TWO CASES OF HAEMATOLOGICAL INTEREST

BY

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(WITH SPECIAL PLATE)

The following two cases are considered uncommon enough to merit publication. The first is that of a healthy adult in whose blood macropolycytes have been observed over a period of four years. The second is a case of agranulocytosis in which marked erythrophagocytosis occurred in the circulation following blood transfusion. Photomicrographs of the blood in each case are reproduced (Plate: Case I, Figs. 1-3 and 5-8; Case II, Figs. 9-15).

Case I: Macropolycytes in Health

The macropolycyte is a rare type of blood cell described in detail by Cooke (1927). It is a giant polymorphonuclear leucocyte more than 16μ in diameter and occasionally exceeding 25μ . There is usually hypersegmentation of the nucleus, and nine or more lobes may be present. Osgood and Ashworth (1937) suggest for this type of cell the name of "polylobocyte." Macropolycytes are found most commonly in pernicious anaemia, but they also occur occasionally in various infections, generally where there is a profound bone-marrow reaction, when they are associated with the presence of myelocytes in the circulation. I have observed them in such varied conditions as tuberculosis, erysipelas, osteomyelitis, malaria, phlebotomus fever, ankylostomiasis, and leprosy. Cooke (1934) reports their occurrence in health.

The present case first came under observation in Iraq in 1935 while investigating the changes in the leucocyte state which occur in health in that country. The individual was one of five healthy airmen, in the blood of all of whom macropolycytes were found (Kennedy and MacKay, 1936). A year later, in Iraq, examination of his blood showed that giant cells were still present. At that time it was suggested that these cells, associated as they were with myelocytes in the blood and with a pronounced deviation of the Arneth count, were an indication of marrow stress due to climatic conditions.

This airman, who is a medical orderly, returned to the United Kingdom in 1937. His blood was again examined, and macropolycytes were found to persist. Repeated examinations during the past year have shown an average of one giant cell in every 500 leucocytes, although the other blood findings—including the haemoglobin, red cell and reticulocyte counts, sedimentation rate, total and differential white cell counts, and the Arneth count—were normal. Clinical investigation has revealed no abnormality, and, apart from phlebotomus fever in 1937, he has had no illness of any kind for more than ten years.

Little is known of the significance or the mode of development of the macropolycyte, but its distinctive morphology lends it interest. It may be that it is a normal polymorph which has developed beyond its usual span, or that it is the result of abnormal stimuli acting on the bone marrow. In any case it would appear that its development may result from two quite different sets of factors. For it is found on the one hand in pernicious anaemia, a leucopenic disease with a shift to the right in the Arneth count, and on the other hand in infective conditions where there is a leucocytosis and a shift to the left in the Arneth count. The suggested explanation that it is a leucocyte which, locked in a tissue space, has grown excessively before being released into the circulation would appear to be as reasonable as any.

Giant cells of the eosinophilic and basophilic series have been described, but they are very rare, and hypersegmentation of the nucleus is seldom found. In the present case two giant eosinophils, one of which is shown in Fig. 3, were seen. For comparison a giant basophil from a case of leprosy is also included (Fig. 4).

Case II: Erythrophagocytosis following Blood Transfusion

The patient was a man aged 44. His history is as follows. He had measles in infancy. During the war he served for three and a half years in the Mediterranean area, where he had dysentery (type unknown) in Gallipoli in 1916, and soon after in Salonika benign tertian malaria with, he stated, twenty to thirty bouts of fever. In May, 1937, he developed a septic leg; in July of the same year he had an influenza-like attack; in October he had a septic hand, and in November pleurisy.

On December 22, 1937, he was admitted to hospital with acute anaemia and ulceration of the mouth. His treatment included pentose nucleotide and blood transfusion. He died eight days after admission to hospital.

The following organisms were found in the ulcers of the mouth and throat: Vincent's spirochaetes and fusiform bacilli, *Oidium albicans*, non-haemolytic streptococci, and coliform bacilli. On admission the blood picture was: erythrocytes, 1,510,000 per c.mm.; haemoglobin, 35 per cent.; colour index, 1.2; leucocytes, 800 per c.mm., of which 80 per cent. were lymphocytes, 18.75 per cent. monocytes, and 1.25 per cent. neutrophils. There was considerable irregularity in the shape and size of the red cells, and normoblasts were present.

The day after admission to hospital the patient had a blood transfusion. This produced only transient benefit, and the next day the red cell count was 1,490,000 per c.mm. and the haemoglobin 30 per cent. The leucocyte count, however, had risen to 2,000 per c.mm. and the white cell picture was considerably altered. Only 1.5 per cent. were neutrophils, the increase in the total count being apparently due to an influx of reticulo-endothelial cells. Typical lymphocytes and monocytes were present and numerous macrophage cells, which varied greatly in size, staining reaction, and configuration of the nucleus. The gradation from the lymphocyte on the one hand and the monocyte on the other to adventitious macrophage was such that it made the differential count very difficult. Small round cells were present, resembling lymphocytes but with deeply basophilic cytoplasm, others were of the plasma cell type. Of the large cells two examples are shown in Fig. 9. One has finely granular cytoplasm like that of the typical monocyte of the blood but the nucleus is divided, while the cytoplasm of the other is intensely basophilic.

A striking feature was the ingestion of erythrocytes by the macrophage cells which was in progress. Of the total leucocytes 9.5 per cent. had ingested one or more red cells. Some examples of this are shown in Figs. 10 to 15. Fig. 10 shows the initial stage of the process, and in Figs. 11, 12, and 13 are cells containing one, two, and four erythrocytes respectively. In Fig. 14 is a macrophage packed with red cells; Fig. 15 shows the end-result where the contained red cells are broken down into globular particles and the macrophage has rounded off and is presumably about to pass from the circulation to the spleen.

The probable diagnosis in this case is that of primary agranulocytic anaemia of the aplastic type; but this diagnosis is unconfirmed, since, unfortunately, permission for post-mortem examination was not obtained. In the treatment of agranulocytic anaemia blood transfusions have been reported as more detrimental than beneficial (Jackson, Parker, and Taylor, 1932). In the present instance the immediate reasons for the failure of blood transfusion are readily understandable. Since the administration of pentose nucleotide produced no leucocytic response none could be expected from the injection of

foreign white cells; and the red cells of the transfused blood were rapidly phagocytosed. There was no other evidence of incompatibility between the patient's blood, which was Group A, and the donor's, which was Group O. There was no reaction following the transfusion and no haemoglobinuria. The death of the patient seven days later was easily accountable by his general condition.

The phenomenon of auto-erythrophagocytosis—ingestion by macrophages of the patient's own red cells—has been observed *in vitro* and has been described as occurring in the circulation in malaria and in trypanosomiasis by Connal (1912) and Macfie and Johnston (1913). In the literature consulted no reference was found to erythrophagocytosis either in agranulocytosis or as a result of blood transfusion. In this case the condition probably indicates either a degree of incompatibility between patient's and donor's blood insufficient to produce other reaction or, more likely, increased sensitivity of the patient to foreign red blood cells.

For the data and material described in Case II I am indebted to Air Commodore H. E. Whittingham.

REFERENCES

- Connal, A. (1912). *J. Path. Bact.*, **16**, 502.
 Cooke, W. E. (1927). *British Medical Journal*, **1**, 12.
 — (1934). *J. Lab. clin. Med.*, **19**, 453.
 Jackson, H., Parker, F., and Taylor F. H. L. (1932). *Amer. J. med. Sci.*, **184**, 297.
 Kennedy, W. P., and MacKay, I. (1936). *J. Physiol.*, **87**, 336.
 Macfie, J. W. S., and Johnston, J. E. L. (1913). *J. Lond. Sch. trop. Med.*, **2**, 212.
 Osgood, E. E., and Ashworth, C. M. (1937). *Atlas of Haematology*, Stacey, San Francisco.

THE ANATOMY OF THE BRONCHIAL TREE*

BY

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Recent advances in radiology, bronchoscopy, and thoracic surgery have called for a description of the anatomy of the bronchi that will allow students and clinicians to visualize readily their distribution to the lung tissue. Current anatomical textbooks do not furnish the clinician or thoracic surgeon with the means for the precise localization of pulmonary lesions. The technically perfect film and the brilliant bronchoscopic field are of little avail if there is uncertainty in the conception of the anatomy of the bronchial tree.

The Work of Ewart and Others

Ewart (1889), in his classical work on the bronchi and pulmonary vessels, feared that excessive detail might cause the student to recoil from the study. His book contains a meticulous description of the bronchi down to the finest peripheral branches. He made continued reference to the work of Aeby (1880), who approached the subject from the point of view of comparative anatomy.

A comparison of the diagram of the bronchial tree of Aeby with that of Ewart shows that the former allows one to visualize the bronchial tree at a glance, while the latter with its multiplicity of smaller branches calls for an intensive study that confuses any who attempt to get a practical conception of the fundamentals. Ewart's description of the foliage and finer twigs tends to obscure the picture of the trunk and main branches, the recognition of which is all-important for a reasonably rapid conception of the general architecture of the lungs. Kramer and Glass (1932), apparently unaware of Ewart's work, used a terminology which allows one to picture readily the location of the segments involved by lung abscess. Nelson (1934), whose untimely death must be deemed a catastrophe to thoracic surgery, published an article dealing mainly with bronchiectasis, in which his lucid exposition of the anatomical data showed that he was conversant with the work of Ewart.

It is a tribute to Ewart and Aeby that, with crude appliances and lack of radiology and modern pathological technique, they arrived at a description which can hardly be improved upon apart from some detail. Ewart was quite definite as to the nature of the broncho-pulmonary segment as later described by Kramer and Glass. The latter state that each tertiary bronchus, with the lung tissue it serves, forms a distinct entity, the limits of which can be defined and the relations of which to the chest wall can be delineated. Ewart said that "within each lung large groups of lobules are kept in practical isolation from each other as regards their air supply. Each of these sublobar groups may be considered as forming separate respiratory districts." The recognition of this fact must cause all clinicians and radiologists to think and speak in terms of segments in the localization of lesions such as infections which apparently in the first instance affect a single broncho-pulmonary segment. Only in gross pathology do they pass over the border into adjacent segments and lobes (Figs. 1 and 2). Ewart certainly places students under a debt of gratitude by applying names to the bronchi that connote their territorial distribution and anatomical relations, thus avoiding the terminology of Aeby with its repetition of similar names qualified only by numerals.

Nomenclature: Some Suggestions

At this point a few observations on the terms used in describing the bronchial tree may be useful. The terms primary, secondary, and tertiary bronchi have been given different interpretations by different writers. It seems to us important to recognize as definite entities those branches which supply separate pulmonary segments that can be sharply defined by inflation and the relations of which to the chest wall can be delineated. By some these have been termed tertiary bronchi. We would suggest that they be termed segmental bronchi. This would avoid all confusion in regard to nomenclature.

The main bronchus extends from the bifurcation of the trachea down to its final subdivision into the axillary basal and posterior basal segmental branches. There appears to be no reason, either anatomical or clinical, for subdividing the main bronchus into one or more parts. We would therefore suggest that it be referred to as the main bronchus. Anatomical and clinical localization is readily afforded by the bifurcation and by the openings of the various vestibules and branches. As the segmental bronchi to the upper and middle lobes on the right side and the upper lobe on the left side (which is common to the upper and middle) do not arise directly from the main bronchus it is necessary to have a name for this intervening part, and we suggest that these areas be

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FIG. 1

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FIG. 6

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FIG. 8

FIG. 9

FIG. 10

FIG. 11

FIG. 12

FIG. 13

FIG. 14

FIG. 15

Macropolycytes in Health.—FIG. 1: Two normal polymorphs. FIG. 2: Typical macropolycyte; nine lobes; diam. 18μ . FIG. 3: Eosinophilic macropolycyte; diam. 18μ . FIG. 4: Basophilic macropolycyte (macromast cell) with two neutrophils; from a case of leprosy. FIG. 5: Macropolycyte; diam. 21μ . FIG. 6: Macropolycyte; diam. 21μ . FIG. 7: Macropolycyte; diam. 19μ . FIG. 8: Macropolycyte; $23 \times 16\mu$. The cells shown in Figs. 1–3 and 5–8 are from Case I.

Erythrophagocytosis following Blood Transfusion.—FIG. 9: Two large macrophage cells. FIG. 10: A red cell in the process of ingestion. FIGS. 11–14: Macrophages with ingested red cells. FIG. 15: The ingested red cells are broken down to globular particles and the macrophage has rounded off.

All stained Giemsa; magnification, 1,000 diameters (approx.).