Wet and dry bulb temperatures were recorded in examination rooms at the midpoint of each screening session in a number of temporary centres attended by 2178 males aged 40-65 years (approximately 75% response rate). Temperatures ranged from 11-18°C wet and 14.4-25°C dry bulb. Blood pressure was recorded using the London School of Hygiene and Tropical Medicine sphygmomanometer with examinees seated and the appearance and disappearance (phase V) of the Korotkoff sounds were used to observe the mean of two consecutive readings.

The table gives correlation coefficients between wet and dry bulb temperatures and systolic and diastolic blood pressure. A normal

Blood pressure and ambient temperature: correlation coefficients between wet and dry bulb ambient temperatures and systolic and diastolic blood pressure (phase V) in 2178 males aged 40-65 at work in west central Scotland

Age (y)	No	Systolic		Diastolic	
		Wet	Dry	Wet	Dry
40-44	100	0.089	0.028	0.198*	0.190*
45-49	311	0.091	0.093*	-0.036	0.052*
50-54	519	0.019	0.085*	0.006	0.114*
55-59	526	0.045	0.113*	0.001	0.069
60-64	429	0.037	0.125*	0.009	0.089*
65 and over	293	0.045	0.174*	0.063	0.167*
Total	2178	0.033	0.112	0.006	0.106

*Significant at 5% level.

distribution of ambient temperatures could not be assumed, so a non-parametric method was used.³ The analysis shows a significant positive correlation between systolic and diastolic blood pressure and dry bulb temperature.

The data raise questions about the role of blood pressure in the homeostatic control of body temperature and the possible effects of impairment of that mechanism in hypertension, and suggest the need to investigate outcome in the long-term follow-up of this cohort and of other cohorts over a wider range of ambient temperature, including the recording of examinees' temperatures. There could also be implications for the validity of cross-cultural comparisons between populations in tropical and temperate climates.

The multiphasic screening studies reported here were supported by grants from the Scottish Home and Health Department and the Renfrewshire King Edward Memorial Trust.

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¹ Hawthorne VM, Greaves DA, Beevers DG. *Br Med J* 1974;iii:600-3.

² Hawthorne VM, Beevers DG, Gilmour H, Stewart GM, Watt H. *Clin Sci Mol Med* 1976;51:6535-65.

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Paraproteins and non-Hodgkin's lymphoma

SIR,—Your leading article (2 February, p 273) did not point out the frequent incidence of paraproteinaemias in non-Hodgkin's lymphoma other than widespread lymphoplasmacytoid lymphoma and chronic lymphocytic leukaemia.

A study of non-Hodgkin's lymphomas based

upon the Kiel classification¹ was carried out at Oxford. Presentation sera and concentrated urines were examined for paraproteins using agarose gel plates and standard antisera. Paraproteins were detected most frequently in the low-grade lymphomas and in particular in the lymphoplasmacytoid lymphomas involving bone marrow. However, there was an appreciable incidence of paraproteins detectable in patients who fulfilled the criteria neither for Waldenström's macroglobulinaemia nor for chronic lymphocytic leukaemia. Thus in low-grade lymphomas the lowest incidence of paraproteins was found in patients with follicle-centre-cell lymphomas and the highest in diffuse lymphocytic lymphomas. In the high-grade lymphomas the most frequent rate of detection was among patients with immunoblastic lymphoma, and here it is of interest to note that the IgA paraproteins (one as part of a double clone) were detected in patients with small-bowel lymphoma. The incidence of paraproteinaemias in other high-grade lymphomas was much less frequent but still well above the expected incidence in this age group. Overall, IgG paraproteins were found almost as frequently as IgM paraproteins, and light chains were detected in urine in association with serum paraproteins but in eight cases alone.

Among patients with widespread lymphoplasmacytoid lymphoma the IgM paraprotein was from 10 to 20 g/l but was frequently lower in the diffuse lymphomas and in the chronic lymphocytic leukaemias. The levels of IgG paraprotein were also 10-20 g/l in most instances, although in one case (a patient with chronic lymphocytic leukaemia) over 40 g/l. Levels of IgA paraprotein were 6-10 g/l.

The incidence of hypogammaglobulinaemia in non-Hodgkin's lymphomas has been reported elsewhere.² There was no association between hypogammaglobulinaemia and detection of paraprotein in the high-grade lymphomas and even among the low-grade lymphomas there was only a slight increase in the tendency for suppression of polyclonal IgG and IgM in association with paraproteins and not at all for IgA. Among the 35 patients who had detectable paraproteins, 18 had no immunosuppression of the other classes.

The purpose of this particular study was to detect the incidence of the paraproteinaemias in relation to the various Kiel histopathology groups. The frequent detection of paraproteins in virtually all the low-grade lymphomas and in the immunoblastic lymphomas is of interest and could be regarded as indirect evidence for a B-lymphocyte origin in these cases.

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¹ Lennert K. *Acta Neuropath* 1975;90 suppl:1-16.

² Leonard RCF, MacLennan ICM, Smart Y, Vanhegan RI, Cuzick J. *Int J Cancer* 1979;24:385-93.

Iatrogenic collapse

SIR,—The correspondence (20 October, p 1001; 24 November, p 1367; 19 January, p 185) relating to the use of intravenous pethidine once again emphasises how much is taken for granted with this widely administered drug.

I was surprised at how few controlled studies had been performed to confirm the clinical impression that intravenous pethidine can be a severe respiratory depressant. A study by Orkin and colleagues¹ is probably the best of the few that relate to the common clinical situation where an intravenous narcotic is used as a supposedly short-acting anaesthetic providing analgesia, sedation, and relaxation in the ambulant patient. Ten healthy unpremedicated patients were given pethidine

100 mg intravenously. They found an increase in respiratory rate which persisted for the one-hour study. More importantly, the tidal volume decreased by 55% and the minute volume by 50%, though these returned to 90% of their baseline values within 15 min. They noted periodic respiration in seven out of the 10 subjects. With morphine 10 mg instead, also given intravenously, the tidal volume was depressed by only 30% and the minute volume by 20%. They questioned the value of respiratory rate as a sign of respiratory depression with pethidine and implicated the use of this index as a cause of the widespread belief that pethidine does not cause significant respiratory depression. Age and sex did not prove to be determining factors as severe periodic breathing was seen in an 18-year-old female. Jennet has noted that the interpretation of tests of respiratory depression,² is both difficult and controversial³ but that the two drugs are almost equally depressant in equianalgesic doses. Where they differ is in producing peak effects at different times and in their dose-response relationships. There is no evidence that pethidine is safer than its rivals with regard to either of these.

Concerning Dr Huntington's other points, I would be surprised if anyone sent in a yellow card recording respiratory depression with pethidine 100 mg almost 40 years after the drug's introduction.

Finally, I recommended naloxone (Narcan) as an antidote, rather than nalorphine, because it carries no risk of further respiratory depression and is now the drug of choice.

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¹ Orkin LR, Egge RK, Rovenstine EA. *Anesthesiology* 1955;16:699.

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Endocrine aspects of liver disease

SIR,—I read with interest the explanations of liver dysfunction in thyrotoxicosis summarised by Dr R G Long (26 January, p 225). He may, however, have omitted the commonest cause of disturbance of liver function now seen in this condition.

Abnormalities of liver function, especially elevation of serum transaminases and alkaline phosphatase, are common in patients presenting with thyrotoxicosis today, their incidence varying from 8% to 50%.¹⁻⁵ In these patients few of the complications mentioned in the article and in which earlier literature abound^{6,7} are in fact present, such as heart failure, thyroid crisis, "severe untreated disease," or associated liver pathology.

When liver biopsies are done in these patients few of the histological changes mentioned by Dr Long are found but more subtle changes of liver ultrastructure are seen on electron microscopy, including elongation of mitochondria, frequent metamitochondria, and hypertrophy of the smooth endoplasmic reticulum with decreased glycogen.⁸ Similar hepatic changes are evident in patients taking potent enzyme inducers and are associated with elevation of liver enzyme levels in the serum.

The plasma half life of antipyrine, whose decay in serum is known to reflect hepatic microsomal metabolism, is shortened in