α-Thalassaemia in Britain

SIR,-The results of surveys for abnormal haemoglobins such as the one reported by Dr. J. Stuart and his colleagues from Birmingham (3 November, p. 284) illustrate the point that if one does not include in any series of screening tests a test for HB H bodies, the diagnosis of α-thalassaemia trait will be missed.1 The Birmingham survey included a haemoglobin electrophoresis and HB A2 and HB F estimations. Of the 6,835 students studied, 26 were found to have the thalassaemia trait and 15 of α-thalassaemia trait. This contrasts with the experience in this hospital, which serves an area containing many Negro and Asian immigrants and where in the past three months there have been 26 new cases of β-thalassaemia trait and 15 of α-thalassaemia trait. The α-thalassaemia patients included 1 Turk, 1 Chinese, 1 Greek, 1 Lebanese, 1 Pakistani, 1 African, 2 Indians, 5 West Indians, and 2 Europeans (1 Pole and 1 Briton with German ancestry). Two of the West Indians were from the same family. The patients with α-thalassaemia trait had similar blood films to those with β-thalassaemia trait and the same abnormal red cell indices (low mean corpuscular haemoglobin and mean cell volume). Their haemoglobin electrophoretic patterns were normal and their HB F levels were in the normal, usually the low normal, range. In each patient HB H bodies were present in a small proportion of red cells and if the patients had not been tested for HB H bodies the diagnosis of α-thalassaemia trait would not have been made.

The conventional search for HB H bodies can be tedious and it has been found possible to make the HB H body test more sensitive. Blood from just below the bony crest of a column of blood centrifuged in a Wintrobe tube is mixed with brilliant cresyl blue stain and incubated at 37°C for three hours. It is usually then possible to find several HB H-containing cells in each high-power microscope field. It is only to be expected that if one is searching for an unstable haemoglobin such as HB H, the best yield would be in the fraction of blood rich in reticulocytes and young red cells. It is not being suggested that every person being screened for abnormal haemoglobins should have an HB H body test. However, if one studies those patients with thalassaemic blood films, abnormal red cell indices, and a normal HB A2 level, there should be a good yield of α-thalassaemia trait in this group. Experience at this hospital suggests that for every two cases of β-thalassaemia trait there will be one of α-thalassaemia trait in a community containing Negro, Asian, and Mediterranean immigrants.

We are, etc.,

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Clinical Diagnostic Process: An Analysis

SIR,—May I comment on the interesting paper by Dr. D. J. Leeper and others (15 September, p. 569). These authors purport to show that the diagnostic process as a monolithic structure does not exist. They show clear patterns of diagnostic behaviour which differ between senior and junior medical personnel, suggesting that the mental procedures used by them should not show basic similarities. Certainly this has been shown by Elstein et al.,2 though their findings were based on simulated consultations which, Dr. Leeper and his colleagues point out, have to be interpreted with caution. The diagnostic processes of general practice do show common patterns of mental approach,3,4 which strongly suggest the possibility of adapting the computer technique based on Bayesian probability.5 This not only may have application to future computer technique but, more importantly, helps to define general practice diagnostic pathways so that they may be taught more effectively, that they may be standardized,6 and that they may also allow general practitioners to discover areas of diagnosis which may be safely and profitably delegated to non-medical members of their team. I am, etc.,

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8. SIR,-Serious complications arising from the use of intravenous catheters are well known. Commonly infection and phlebitis occur, while less well-known complications include separation of the sheath of catheter from the needle,1 catheter tips cutting out of veins,2 and catheters knotting within the veins.3 Brachial plexus damage has been recognized as a complication of percutaneous subclavian vein puncture but does not seem to have been previously reported in connexion with the introduction of intravenous catheters via the basilic vein.

A patient was admitted to the wards in an emaciated state and it was decided that parenteral feeding was necessary preoperatively. The technique used on our unit was to introduce a long polyethylene catheter via the basilic vein until its tip was estimated to be lying in the subclavian vein. The higher blood flow in these larger veins prevents the phlebitis so often associated with parenteral feeding solutions. The catheter tip was shown to be in the vein by allowing blood to run back down the catheter. Twelve hours after the infusion was started the patient complained that he could not move his arm. He did not complain of any discomfort. On examination extravasation of a clear, serous fluid was noted. On palpation a hard, indurated area was present in the subclavian fossa. The catheter was observed to have a flaccid paralysis and a dense sensory level from the subclavian fossa to T1. Eight weeks later the neurological signs were unchanged and electromyography confirmed severe axonal degeneration.

The brachial plexus palsy in this patient was probably due to extravasation of the irrigant following perforation of the vein by the catheter tip. Such perfusion has previously been reported.4 In one of Adar's cases the right atrium was perforated. Once extravasation of the highly irritant irrigant has occurred, irreparable damage would be expected in that region.

In conclusion, one wonders if splitting the arm to allow flexion might not reduce excessive movement of the catheter tip and hence the risk of perforation. I am, etc.,

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Health as a Quantifiable Property

SIR,—In spite of efforts to prevent disease, medicine has tended to become a huge industry, gravely neglecting the potentialities of the individual to cause or cure disease and the social milieu that affects that potentiality. More attention to the quality of the individual1 would suggest, enormously reduce the expensive problems of m'dicine. Efforts to measure one's quality and health status may help to assess the potentialities of the individual2 for disease.3 They risk overcomplexity even though practical measures have been proposed4 for estimating rates of cure. These efforts will fail so long as we lack a positive and usable definition of health.

I have elsewhere defined health ecologically as "a continuing property that can potentially be measured in terms of one's ability to rally from challenges to adapt ("insults")5...Rallying is measured by the speed of recovery and success (the level of health reached afterwards). Established tolerance, function, and other rallying tests measure parts of health in absolute units. Present health may reflect amount reserves; or it may be lowered to some threshold as with a child malnourished and therefore vulnerable to a relatively small decrease; or health may be due to compensation, or accompany some genetic or ontogenetic predisposition.

If a property is charitable, it is potentially more capable. Even speculative charting can be realistic (fig. 1a, U). General health will rise with maturation and sink with senescence. Superimposed on the curve will be circadian and other physiological rhythms, and frequent fluctuations during coping episodes, some of which will be labelled as