Correspondence

Deoxyhaemoglobin concentrations in the detection of central cyanosis

SIR,—Although not explicitly stated in their article, Dr Geraldine Goss and her associates (March 1988;43:212–3) appear to share with others[1] the misconception that Lundsgaard and Van Slyke[2] believed cyanosis first could be detected when 5 g/dl reduced haemoglobin was present in the *arterial* blood. Lundsgaard and Van Slyke wrote, "About 5 grams of reduced haemoglobin per 100 c.c. of capillary blood appear necessary to cause cyanosis . . ."[2] They calculated mean capillary unsaturation by averaging the arterial and venous unsaturations. The difference between mean capillary unsaturation and arterial unsaturation would depend on the haemoglobin affinity for oxygen, perfusion of the tissue being viewed, and oxygen utilisation by that particular tissue. Since these factors were not reported by Dr Goss and colleagues, their data cannot be used to determine the correctness or incorrectness of the original conclusion of Lundsgaard and Van Slyke. Admittedly, one should have reservations about that early assertion after considering the fact that Lundsgaard and Van Slyke did not report analyses of arterial blood specimens on patients—only free flowing peripheral venous specimens had been analysed.

It is interesting that reanalysis of Stadie's data from 1919,[3] which did involve arterial blood sampling, would lead one to much the same conclusion as was reached by Dr Goss and her associates. None of his five patients without cyanosis had more than 1·3 g/dl arterial unsaturation while 4/27 with cyanosis had values less than 1·5 g/dl arterial unsaturation.

HL SNIDER
TM ROY
Respiratory and Environmental Medicine Division
University of Louisville School of Medicine and Veterans' Administration Medical Center
Louisville, KY40206, USA

1 Barnett AB, Holland JG, Josenhans WT. When does central cyanosis become detectable? *Clin Invest Med* 1982;5:39–43.
2 Lundsgaard C, Van Slyke DD. Cyanosis. *Medicine (Baltimore)* 1923;2:1–76.
3 Stadie WC. The oxygen of the arterial and venous blood in pneumonia and its relation to cyanosis. *J Exp Med* 1919;30:215–40.

AUTHORS' REPLY—We appreciate the interest shown by Drs Snider and Roy in our paper, and in particular their reanalysis of Stadie's data (their ref 3). Although misconceived many still believe, teach and perpetuate that 5 g of reduced haemoglobin/dl are necessary before central cyanosis is clinically detectable. In our paper we have been successful in clearly demonstrating that central cyanosis can be reliably detected at deoxyhaemoglobin concentrations of 1·5 g/dl and above.

G W BURDON
Respiratory Medicine Unit
St Vincent's Hospital
Fitzroy, Victoria 3065
Australia

Metastatic renal cell carcinoma mimicking pleural mesothelioma

SIR,—It was with much interest that we read the paper presented by Dr DR Taylor and others (November 1987;42:901–2), which reported a case of pleurisy secondary to renal adenocarcinoma but mimicking a mesothelioma. The history of previous occupational exposure to asbestos in this case suggested a primary pleural tumour. This observation permits us to recall that all secondary pleural tumours may simulate a primary tumour when evaluated radiographically, with computed tomography, and even at necropsy.

Other cancers, besides pleural pulmonary tumours, have been observed in workers exposed to asbestos—particularly gastrointestinal cancer and cancer of the larynx, but also renal cancer, where the incidence is twice that of the general population.[1] This is reported in the paper of Dr Taylor and his colleagues as well. We reported a case of renal cancer in a patient with work related pleural pulmonary asbestosis[2] and found raised concentrations of asbestos fibres in the kidney, estimated to be 2×10^6 fibres/g dry tissue. A similar observation was published in 1988 concerning an adenocarcinoma of the colon.[3] This emphasises the interest of adding mineralogical studies to the usual histological and histochemical studies.

PHILIPPE MULLIEZ
Service de Pneumologie
Hôpital Saint Philibert
F 59462 Lomme Cedex, France

1 Selikoff IJ, Hammond EC, Seidman H. Latency of asbestos disease among insulation workers in the United States and Canada. *Cancer* 1980;46:2736–40.
2 Mulliez P, Gaudichet A, Dufour G, *et al.* Cancer du rein associé à une asbestose pleuro-pulmonaire. *Presse Méd* 1985;14:2302.
3 Ehlrich A, Rohl AN, Holstein EC. Asbestos bodies in carcinoma of colon in an insulation worker with asbestosis. *JAMA* 1985;254:2932–3.

Book notices

UK Smoking Statistics. N Wald, S Kiryluk, S Darby, SR Doll, M Pike, R Peto. (Pp 197; £35.) Oxford: Oxford University Press, 1988.

This book presents comprehensive data on smoking in the UK. The tables and graphs are clear, easily used, and attractively set out. The accompanying text and explanatory notes are likewise well presented and readable. The book does not deal with the relationship of smoking to disease, nor does it cover variations in the epidemiology of smoking related diseases since smoking patterns and the contents of cigarettes have changed. Acquiring this succinct book for oneself would permit the throwing away of many "tearouts" and notes taken at lectures. I can recommend it to anyone who would find it useful to have ready access to information about the pattern of smoking in Britain over the last 100 years. It should be in the library of every hospital and medical school.