a suitable microscope would involve appreciable outlay, though a rheumatological unit will usually have adequate facilities for microscopy. The substrate and reagents can be purchased in kit form from the makers of the ferrography analyser (Foxboro Analytical, Milton Keynes, Bucks) at a cost for one examination comparable to that of a chest x-ray.

Discussion

The most obvious source of “wear particles” in human pathology must be the joints. Ferrography enables isolation of synovial fluid particles and an example of one believed to be cartilage is shown in Fig. 2. Particulate material was described by Ropes and Bauer in 1953,6 and since then the proposal that large numbers of fibrils in joint fluid signify extensive cartilage destruction7 has remained unchallenged. Lessons from metallurgy suggest that size and shape of particles may be at least as important as numbers. The use of joint replacements has increased the need to assess wear in human joints, both in terms of prosthesis lifespan and their effects on remaining human tissues. We would also suggest that ferrography may be used to prepare microscopic preparations of particulate material in bile or urine from patients who form calculi, and sputum in patients exposed to occupational hazards.

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Letters to the Editor

Pulmonary haematoxyphil bodies

One of the interesting observations in the article “Effects of oxygen on the lungs after blast injury and burns”1 was that of giant nuclear masses which the authors regard as being derived from endothelial cell nuclei similar to those reported by Brown et al2 in experimental defibrination. I have found similar large nuclear masses in human lungs and in an experimental model of smoke inhalation in which acrolein was inhaled by rabbits3 (Fig. 1).

By electron microscopy these nuclear masses are found to have a rim of residual cytoplasm containing granules of the same type as those found in platelets (Fig. 2) which identifies them as megakaryocyte nuclei. Where many of these are present it is usually also possible to identify recognisable megakaryocytes even at light level.4 Brown et al3 could distinguish giant endothelial nuclei from megakaryo-

![Fig. 1 Numerous lobulated haematoxyphil masses in the lung of a rabbit which had inhaled acrolein (arrows). Haematoxylin and eosin, original magnification x 300.](http://jcp.bmj.com/ on November 7, 2017 - Published by group.bmj.com)
cytes but the lobulated mass shown in Fig. 4 of Hasleton et al. looks very like the megakaryocyte nuclei which I have seen and which are also mentioned by Corrin as occurring in shock lung.

I have also observed a further type of haematoxyphil body derived from nuclei in the lungs of some babies dying in the neonatal period with respiratory distress syndrome. In this case the Feulgen-positive rounded masses are in the alveoli rather than in the capillaries or alveolar wall (Fig. 3). They appear to develop from the aggregation of the pneumocyte nuclei and are often seen adjacent to hyaline membranes.

It would thus appear that pulmonary haematoxyphil giant nuclear masses are of at least three different derivations.

**Figure 2** Electron micrograph of a haematoxyphil mass from the same animal as Fig. 1 showing platelet-like granules (arrows) in the residual rim of cytoplasm, original magnification ×7500.

**Figure 3** Intra-alveolar haematoxylinophilic rounded nuclear mass in lung from baby dying with respiratory distress syndrome (arrow). Haematoxylin and eosin, original magnification ×300.

**References**


Evaluation of buffy coat microscopy for the early diagnosis of bacteraemia-meningococcal septicaemia

The extensive study of Dr Coppen and colleagues shows that the general use of this technique for the rapid diagnosis of bacteraemia “has little practical value because of false-positives and false-negatives.” There may, however, be one exception to this disappointing finding—namely, in meningococcal septicaemia.

My interest in examining buffy coat preparations from patients with suspected meningococcal septicaemia was primarily to look for damaged endothelial cells and giant nuclear masses (possibly derived from them), as evidence of endothelial damage associated with disseminated intravascular coagulation but, during searches for these structures in Romanowsky-stained preparations, characteristically shaped diplococci (confirmed in other smears as Gram-negative) were found in neutrophil polymorphonuclear leucocytes in four of five culture-proven cases.

These patients were all acutely ill children with haemorrhagic rashes, some extensive, with necrosis, others petechial. In no other cases of purpura (thrombocytopenic or anaphylactic) have any organisms, far less Neisseria, been seen.

The speed with which patients with meningococcal septicaemia deteriorate makes it imperative that any diagnostic test, to be useful, must be rapid. Even in patients without clinical or microscopic evidence of meningitis, microscopy of Gram-stained smears of scrapings from skin lesions may be successful, but, when the purpuric rash is only finely petechial,
Pulmonary haemotoxyphil bodies.

J Crow

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