malignant mesenchymomas but urge caution and application of strict diagnostic criteria in order to avoid a "diagnostic wastebasket". This area is therefore obviously controversial and our tumour could equally be regarded as a malignant mesenchymoma as it would appear to meet the strict definition of that entity. Enzinger and Weiss suggest classifying such a tumour as malignant mesenchymoma, stating the predominant tissue elements which in our case would be "malignant mesenchymoma (combined myxoid and pleomorphic liposarcoma with focal rhabdomyosarcomatous differentiation)". In this paper we have preferred to classify the tumour according to the predominant component present as the rhabdomyosarcomatous elements were only present focally. This approach has also been taken by others.4

The clinical significance of heterologous elements is unknown but myxoid or pleomorphic liposarcoma alone or in combination each have potential for metastasis (as occurred in our case) as well as local recurrence. It has been tentatively suggested that the presence of heterologous elements in liposarcoma in itself probably does not alter the prognosis.1

Immunohistochemical confirmation of rhabdomyosarcomatous differentiation in previously described examples of liposarcoma has been limited and electron microscopy has only been performed in one previous case.7 Myoglobin reactivity was found in the three cases tested and myosin in one case tested. No other skeletal muscle markers had been reported previously. Myoglobin was demonstrated immunohistochemically in all cases showing rhabdomyosarcomatous differentiation and classified as malignant mesenchymoma.11 The immunohistochemical and ultrastructural evidence which we have presented confirms the presence of rhabdomyoblasts in our case beyond all doubt. This phenomenon might be more frequent in liposarcoma than is generally realised.

We are grateful to Dr I Seddon of the Royal Oldham Hospital for allowing us to describe this case.

Pseudopyropoikilocytosis: a striking artefact

Figure 1 Red cell and white cell scatter plots and red cell and platelet histograms produced by a Technicon H.2 automated blood cell counter. In the red cell scatter plot (RBC) red cell size (V) is plotted against red cell haemoglobin concentration (HC). In the PEROX white cell scatter plot forward light scatter (largely determined by cell size) is plotted against light absorbance (largely determined by peroxidase activity). The instrument printout from the patient (a) is shown in comparison with a normal printout (b).

centration 33.1 g/dl; red blood cell distribution width index 20.5 (normal range 11.6–13.9); haemoglobin distribution width 4.24 (normal range 1.82–2.64); and platelet count 1442 × 10^9/l. The instrument histograms and scatter plots (fig 1) showed increased numbers of microcytes, hypochromic cells and hyperchromic cells. The histogram of platelet size distribution was very abnormal and the peroxidase channel showed a broad abnormal band in the area where platelet aggregates would be expected to fall. A blood film (fig 2) showed severe poikilocytosis with bizarre poikilocytes including budding erythrocytes, microspherocytes and irregular fragments. Intact normally staining platelets formed large aggregates together with weakly basophilic particles which resembled degranulated platelets but may have been precipitated plasma proteins. Many of the neutrophils appeared to be necrotic. The large numbers of very small red cell fragments was considered to be the explanation of the apparent thrombocytosis. The red cell morphology was reminiscent of hereditary pyropoikilocytosis or severe burns but as the blood specimen was known to be a routine antenatal sample, an artefact created by careless transport was suspected. The clinical staff were contacted and confirmed that the specimen had been taken by a midwife on a house call and had been left on the dashboard of a motor vehicle for several hours during transport to the hospital. A repeat blood count and film the next day did not show any unusual features.

Heating blood above 49°C leads to spherocyte formation and budding of microspherocytes from the cell surface. The red cells of patients with hereditary pyropoikilocytosis show similar changes at a lower temperature. Severe thermal burns cause such changes to occur in vivo. We have now observed four examples over about 10 years of similar changes caused by overheating of blood samples in motor vehicles. Even on an English spring day blood samples inside a car can become sufficiently hot for this to occur, particularly if the samples are in direct sunlight. The abnormalities induced cause alarm in the laboratory and tend to lead to urgent recall of the patient if their true nature is not recognised.

Pseudopyropoikilocytosis: a striking artefact.

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