Guy's, The London, Haywards Heath, Bart's, and the Brook do not yet have E.M.I. scanners, although they are established centres with an immediate claim for this machine.

You seem to think (incorrectly in our view) that neurological centres do not deal with patients with head injury, strokes, and dementia, all diseases in which the E.M.I. scanner can be profitably deployed. If these diseases are as common outside recognised neurological centres as you imply, then they constitute a pressing reason for having more neurological centres each properly equipped with one or more E.M.I. scanners.

The warning example is not the isotope scanner, but the E.E.G. machine which in its day was parked away in mental hospitals devoid of the skill or experience to make use of it. The community will demand that the revolutionary E.M.I. scanner be put in the hands of those who have the skills and knowledge to use it most wisely.

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SKIN WRINKLING AND NERVE FUNCTION

SIR,—After Dr Moynahan's lucid explanation (Oct. 12, p. 907) of how skin wrinkling in cystic fibrosis varies with the salt concentration in sweat, are we to conclude that the loss of wrinkling due to peripheral nerve lesions may simply be a result of interruption of sympathetic fibres ?

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CYTOMEGALOVIRUS IN NON-B POST-TRANSFUSION HEPATITIS

SIR,—Dr Prince and his colleagues (Aug. 3, p. 241) demonstrated that 71% of cases of post-transfusion hepatitis are due neither to hepatitis-B virus (H.B.V.) nor, in view of the long incubation period and other clinico-epidemiological features, to hepatitis-A virus (H.A.V.). Cytomegalovirus (C.M.V.) was ruled out by the absence of a significant difference between antibody-response rates of non-B-hepatitis cases (19% response) and patients without hepatitis (24% response). Only pre-transfusion and 4–6 months post-transfusion specimens were tested, however, for C.M.V. antibody.

We have studied 32 patients (see accompanying table) prospectively for 2 to 8 months after open-heart surgery (mean 4·1 months), at weekly intervals for 3 weeks and at monthly intervals thereafter. Patients were evaluated clinically and virologically for infections possibly transmitted in blood: H.B.V., C.M.V., herpes simplex virus (H.S.V.), Epstein-Barr virus (E.B.V.), measles virus, and adenovirus.

Buffy-coat leucocytes, red cells, and urine were cultured in human fibroblasts.[1] HB_sAg and anti-HB_s were measured by the direct solid-phase radioimmunoassay (R.I.A.) techniques.[2] Immunofluorescent antibodies against E.B.V. capsid antigen (V.C.A.) and early antigen (E.A.) were determined in the laboratory of Dr W. Henle. Complement-fixing antibodies against C.M.V. (AD-169) strain, H.S.V., measles, and adenovirus antigens were measured by the microtitre technique modification of the Kolmer technique (California State Health Department). Hæmagglutination-inhibiting antibodies to measles were measured by Dr D. W. Barry.

Hepatitis was defined as a rise in serum-transaminase above 100 units per ml. on 2 occasions. Causes of liver dysfunction, such as drugs, congestive heart-failure, and reduction in liver perfusion during surgery, were excluded by history and appropriate examinations. A primary anti-HB_s response was defined as a negative R.I.A. titre before operation, and a positive titre more than 4 weeks after operation. An anamnestic anti-HB_s response was defined as a rise in binding in the R.I.A. test of more than 2000 c.p.m. or as a conversion from negative to positive anti-HB_s titre in the first 4 weeks after operation. Passive transfer of anti-HB_s was judged to occur when the R.I.A. anti-HB_s titre of the patient's blood before operation was negative, when the residual blood in the heart-lung machine had high R.I.A. anti-HB_s titre, and when anti-HB_s titre declined after operation to a negative titre over the next 2–3 months.

Post-transfusion hepatitis, as defined by S.G.O.T. elevation above 100 units per ml., was diagnosed in 9 (28%) of the 32 patients. Mean onset of transaminase increase was 76 days after surgery. None of the 9 patients had an incubation period less than 37 days, and all represent, therefore, long-incubation-period hepatitis. Only 1 patient was icteric. 3 of 9 patients with hepatitis had an anamnestic anti-HB_s response compared with 3 of 23 without hepatitis. In the study of Prince et al., 23% of patients with anamnestic anti-HB_s response developed hepatitis; thus, at most, 1 patient in our study had hepatitis B. The analysis of serological responses and virus isolation data (see table) revealed that only antibody responses to C.M.V. were significantly more frequent in patients with hepatitis compared with the rest of the group—5 of 9 (55·6%) *v.* 3 of 23 (13%) ($P=0·023$).

The onset of C.M.V. antibody rise (mean $\log_2$ rise 3·3 $\log_2$) preceded hepatitis in 3 patients, whereas it followed hepatitis in 2 patients (mean $\log_2$ rise 2 $\log_2$). Although, in the latter 2 patients, the time relationship and the magnitude of antibody response did not particularly suggest a relationship between C.M.V. infection and hepatitis, this might be related to antigenic differences between the C.M.V. strain used for testing (AD-169) and the C.M.V. strains responsible for hepatitis. Although we have no direct evidence on this

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RATES OF ANTIBODY RESPONSE TO CYTOMEGALOVIRUS, HERPES-SIMPLEX VIRUS, ADENOVIRUS, MEASLES VIRUS, EPSTEIN-BARR-VIRUS CAPSID, AND EARLY ANTIGEN, HEPATITIS-B ANTIGEN, AND VIRUS ISOLATION RATES IN RELATION TO POST-TRANSFUSION HEPATITIS

Clinical picture	No. of patients	Serological responses (%)								Virus isolation	
		C.M.V.	H.S.V.	Adeno	Measles	E.B.V. V.C.A.	E.B.V. E.A.	Anti-HB_s	Passive transfer of anti-HB_s	C.M.V.*	H.S.V.†
Hepatitis	9	5/9 (55·6)	1/9 (11·1)	2/9 (22·2)	0/9 (0)	0/3 (0)	0/3 (0)	3/9 (33·3)	2/9 (22·2)	0/9 (0)	1/9 (11·1)
No hepatitis	23	3/23 (13·0)	5/23 (21·7)	1/23 (4·3)	4/23 (17·4)	1/10 (10)	0/10 (0)	3/23 (13·0)	4/23 (17·4)	2/23 (8·6)	0/23 (0)
Total	32	9/23 (28·1)	6/32 (18·7)	3/32 (9·4)	4/32 (12·5)	1/13 (7·7)	0/13 (0)	6/32 (18·7)	6/32	2/32 (6·2)	1/32 (3·1)

* C.M.V. was isolated from the " buffy-coat " leucocytes collected from one patient just before operation and from another patient one week after operation.

† H.S.V. was isolated from the red-cell layer from blood collected 3 weeks after operation. The patient had herpetic lesions on his forearm at that time.

point, antigenic heterogeneity of cytomegaloviruses is suggested by neutralisation with sera of infants [3] and by complement-fixation tests with sera of donors undergoing plasmapheresis.[4]

C.M.V. has been associated with some degree of liver damage in both congenital and postnatal infections.[5] The association has been based usually on the demonstration of antibody rise and virus isolation from urine. In renal-transplant recipients, high C.M.V. antibody titres correlate with hepatitis.[6,7] In 1 of our patients, C.M.V. was isolated from a liver biopsy.[7]

Purcell et al.[8] found only insignificant difference in the C.M.V. seroconversion-rate between patients with post-transfusion hepatitis and controls. In their study, a relationship of C.M.V. to hepatitis might have been obscured by the high seroconversion-rate among pre-transfusion antibody-negative patients. In our study, patients without pre-transfusion C.M.V. antibody had a seroconversion-rate (25%) as low as patients with pre-transfusion antibody (24%). The role of C.M.V. in hepatitis deserves further study by newer serological techniques and also by immunohistochemical techniques to identify C.M.V. antigen in liver biopsy specimens.

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VIRUS-LIKE PARTICLES IN HEPATITIS A

SIR,—We read with interest the report by Dr Almeida and her colleagues (Sept. 28, p. 748) and the reply from Dr Locarnini and others (Oct. 26, p. 1007). Although we concur with Dr Almeida about the possibility that serum-antibody responses to ætiologically unrelated antigens may be stimulated in acute viral hepatitis, we believe that the 27 nm. virus-like particles first observed by Feinstone et al.[9] in stool of human volunteers experimentally infected with the MS-1 strain of hepatitis-A virus, recovered by ourselves from acutely ill patients with hepatitis A during a common-source epidemic in Arizona (in the press), and now similarly observed in Australia by Locarnini and his colleagues, are more specifically related to hepatitis A than might be inferred from the comments of Dr Almeida and her associates. As with the Australian workers, we have established, by extensive exchange of reagents, the serological identity between the 27 nm. particles seen by us in Arizona and those first observed by Feinstone et al. The identity of the particles recovered in the U.S.A. and Australia, therefore, seems well supported. Further, serum-antibody rises to our particles do not occur between acute illness and convalescent phase sera from cases of hepatitis B, thereby further supporting the specificity of their association with hepatitis A. Chimpanzees inoculated with human stool filtrate rich in these 27 nm. particles have developed viral hepatitis with concomitant excretion of identical particles and have seroconverted to the same particles. Additionally, we have induced viral hepatitis in marmosets after inoculation of particles derived from $CsCl_2$ banding experiments.

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LEGAL STATUS OF THE UNBORN

SIR,—Professor Emery's letter (Sept. 21, p. 715) prompts this brief note of clarification. Although it is true that a bundle of quasilegal rights attach retrospectively to a liveborn child in the United States, the possibility of a tort action for "wrongful life"[1] appears remote. Such lawsuits were initiated on at least four occasions [2] before the liberalisation of abortion laws in the United States.[3] I know of one lawsuit decided after the change in the abortion laws.[4] All five published decisions refute the notion that an action for wrongful life should be countenanced. Thus, common law decisions in America are in ad-hoc agreement with the position of the British Law Commission as reported by Professor Emery.

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SEASONAL VARIATION OF HISTOLOGICAL OSTEOMALACIA IN FEMORAL-NECK FRACTURES

SIR,—I have been following the correspondence between Dr Hodkinson and Dr Marshall and Professor Nordin (Aug. 24, p. 463, and Oct. 26, p. 1008) arising out of the article by the latter workers (July 10, p. 84). Could I be allowed to make some fairly general comments about what it seems to me is at the root of the discussion, and is alluded to in the final paragraph of the last letter from Marshall and Nordin?

It is not true that experimental results can prove a hypothesis to be wrong but not right. We are concerned necessarily in science with inductive inference, being that basic form of inference where uncertainty is always present. This is in contrast with deductive inference, which is mainly of abstract interest. The main purpose that statistics serve is the provision of techniques for making inductive inferences and for measuring the degree of uncertainty associated with such inferences. Any such measure always depends on the model used to specify the process under study. If the model is inappropriate then measures of probability are similarly inappropriate, as has been stressed by, for example, the Editor of the *New England Journal of Medicine*.[5]

The example under discussion can be compared with the classic attempt to predict the results of an American presidential election in the 1950s by asking the voting intentions of a random selection of telephone subscribers by ringing them up. The model implicitly stated that these people were representative of the voting population as a whole. Hence a "prediction" of the result could be made with appropriate limits of confidence, but the result came and was so far outside these limits that, yet again, the lesson was learned that the full implications of the (implicit or explicit) model should be understood in terms of assign-

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