the other hand may consider that because of inadequate nursing staff in the hospital he should not be admitted, as this would mean inadequate nursing not only for this patient but for many others. The latter judgement means that I am performing a *nursing* management function rather than a medical one.

It would do a great deal for the morale of nurses—and thus for their efficiency—if consultants (sic) insisted on acting as *advisers* to nursing staff rather than as *directors* of them. Many nurses would have initial difficulty in accepting this responsibility, of course, but they can only learn if given the opportunity.

Until our profession is prepared to relinquish its defensive fantasies of omnipotence in hospitals, the difficulties which lead to the present discussion of a code of nursing practice are likely to continue.

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DEAR SIR,

I am writing in response to the invitation of the President for views about recent suggestions that there should be either 'Guidelines' or a 'Code of Conduct' for psychiatric nurses, with special reference to the problem of handling violent patients.

Something closely resembling such a code of conduct is to be found in Chapter 5 of the First Edition of the R.M.P.A.'s 'Red Handbook', which was published in 1885 with the title, 'Handbook for the Instruction of Attendants on the Insane.' As successive editions of the 'Handbook' appeared, the method of presentation changed to that of a modern textbook of psychiatry for nurses, and the problem of handling violence was dealt with by showing that good methods arose logically and naturally from a proper knowledge and understanding of mental illness. It seems then that any attempt today to produce either 'Guidelines' or a 'Code of Conduct' for nurses must ignore the direction in which progress has been made, and indeed is simply to put the clock back for nearly a century.

Such action would be doubly unfortunate, as the point requiring most urgent attention has been stressed again and again in the 'Red Handbook'. This is that if a nurse anticipates violence on the part of a patient, he or she should ensure that adequate help is summoned. Overwhelming superiority in numbers usually results in avoiding violence; even if it still occurs, it can in these conditions be overcome with the least possible risk of injury to the patient. If, however, there is a shortage of staff, the nurses are deprived of the most important help they require in handling potentially violent patients, and the real responsibility for this rests with Management.

Indeed, as the Farleigh Report suggests that the tragic events there were the end product of years of mismanagement, perhaps 'Guidelines' or a 'Code of Conduct' should be prepared for the benefit of those responsible for management in the Health Service.

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ANOREXIA NERVOSA

DEAR SIR,

According to Wright *et al.* (1969) one of the most consistent symptoms of anorexia nervosa is the presence of lanugo. Lanugo is characteristic of intra-uterine life from the fourth month until term, reaching a peak in the seventh and eighth months and falling off thereafter. Most hair growth is controlled by steroids; the steroids present in abnormally high concentration in the foetal placental circulation are dehydroepiandrosterone (DHA) and its sulphate (DHAS), also 16oH-DHAS, oestriol and progesterone. The last two of these continue to rise until parturition, but the others probably decline towards the end of pregnancy because the foetal zone, which produces DHA, decreases relatively to the rest of the adrenal as pregnancy advances. Also DHA is found in the urine of premature babies but not in that of full-term ones (Birchall *et al.* 1961). Since lanugo declines in the same way towards the end of pregancy it seems possible that it is caused by DHA. (DHAS and 16oH-DHAS are less likely to be responsible because conjugated steroids have relatively little biological activity.) Since DHA is thought to be produced only by the adrenals this would seem to implicate the adrenals in the causation of anorexia nervosa.

Against this are the facts that it is practically confined to teenage girls, and is associated with amenorrhoea and also with low oestogen levels, all of which suggest an ovarian disorder. Typically it starts a year or two after menarche (Crisp, 1965). For the first few years after menarche the menstrual cycles are anovular, i.e. there is no luteal phase; so it looks as if anorexia nervosa is associated with the onset of ovular cycles, which would incriminate the corpora lutea. This supposition is strengthened by the fact that the low oestrogen levels (Russell, 1965) are almost wholly due to low levels of oestriol,

the oestrogen typical of the luteal phase of the cycle when it forms 50 per cent or more of the total oestrogen.

It has recently been shown (Rennels and Singer, 1970) that if immature female rats are treated with PMS/HCG (equivalent to FSH plus LH) ovulation occurs and corpora lutea develop. If a larger dose is given the corpora lutea become heavily luteinized, and at the same time the adrenals decrease in size. The authors demonstrate fairly convincingly that the adrenal suppression is due to output of DHA by the over-stimulated immature ovaries, the DHA acting as a negative feedback on the output of ACTH by the pituitary.

Girls who later develop anorexia nervosa reach menarche earlier than normal girls (Wright *et al.*, 1969), so perhaps the ovaries are not fully mature. If the steroid metabolism of the ovaries were analogous to that of the adrenals (which it is likely to be because embryologically they are derived from the same coelomic epithelium), one might expect that the route of synthesis of oestriol by these immature ovaries would be that of the foetal adrenals, namely: pregnenolone → 17OH → pregnenolone → DHA → DHAS → oestriol. In the case of the foetus the final stage is performed by the placenta. Presumably in the case of the ovaries this would be the role of the corpora lutea, which, from a chemical angle, have a function very similar to that of the placenta. If the corpora lutea were unequal to the task the result would be a condition analogous to that of the foetal adrenals at mid-term i.e. high DHA and low oestriol levels. The high levels of DHA might cause lanugo and also depress the adrenals. The latter might well cause anorexia, since anorexia is a symptom of Addison's disease.

Although anorexic girls have a number of symptoms found after adrenalectomy e.g. low blood pressure, bradycardia and inability to excrete a water load efficiently (Russell, 1965) they do not have all the symptoms of adrenal insufficiency. The simplest explanation would seem to be that they have a deficiency of *one* of the enzymes of the adrenal cortex, and that the adrenal depression (about 25 per cent in rats) is sufficient to put this enzyme completely out of action while leaving the others at the low end of the normal range.

It is well known that in man there is an interplay between the adrenals and the gonads. Thus, gonadectomy in both sexes leads to a rise in blood oestriol levels (Lemon, 1970) due to the adrenals. So the adrenals normally compensate for gonadal inadequacy in this way. On the other hand, adrenalectomy not infrequently leads to premature menopause in women as does also Addison's disease, so there are women who depend upon their adrenals for normal ovarian functioning. Presumably, in their case, the ovaries lack some substance (oestriol?) which the adrenals can provide.

If anorexic girls were in this position and had also an adrenal disorder leading to a deficiency of the substance required by the ovary, a vicious circle would develop. The inadequacy of the immature ovaries would lead to flogging by gonadotrophins from the pituitary; this would raise DHA levels which in turn would further depress the already inadequate adrenal enzyme. The most likely candidate for the adrenal enzyme would be one which takes part in the synthesis of oestriol.

This explanation of the illness has the advantage of being in accordance with the known genetic character of anorexia nervosa, which is rare and likely to be due to the inheritance of two mutant recessive genes. Generally, of course, such genes are at the same locus, which here they obviously could not be since one disorder is in the ovary and the other in the adrenal. However, if the two genes had the same function e.g. if both were involved in the synthesis of oestriol, the result might not be so very different.

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MIGRAINE, ANOREXIA NERVOSA AND SCHIZOPHRENIA

DEAR SIR,

Dr. Avery, in his letter in the February 1971 issue of the *Journal* (Vol. 118 p. 255), has (as usual) missed the point I was trying to make in my own letter, which was that it seems unnecessary and unfair to deny the use of oral contraceptives, with their many obvious advantages, to all women who have a history of migraine. Quite often the migraine history will be associated with disturbances of sex-hormone balance, and it would seem reasonable to try to stabilize the levels by careful choice of an oral contraceptive. Obviously, most of us try to