In a healthy subject HPV infection can induce a transient arrest of erythropoiesis, which does not lead to clinical symptoms because the normal erythrocyte life span exceeds that of any effect of HPV on marrow. In patients with haemolytic anaemias the haemoglobin concentration drops greatly with HPV infection, because the red blood cell life span is shortened.[2] In our case the apparent HPV aplastic crisis was similar to those in previous reports,[3] but erythroblastopenia associated with HPV in non-haemolytic anaemia has not been reported as far as we know. It seems that the reticulocytopenia induced by HPV infection can thus worsen a pre-existing anaemia. Our bone marrow findings also showed that HPV, besides its known erythroblastopenic effect, can cause a moderate and transitory decrease in the platelet count, secondary to a hypoplastic effect on the megakaryocytes. Our patient did not have any erythema arthralgia, or vascular purpura, all of which may be seen in HPV infection.[4-6]

We suggest that when a patient with a haemolytic or any other type of anaemia, presents with a rapid and unexplained fall in peripheral blood haemoglobin concentration compared with steady state values, it seems logical to look for HPV infection.

JJ LEFRERE*
H BOURGEOIS†
*Centre national de transfusion sanguine, 6 rue Cabanel, 75015, Paris, France,
†Centre hospitalier, Evreux, 27000, France.

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Evaluation of strategy for testing thyroid function applied to hypothyroidism

In their interesting account of an evaluation of a strategy for thyroid function testing, Corns and Miller[1] illustrate the particular importance of relevant clinical information accompanying requests to the laboratory.

Tests for thyroid function services have been changing rapidly over the past two decades, and in many hospitals the laboratory has had to take the initiative in the selection of appropriate tests for individual patients. Various strategies have been argued recently,[2 3] with agreement that a cost effective laboratory service can be achieved if there is cooperation between clinician and pathologist.

For 15 months we have been operating a thyroid function strategy based on ideas of Britton *et al*,[2] but using free thyroxine (Amerlex-M Free T4 RIA kit) as the primary screening test and thyroid stimulating hormone as a discretionary test. The strategy requires a set of decision aiding reference ranges and levels. For detection of hypothyroidism, the relevant values were: euthyroid reference range 9·9–23·7 pmol/l (0·77–1·84 ng/100 ml); a therapeutic reference range for patients receiving thyroxine 12·9–30·8 pmol/l (1·0–2·39 ng/100 ml), and a further decision aiding range 12·3 pmol/l (0·9 ng/100 ml) to select thyroid stimulating hormone in patients presenting with features of hypothyroidism. Thyroid stimulating hormone was also assayed with free thyroxine as the primary investigation, whether or not requested in the following: paediatric cases; patients receiving lithium, amiodarone, or fenclofenac; pregnant patients; those with carcinoma of the thyroid; and goitre with antibodies (we called all these "the problem group.").

Continuous review of our results over 12 months showed that:

(i) 22 cases of thyroid stimulating hormone were not tested because, in the absence of clinical details, the patient had not been allocated to the "problem group." Subsequent testing showed that nine of these patients had raised thyroid stimulating hormone values.

(ii) Fifty six patients with low free T4 values had normal thyroid stimulating hormone results, and all of these were acutely sick in patients with no features of thyroid disease.

(iii) Three patients with repeatedly normal serum free T4 concentrations had consistently raised thyroid stimulating hormone values. They all had clinical features of hypothyroidism. On subsequent testing clinically important titres of antithyroxine antibody were detected in the sera (anti-T3 was also detected in one patient). Reassay of hormone after precipitation of interfering immunoglobulin[4] gave more accurate free T4 values (table).

We agree that relevant clinical details with requests for thyroid function testing are necessary to the successful management of laboratory testing strategy.

Screening for thyroid disease in acutely ill patients with no suspicion of thyroid disorder seems to be wasteful of essential resources, and the results can be misleading. Accordingly, we requested house doctors not to screen patients during acute illness.

It seems that interfering antihormone antibodies may occur more commonly than originally suspected, and both laboratory staff and clinicians should be alert to this possibility when confronted with anomalous results.

AI POLANSKY
District Laboratory, The General Hospital, South Shields, Tyne and Wear, NE34 0PL

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Table *Thyroid function test*

Case No	*First free T4 (pmol/l)*	*Thyroid stimulating hormone mu/l*	*Percentage counts precipitated as antibody*	*Supernatant free T4 (pmol/l)*	*Clinical details*
1	24·7 (1·92)	>60	49	3·6 (0·3)	"Puffy face" look, myxoedematous
2	11 (0·85)	15·5	11	8·0 (0·62)	Gaining weight, enlarged thyroid
3	10·1 (0·78)	31·9	13	4·5 (0·35)	Deep voice, slowing up, myeloma

Patients with circulating antithyroxine antibody (values in parentheses given in ng/100 ml.)

J Clin Pathol 1986;39:1279–1280

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Simplified techniques for identifying *Campylobacter pyloridis*

Recent correspondence has focused on simplified techniques for identifying gastric *Campylobacter pyloridis* on tissue sections as the Warthin-Starry stain is both unpredictable and time consuming. Pinkard *et al*[1] suggested phase contrast microscopy and Walters *et al*[2] suggested fluorescence staining with acridine orange. Although we agree these are simple techniques, they rely on having fluorescence or phase contrast microscopes easily available, which in many hospitals is not feasible.

We favour a modified Giemsa technique that is simple, permanent, and quick to perform with the organisms easily visible under light microscopy (figure). Paraffin embedded sections are routinely dewaxed and taken to water and then incubated in 2% Giemsa solution in distilled water for 30 minutes at room temperature. After rinsing in tap water the sections are quickly dehydrated through ethanol solutions before being cleared with xylene and mounted in DPX.

To check on the accuracy of the modified Giemsa stain in identifying *C pyloridis* a comparison between Giemsa and Warthin-Starry stained sections in 35 patients was made by a single histopathologist. No difference was found in the rate of identification for *C pyloridis*, with many of the Giemsa stained sections being easier to interpret. The technique is thus quick, simple, possible in all laboratories and as accurate as the Warthin-Starry stain, which it has replaced in our laboratory.

SALLY F GRAY
JUDITH I WYATT
*BJ RATHBONE
*Departments of Pathology and *Medicine, St James's University Hospital, Leeds LS9 7TF.*

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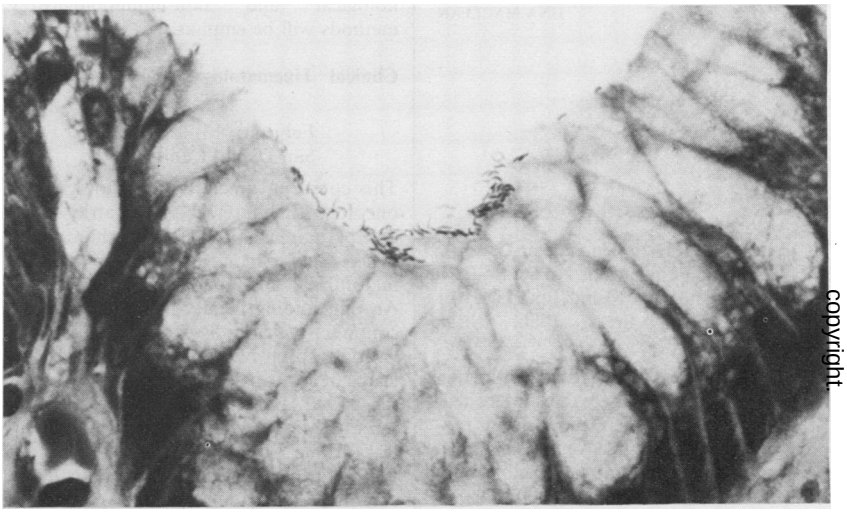

Book reviews

Atlas of Cancer in Scotland 1975–1980. Incidence and Epidemiological Perspective. Ed in Scotland: I Kemp, P Boyle. IARC Scientific Publications no 72. (Pp 282; £35.) Oxford University Press. 1985. ISBN 92 832 1172 3.

This costly and ambitious atlas of cancer incidence is the outcome of collaboration between Scotland's cancer registries and the International Agency for Research on Cancer, a WHO organisation based in Lyon, France. For the benefit of foreign readers, the substantive core of the book is preceded by brief chapters on Scotland and its people, with rather special emphasis on diet, alcohol intake, and tobacco consumption. The book is a modern manifestation of what was once called "geographical pathology."

The main data are drawn from the five separate regional cancer registries, which differ somewhat in their techniques of registration, especially in the extent to which they depend on discharges recorded by the Scottish Hospital Statistics Scheme (SMR 1). The authors claim that the registration system is now efficient, being subjected to several internal checks that take death certification into account. It is admitted, however, that there can be weak links in a chain, dependent on the assiduity of numerous hospitals. The material is presented according to Scotland's 56 local government districts and four main cities. For each cancer site the male and female incidence is first described, it is next compared with that in other parts of the world, then examined for statistical evidence of clustering among adjoining districts, finally the cancer is discussed briefly in terms of possible explanations or risk factors. The maps are both in colour, on a relative scale, and in black and white, on an absolute scale. Accompanying tables detail crude rates, age, standardised rates, and assessments of which district rates differ significantly from those of the rest of Scotland.

For a full appreciation of the technical features of this atlas, readers must carefully study Appendix II, which provides an explanation of the advantages of the absolute and relative scales, an account of why the red and green colour notation was chosen, and very important notes on the way of calculating the radomness or otherwise of observed spatial patterns of incidence.

Clearly these maps and the data on which