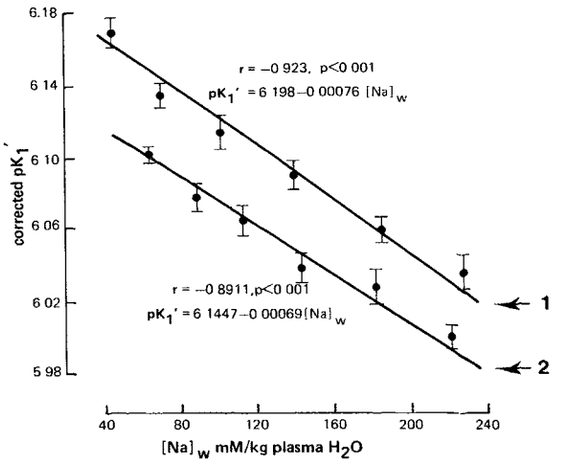

Fig. 2

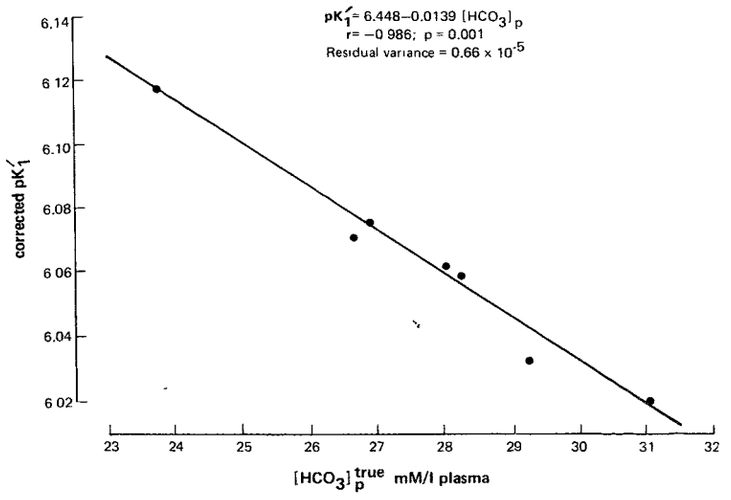

Fig. 3

Fig. 2 also shows that difference in plasma bicarbonate levels are likely to alter pK'_1; and this is confirmed in fig. 3 which presents the pK'_1 values determined on fresh *undiluted but similarly tonometred* plasma from a healthy male on seven different occasions. We cannot explain why this should be so.

Data will be presented in full elsewhere.

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AUTOMATIC BLOOD-PRESSURE MEASUREMENT

SIR,—An editorial earlier this year[1] referred to the availability of an automatic blood-pressure (BP) machine ('Vita Stat') in a London store and stated that the scheme should be encouraged "provided that the measurement is reasonably accurate". We were interested in such a machine, especially for outpatients departments, because research at another London teaching hospital had shown how few patients attending hospital, as inpatients or outpatients, have their BP taken.[2]

An opportunity to evaluate the machine arose when a health fair was held for employees of a local psychiatric hospital. BP measurement was one of the services offered. All employees who had their BP measured had it done twice—once on a vita stat machine and once on a random-zero sphygmomanometer.[3] The order in which the measurements were taken was partly left to the discretion of the subjects.

MEAN SYSTOLIC BP RECORDINGS (mm Hg) SHOWING DIFFERENCES BETWEEN OBSERVERS AND BETWEEN RECORDINGS TAKEN FIRST OR SECOND ON SPHYGMOMANOMETERS OR VITA-STAT MACHINE

Observer	Vita-stat		
	Machine first	Machine second	Difference
A	146·4 (n=22)	140·6 (n=17)	−5·8 (p<0·05)
B	146·3 (n=64)	141·9 (n=63)	−4·4 (p<0·01)
C	148·5 (n=76)	140·4 (n=81)	−7·9 (p<0·01)
	Sphygmomanometer		
Observer	Sphyg. first	Sphyg. second	Difference
A	126·4 (n=17)	129·5 (n=22)	+3·1 (NS)
B	107·6 (n=63)	114·1 (n=64)	+6·5 (p<0·01)
C	123·5 (n=81)	127·5 (n=76)	+4·0 (p<0·01)

Three observers recorded BPs on two random-zero sphygmomanometers. The considerable inter-observer variation is shown in the table.

The table also shows that the mean recordings from the vita stat automatic machine were higher than the mean observer readings for each observer. This suggests a systematic tendency of the automatic machine to record high (even if it does abolish inter-observer variation).

The data suggest that use of the machine may increase BP—i.e., high recordings are not simply a measurement error. For each observer, the mean reading was higher when the sphygmomanometer was used for the second recording, after the machine. This may be because individuals with high BP used the machine first. In addition, the automatic machine may cause anxiety which is reflected in the raised BP.

Beevers et al.[4] found that automatic machines tend to read high, and so did L. N. Jones and colleagues in a paper given at the Fifth National Conference on High Blood Pressure Control, held in Arlington, Virginia, in April, 1979. Until the hypothesis that such machines cause BP to rise has been refuted, their widespread use cannot be recommended.

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HYPERPHENYLALANINÆMIA AND PREGNANCY

SIR,—The need for further studies on dietary treatment of pregnant women with phenylketonuria (PKU) is emphasised in your editorial[1] and by Dr Buist and colleagues (Sept. 15, p. 589). Our experience in Michigan points to a problem not mentioned in these contributions.

In the twelve years 1967–78 the Michigan Department of Public Health has found 80 newborns with PKU and 77 with hyperphenylalaninæmia (hyperPhe) in 1 759 510 screenings. PKU incidence was approximately 1 in 23 000; hyperPhe was 1 in 22 000; overall abnormal incidence was 1 in 11 000. We have noted, in both male and female infants with hyperPhe, that, as they grow to childhood and adolescence, their blood Phe levels have fallen—perhaps because of reduction in ingested protein per kilogram, maturation of metabolic processes, or for other reasons. So blood screening of women in the childbearing years may miss some of these and the heterozygotes[2] at risk of having affected infants.

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It would have been of interest if Buist et al. had listed the cord or newborn Phe blood levels in their series, along with the maternal levels. Cord blood Phe levels seem much higher than (perhaps twice as high as) maternal levels.[3,4] Does prenatal serological blood testing of adults identify a new group of women who tested normally for Phe at birth?

It seems likely that slightly positive newborn tests are obscured by maturation and that adult normal Phe levels will be normal while pregnancies remain at risk. Certainly caution is needed in counselling these groups of women on the risks of a damaged newborn. We must search for increased blood Phe under all circumstances and under a variety of guises.

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B-CELL ACTIVATION IN ANGIOIMMUNOBLASTIC LYMPHADENOPATHY AFTER IMMUNISATION WITH MULTIVALENT PNEUMOCOCCAL POLYSACCHARIDE VACCINE

SIR,—Pneumococcal infection remains a small but definite hazard in patients who have had their spleens removed, in immunocompromised hosts, and in patients with malignant disease. For this reason, immunisation with polyvalent pneumococcal polysaccharide vaccine ('Pneumovax') in such patients has received recent attention.[5]

While evaluating this vaccine in patients with immunological disorders or malignant disease we immunised two patients with angioimmunoblastic lymphadenopathy (AILD) in a quiescent phase. Both had a sudden reactivation of their disease after immunisation.

A 75-year-old woman presented with fevers, night sweats, cough, anorexia, and weakness, with diffuse lymphadenopathy and hepatosplenomegaly. Investigations revealed Coombs positive hæmolytic anæmia and polyclonal gammopathy. Lymph-node biopsy confirmed AILD. After a course of chlorambucil, vincristine, and prednisone symptoms and physical findings resolved. On maintenance therapy with prednisone her disease was completely suppressed for 4 months, but 3 weeks after receiving 0·5 ml pneumovax she had a sudden onset of high fevers, adenopathy, and recurrent organomegaly progressing to renal failure and pancytopenia despite aggressive support and combination chemotherapy. Immunological studies before death demonstrated an IgG kappa monoclonal gammopathy of 654 mg/dl with free kappa chains in the urine. Permission for necropsy was refused.

The second patient was a 62-year-old man who presented with spiking fevers and diffuse lymphadenopathy with an IgG monoclonal gammopathy. Lymph-node biopsy was consistent with AILD. He had Coombs positive hæmolytic anæmia and thrombocytopenia despite adequate megakaryocytes on marrow aspiration and biopsy. Because of incomplete control with corticosteroids, he received a short course of doxorubicin and vincristine. He was maintained on tapering doses of prednisone alone for 5 months. At a time of inactivity of the disease, the patient received 0·5 ml pneumovax while on 10 mg/day of prednisone. 3 weeks later he suddenly had shortness of breath, fever, diffuse adenopathy, and organomegaly. His disease course was poorly controlled by high-dose corticosteroids, and bacterial sepsis developed. A repeat biopsy revealed immunoblastic sarcoma. After treatment with cyclophosphamide, vincristine, doxorubicin, and prednisone, he is once more in remission.

AILD is unusual in that it has features of both a collagen-vascular disease and a neoplastic process.[6,7] Decreased numbers of T-cells, hypergammaglobulinæmia, loss of delayed sensitivity, IgA deficiency, and Coombs positive hæmolytic anæmia have been noted.[8,9] The disease may show a malignant course with poor survival.[10]

Some investigators have suggested that AILD results from unrestrained B-cell stimulation by exogenous stimuli; if so, immunisation by an antigen such as pneumococcal polysaccharide, might further accelerate the B-cell dyscrasia. Sudden deterioration after immunisation pneumovax may be the clinical consequence. The close temporal relation between immunisation and reactivation of the underlying disease does not prove cause and effect. However, because of the abnormal immunological indices noted in AILD,[8,9] which may enhance B-cell activation perhaps by loss of T-cell suppressor function, pneumococcal polysaccharide immunisation should be considered with caution in such patients.

We thank Ms Marie Sweeny for secretarial assistance.

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LYMPHOCYTOTOXIC ANTIBODIES IN LABORATORY PERSONNEL EXPOSED TO SLE SERA

SIR,—Lymphocytotoxic antibodies (LCTA) are found in most patients with systemic lupus erythematosus (SLE). They are also found in the consanguineous and non-consanguineous relatives of patients with SLE,[1-3] and they have been postulated to be markers of exposure to an environmental agent which may be important in the pathogenesis of this disease. Other data[4] suggest that laboratory personnel working with SLE have an increased incidence of antibodies to DNA.

LCTA were, therefore, measured[1,5] in coded sera from laboratory personnel, primarily technicians and research physicians, who were regularly exposed to SLE sera. Control sera were obtained from general hospital and nucleic-acid laboratory personnel having little or no contact with SLE, physicians caring for SLE patients without exposure to patient's sera, lymphocyte-typing laboratory technicians not handling SLE sera, non-medical personnel, and 15 patients with SLE. Results were analysed by Fisher's exact test. Multiple sera were run in duplicate to control for any observer variability.

The frequency of lymphocytotoxic activity was significantly higher in the laboratory personnel who handle SLE sera than in all other control groups ($p<0.005$) but was significantly less than that found in SLE patients ($p<0.001$). 5 laboratory personnel were examined for LCTA before the start of work in the SLE laboratory and again after six months. Significant LCTA developed in 3 individuals after they had been working with

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