venous thrombosis contain a number of very different progestogens, while only two very similar oestrogens are used in drugs marketed in Britain. There is other evidence that oestrogens may have an effect on blood clotting.[2 3] These findings suggest that it is the oestrogen rather than the progestogen which is responsible for the thrombosis, and if this is true there are three practical conclusions. Firstly, the sequential types of oral contraceptive may be more dangerous, since they contain more ostrogen than the combined pills. Secondly, the continuous-low-dose-progestogen technique (not considered in the trials reported), using such compounds as chlormadinone acetate,[4] requires no use of oestrogen and may afford a means of avoiding thromboembolic effects. This technique is not free of disadvantages but it seems to be well worth careful evaluation with respect to the danger of thrombosis. Thirdly, the administration of oestrogen for any purpose—not only contraception—should be regarded as carrying a definite risk if the oestrogen is given for a long time or in high dosage. The use of oestrogens to reduce blood cholesterol in patients with coronary insufficiency or to suppress lactation[2] are particular cases in point.

The occurrence of thromboembolic disease in one out of every 2,000 women on the pill each year is disquieting. Should a doctor give a healthy young woman a prescription for an oral contraceptive if it may lead to her death? The picture must be seen in the perspective of the effect on the whole population. Other forms of contraception—apart from male and female sterilization—have high failure rates. The risk of death in pregnancy, even from thrombosis alone, is greater than that of taking oral contraceptives for the same length of time, though how many pregnancies would result from changing to other forms of contraception than the pill is speculative. So, while there is no cause for panic about the possible consequences of widespread use of the present types of oral contraceptives, neither is there room for complacency. No chair in clinical reproductive physiology exists in Britain, but co-ordinated interdisciplinary research in this field is urgently needed. The goal must be effective contraception, free of all risk, and psychologically fully acceptable. Women would give high priority to such research.

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Assessing "Clinical Competence"

Tests of clinical competence make up an important part of the final examination in medicine. Traditionally in Britain the clinical examination has comprised one principal case and one or more short cases. But how certain are we that these tests are valid and satisfactory? Analyses of clinical tests have shown many disadvantages. The correlation between the opinions of the two pairs of examiners concerned is often very poor, and patients used in the examination vary tremendously.[1] So in a situation where variation among candidates is the single important factor to be measured the additional variables of an examiner and a patient are introduced. Moreover, the all-embracing term "clinical competence" is made up of many attributes—the ability to elicit a good history from a patient and to carry out a competent examination, to recognize the various physical signs, and to use the clinical features to arrive at a diagnosis, and hence a rational plan of management. In other words, much more is being tested than mere recall of isolated information, which is the main aim of multiple-choice examinations. We are testing a higher level of intellectual process—the ability to synthesize the variety of elements in a clinical situation into an original and meaningful whole.[2]

What improvements must be made in our tests of clinical competence? Firstly, and most importantly, the different skills and attitudes which go to make up clinical competence must be defined. Only then can we set about the task of devising tests to examine these objectively. The ability to take a case history and perform a physical examination can be analysed by the use of check lists of the various important features. In Newcastle upon Tyne the ability to recognize clinical signs has been used as a separate test which is easily and objectively marked. By the use of a wide range of assessments of clinical competence more reliable and reproducible results can be obtained. Nevertheless, the major problem of variation between patients is difficult to overcome with our present methods.

In the United States the National Board of Medical Examiners, of Philadelphia, and G. E. Miller and his colleagues at the University of Illinois, in Chicago, have been attempting to rationalize and standardize practical examinations.[1] By the use of cine films, *x*-radiographs, pictures, and tape recordings they can do away with patient variation; and by making the marking wholly objective they can eliminate examiner variation. Trained examiners or professional actors have been used to play the part of patients to test history-taking ability, but many examiners feel that such devices are far removed from the real clinical situation. These groups have also pioneered the use of simulated patient-management problems, both as an educational device and as a method of examination.[3] In this technique a description of an actual clinical situation is presented to the student, who is asked to select from a list the responses he feels to be most appropriate. He does this by erasing an opaque overlay alongside his choice. Under the overlay he finds relevant information, which may be purely descriptive or which may indicate how the position has been altered by his management, then referring him elsewhere in the programme. By analysing the pattern of erasures the examiner can assess the student's response. A further refinement of this technique for both teaching and testing is the use of the computer in this type of situation.[4] The student types in his request for data and the computer provides the appropriate response. Such dialogues between students and computer may well be found to be increasingly valuable in the future.

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Few would disagree that tests of clinical competence are important and that they could be made much more objective. They should be supplemented by multiple-choice questions and careful assesment of clinical ability during the student's career. Even such apparent imponderables as medical students' attitudes can, it seems, be analysed and tested.[5]

Measles and Panencephalitis

Virologists have been increasingly turning their attention to subacute and chronic diseases of the central nervous system, many of which have hitherto had no satisfactory aetiological explanation. The impetus was increased by the findings that the chronic diseases visna, scrapie, and kuru appear to be caused by virus-like agents and by the concept of slow viruses and slow infections.[1]

Accumulating evidence that subacute sclerosing panencephalitis (S.S.P.) may be due to measles virus was recently reviewed and discussed at a conference at Bethesda in the U.S.A.[2] S.S.P. usually affects children of school age some years after they have had measles. There may be an insidious onset of intellectual deterioration or psychological disturbances, so that after a time the child becomes ineducable, or there may be a sudden onset with convulsions and a variety of neurological manifestations. Within weeks or months in either case the patient becomes bed-ridden, blind, and disorientated, the arms and legs are flexed and have plastic rigidity, myoclonic spasms occur 4 to 12 times a minute, the eyes wander purposelessly, and only painful stimuli elicit a feeble cry. Eventually coma leads to death unless there has been a previous fatal intercurrent infection. Remissions are common, however, and may last for weeks or years.

J. R. Dawson[3 4] first noted the presence of intranuclear inclusions in S.S.P. in 1934 ; later H. Pette and G. Döring[5] produced an encephalitis in rabbits, mice, and monkeys by inoculation of nervous tissue from patients, but no infective agent was identified. Numerous attempts to repeat these experiments have failed. F. E. Sherman and his colleagues[6] first demonstrated fluorescent staining of the cytoplasmic inclusions by labelled herpes simplex antibody. Other virus aetiologies have been suggested: infectious hepatitis,[7] vaccinia,[8 9] Coxsackie,[10] and the poliomyelitis vaccine virus.[11 12] Virus particles (60–80 mμ diameter)[13] have been described in astrocytes from the brains of patients with S.S.P. by several authors[14–16] but also in other diseases.[17]

At the conference R. M. Herndon and L. J. Rubinstein[13] reported that they had found particles indistinguishable from large myxoviruses budding from the cytoplasmic inclusions. E. H. Lennett and his colleagues[18] reported six cases of S.S.P. in which measles antigen could be demonstrated by fluorescent antibody in the nervous tissue in positions similar to the inclusion bodies ; all the cases had high levels of measles antibody—three of them showed increases over several months during the illness. Five of the patients had unusual amounts of measles antibody in the cerebrospinal fluid. B. R. Adels and his co-workers[19] reported on 55 cases: the levels of measles antibody in the S.S.P. were higher than those found in natural measles even if complicated by encephalitis. Repeated attempts to isolate virus from the nervous system in S.S.P. have so far failed.[18–20] S. A. Schneck[21] reported one case of S.S.P. with an onset three weeks after live measles vaccination and reviewed other central nervous system complications of live measles vaccination. In contrast J. Adams[22] described the pathological findings in the various forms of measles encephalitis which have the common features of cytoplasmic and intranuclear inclusions and small multinucleated giant cells.

There are four pieces of evidence that S.S.P. may be caused by measles virus: the presence of particles morphologically resembling it in the central nervous system ; the presence of measles antigen, demonstrated by fluorescent antibody ; exceptionally high levels of measles antibody, especially in the cerebrospinal fluid ; and suggestive evidence that measles virus invades the central nervous system in a proportion of cases even without clinical encephalitis.[23] Each of these pieces of evidence can be challenged individually,[2] but together they make a good case worthy of more intensive study. Isolation of the virus from the nervous tissue of patients with S.S.P. would be helpful and is probably the only way to produce incontrovertible evidence that an infection with measles exists ; the major difficulty may be the presence of a high antibody concentration, but this can be overcome if the infected cells can be cultured. The virus may, however, be present in a defective form with an altered mechanism of multiplication.[24] But the presence of virus does not in itself prove an aetiological relationship—it might be that the altered condition of the central nervous system in S.S.P. enables measles virus to persist. In the same sense it might be argued that the high level of antibody in these patients indicates merely exceptional immunological responsiveness, and that this might be related to the pathogenesis of the disease. However, there seems to be a reasonable probability that S.S.P. is caused by measles virus. Further support is given by the finding that measles encephalitis can assume a chronic form in which there is severe brain damage, including marked gliosis and diffuse demyelination.[22]

This type of consequence of persistent latent infections in

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