous return lowers the heart rate and output, with a consequent lack of blood to the brain and loss of consciousness. Other examples of ischaemic faint may be seen in the supine hypotensive syndrome of late pregnancy and in the Valsalva manoeuvre. The most convincing evidence against vagal involvement in this form of syncope is the fact that atropine does not prevent it.[1]

The cardiovascular reaction to fear is prevented by the longer-acting benzodiazepines such as lorazepam which block the ability to be afraid.[2] Sedative drugs have no effect on the cardiovascular reaction to pain. The cardiovascular reaction to pain or injury is independent of the conscious perception of pain because the reflex arc is complete in the brain-stem below the level of the sensory thalamus.[3] Psychotropic drugs such as the natural and the synthetic opiates, which abolish the conscious perception of pain, have no effect on the reflex reaction of the sympathetically innervated blood vessels to traumatic stimuli. The peripheral vascular reaction to pain is prevented by blockade of the sensory afferent nerves from the injured area or by blockade of the sympathetic efferent nerves to the blood vessels at adrenoceptor, ganglionic, or preganglionic level. A more complete account of the work concerning the recognition and the control of the overall cardiovascular reaction to the adrenergic effect of fear and pain, with relevant plethysmographic and electrocardiographic illustration, has been published.[4]

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[1] Weissler, A M, *et al*, *Circulation*, 1957, **15**, 875.
[2] Johnstone, M, *Anaesthesia*, 1976, **31**, 868.
[3] Ackner, B, *Journal of Psychosomatic Research*, 1956, **1**, 3.
[4] Johnstone, M, *Anaesthesia Rounds 10: Adult Preoperative Medication*. Macclesfield, Imperial Chemical Industries Ltd (Pharmaceuticals Division), 1976.

Double negatives and euphemisms

SIR,—For some time I have struggled with the wording on the revised Temporary Resident form (EC 19 Rev 1972), which states: "Not more than 15 days" or "More than 15 days," which surely should be "Less than" or "More than." We are now faced with a similar nonsense on the new sick notes (Form Med 3), which states (*a*) You need not refrain from work and (*b*) You should refrain from work, which surely should read (*a*) Fit for work and (*b*) Unfit for work.

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Dangers of tinted glasses for driving

SIR,—In reply to Dr C E Connolly (21 August, p 478) I have the following comments to make regarding the wearing of photochromic lenses while driving.

In fact, the various horrifying tales of what will happen on driving out of bright sunlight into a dark tunnel are no more true of wearers of these lenses than of any other driver. There are two main reasons for this. The most important is that it is the ultraviolet light in ordinary sunlight which makes the lenses go darker. When the wearer is sitting in a car all or about 98% of the ultraviolet light will have been filtered out of the sunlight by the windscreen before it reaches the lenses. In