pylori, may cause chronic active gastritis. Like C pylori, the number of organisms can probably be suppressed but complete eradication will probably be difficult. Was case 2 endoscoped and assessed symptomatically at a later date. Analysis of any organisms may well have been detectable. Dr Logan and his colleagues may be right that “*Gastrospirillum hominis*” causes non-ulcer dyspepsia, but larger studies are needed, and I think it is still premature to draw conclusions from single patient histories.

**Lack of significant effect of therapeutic propranolol on measurable platelet function in healthy subjects**

Pamphilon and colleagues have shown that propranolol, a non-selective β blockers, does not significantly inhibit platelet function when therapeutic doses are administered to healthy subjects.1 They suggest that a different response might be obtained in patients with cardiovascular disease and hyperactive platelets. Some additional comments may be of significance.

The authors discuss the possibility that β blockers exert their action in situations where circulating catecholamine concentrations are very high. We assessed the effect of acute hypoglycaemia (during insulin stress tests) on various monocyte and platelet variables in healthy subjects who had been given either placebo, nadolol, or propranolol, orally, for 10 days.2 These non-selective β blockers significantly inhibited some of the catecholamine-mediated effects (hypokalaemia, a rise in factor VIII), but only propranolol had a marginal inhibitory effect on platelet aggregation.

In another study we found that platelet aggregation induced by adrenaline was significantly decreased in samples obtained from patients with a diagnosis of acute myocardial infarction or ischaemic heart disease who were taking β blockers when compared with a similar group of patients not taking such medication.3 Beta blockade may, in fact, be associated with platelet antiaggregatory effects but that these may only be elicited in conditions associated with pronounced platelet hyperaggregability such as myocardial infarction and ischaemic heart disease.

There are also important methodological considerations in assessing the effect of drugs on platelet function. A platelet function test (for example, whole blood platelet aggregometry (impedance or “free” cell count techniques) allows the effects of drugs on platelet, red, or white cell interactions to be measured. This has occasionally resulted in a better definition, even at lower doses, of the effects of several drugs, such as nifedipine, diprydamole, heparin and ethanol, on platelet aggregation.4 Mean platelet volume, measured by very sensitive techniques (using a Coulter C256 channeler), may also be another very sensitive method to assess the effect of a variety of agents on platelets.5


**Dr Pamphilon and colleagues:**

The comments of Barradas and Mikhailidis are of interest and point to the relevant mechanism whereby β blockers may exert a beneficial effect in patients with myocardial infarction and ischaemic heart disease—that is, modulation of platelet hyperaggregability in susceptible subjects.1 The observation that propranolol but not metoprolol enhances platelet aggregation concludes with a final caution.


**Leukaemic phase of mantle zone (intermediate) lymphoma: its characterisation in 11 cases**

In his parody of lymphoma classifications a distinguished editor of the *Journal of Clinical Pathology* reflected the confusion and cri- nicism of the average pathologist at the per- ceived hair-splitting of the lymphomaniacs in their attempts to categorise non-Hodgkin's lymphomas.2 The confusion over the classification of low grade non-Hodgkin's lymphoma is well illustrated in the report on the leukemic phase of mantle zone (inter- mediate) lymphoma by Pombo de Oliveira et al.3 The category of intermediate cell lymphoma was first proposed by Berard et al.4 It was noted that the category contained some lymphocytes resembling those of well differenti- ated lymphocytic tumours and some atypical cells more closely resembling the poorly differentiated lymphocytes of nodular or follicular lymphoma.5 It is now widely accepted that lymphocytic lymphoma of intermediate differentiation (INT) and mantle zone lymphoma (MZL) are the same tumour, perhaps at different stages of evolution,6 and Jaffe et al have suggested that MZL should be the preferred term.7

The category of centrocytoc lymphoma was introduced into the Kiel classification to describe a diffuse lymphoma of small, slightly irregular lymphoid cells that were originally thought to be of follicle centre derivation (hence the name centrocye).8 Subsequent studies have shown that centrocye lymphoma is a distinct entity, clinically, morphologically, and phenotypically distinct from follicle centre cell lymphoma.9 It is unfortunate and confusing that the term centrocye lymphoma9 is still applied to this tumour even though it has been shown not to be of centrocye origin.

Many authors have suggested that INT/ MZL is the same entity as centrocye lymphoma. In a review of INT/MZL, Jaffe et al concluded that this tumour, “clinically, morphologically, and immunophenotypically appears virtually identical to centrocye lymphoma of the Kiel classification.”10 Pombo de Oliveira et al suggested slightly from that position and conclude that, “centrocye lymphoma and INT are clearly closely related morphologically and pheno- typically but may not be entirely inter- changeable pathological entities.”11 Both authors further state that, “perhaps the application of molecular, genetic, or cytogenetic markers may allow such cases to be appropriately classified in future”. In fact, cytogenetic studies suggest a closer relationship between INT and lymphocytic lymphoma.12 It is my opinion that the recognition of INT based on the finding of a mixture of cells with round and cleaved nuclei is flawed. It has been recognised for years that quite apart from biological variability, fixation and processing artefacts can induce considerable variation within a single neoplasm. Lymphocytic lymphomas and centrocye lymphomas have been cell by cell, and screened histologically. It can, however, be difficult to separate these neoplasms on the morphology of the lymphoid cells alone, particularly in suboptimally fixed tissues. There are, however, other many other features that assist in the differentiation of these neoplasms, perhaps the most reliable being the identification of parainmunoblasts which are always present in variable numbers in lymphocytic lymphomas and never found in centrocye lymphomas. On the basis of this criterion, figs 7 and 8 of the paper by Pombo de Oliveira et al1 show a centrocye lymphoma and figs 10 and 11 a lymphocytic lymphoma. Clearly, the locus of INT will not be resolved until a more reliable means of identification, other than variability of nuclear shape, is used for cases to select for study.

MA BARRADAS D MIKHAILIDIS5

Department of Chemical Pathology and Human Metabolism, Free Hospital and School of Medicine, Pond Street, London NW3 2QG


