contributed to the low level, but in only three of this group was there any suspicion of ulcer, and in only one was this confirmed radiologically. Nine patients were severely debilitated, and the low haemoglobin in the other six may have been related to disease activity, though a silent ulcer or gastro-intestinal haemorrhage cannot be excluded.

Conclusion

A study of 244 patients with rheumatoid arthritis followed for about six years has been made with particular reference to their intake of salicylates, haemoglobin levels, and the incidence of dyspepsia or ulcer. The great majority were on regular doses of aspirin.

Over the six-year period the group showed a significant increase in haemoglobin level. A smaller group not on regular aspirin showed a comparable increase.

Only three patients (1.2%) were known to have developed clinical or radiological evidence of peptic ulcer while on aspirin. Seven other patients had or were suspected of having an ulcer before they developed rheumatoid arthritis.

The incidence of peptic ulcer in this series compares favourably with that in the general population.

It is concluded that the great majority of patients with rheumatoid arthritis can tolerate regular aspirin without an increase in anaemia. The dangers of causing peptic ulceration or precipitating gastro-intestinal haemorrhage appear to have been greatly exaggerated.

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The National Marriage Guidance Council has recently issued a new publication, *Starting Your Family* (price 2s. 6d.). It includes advice on planning, on financial, medical, routine, and personal topics, and on infertility. There is an appendix on contraceptive methods, and a list of recommended books. Revised editions of *All About Your Wedding* and *The L.S.D. of Marriage*—both priced at 2s. 6d.—are also available. All three booklets may be obtained (3s. each post free) from the Book Room, 78, Duke Street, London, W.1.

FULMINATING AND FATAL PNEUMONIA AND SEPTICAEMIA DUE TO ACHROMOBACTER ANITRATUS

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Achromobacter anitratus was first described by Schaub and Hauber in 1948. Since then several authors have isolated this organism from patients with various illnesses, but without convincing proof of its pathogenicity. The following report gives evidence that the organism may be highly pathogenic.

Case History

A man aged 50 had had chronic bronchitis for ten years. In 1956 he had lobar pneumonia and recovered slowly but satisfactorily. He remained well until October 24, 1957, when he felt abnormally tired but noticed nothing else. At 2 a.m. on October 25 he was awakened by a rigor; he felt progressively feverish and unwell and began to cough up pink froth. He complained of pleuritic pain over the anterior and upper part of the left side of the chest. His condition gradually deteriorated, and when seen by his family doctor at 1 p.m. the same day he was cyanosed and orthopnoeic; his temperature was 100° F. (37.8° C.) and the pulse very weak.

He was admitted to hospital at 5 o'clock the same evening and was obviously very ill, distressed, and confused, with central cyanosis and marked tachypnoea. The pulse volume was poor, the rate 100, and the blood-pressure 110 mm. Hg systolic and 70 mm. diastolic. The percussion note was impaired over the left upper lobe anteriorly and there was bronchial breathing with medium crepitations in the same area. Medium crepitations were also heard over both lower lobes. The white cell count was 13,200/c.mm. (77% polymorphonuclear neutrophils, 3% eosinophils, 5% monocytes, and 15% lymphocytes), and the haemoglobin 98% (14 g./100 ml.). A diagnosis of fulminating lobar pneumonia was made. The probable pathogenic organism was thought to be *Staphylococcus aureus*, which had often been found in similar cases at this time during the epidemic of Asian influenza. Penicillin 250,000 units and streptomycin 0.25 g. were given intravenously at once, and 3 million units of penicillin with 0.5 g. of streptomycin intramuscularly and repeated every two to three hours.

An hour after admission the patient's condition had further deteriorated, and both the cyanosis and the dyspnoea had greatly increased. The systolic blood-pressure had fallen to 90 mm. Hg, the diastolic being unrecordable. An intravenous drip of glucose-saline was set up, and by this means 5 mg. of noradrenaline was rapidly given. Hydrocortisone 100 mg. was also given intravenously, and erythromycin 1 g. by mouth. The blood-pressure returned to 110/70 mm. Hg for a few minutes, but it quickly sank again to unrecordable levels although a total of 20 mg. of noradrenaline was given during the next six hours. His condition continued to deteriorate rapidly; he became comatose, and at 2.15 o'clock the next morning he died, 24 hours after the initial symptoms.

Bacteriology

Smears from a specimen of sputum examined on admission to hospital showed the presence of numerous Gram-negative diplococci morphologically indistinguishable from *Neisseria*. Cultures of the sputum on horse-blood agar, incubated for 24 hours at 37° C., gave a heavy growth of shiny, smooth, convex, circular colonies with an entire edge. The diplococci were encapsulated and non-motile. The biochemical reactions are given in the accompanying Table. The organism was finally identified at the Central Public Health Laboratory, Colindale, as a strain of *A. anitratus* (Schaub and Hauber) Brisou.

Biochemical Reactions of the Present Strain Compared with Those of *A. anitratus* (Schaub and Hauber) Brisou

Organism	Motility	Lactose	Glucose	Sucrose	Xylose	Mannite	Indole	Methyl Red	Voges-Proskauer	Citrate	Nitrates	Urease	Milk Plus B.C.P.
Present strain	-	-	A	-	A	-	-	-	-	+	-	+	A clot 12 days
<i>A. anitratus</i>	-	-	A	-	-	-	-	+	-	+	-	+	A clot 5-10 days

The organisms were resistant to 1 unit of penicillin per ml. of 2% blood agar, 10 µg. of chloramphenicol per ml., 1 µg. of streptomycin per ml., and 1 µg. oxytetracycline per ml., but sensitive to 10 mg. of sulphathiazole per 100 ml.

Gram-negative kidney-bean-shaped diplococci were seen in smears from bronchial pus obtained at necropsy but attempts to isolate the organism on culture failed owing to a heavy growth of *Escherichia coli*.

Post-mortem Examination (10 Hours After Death)

The body was that of well-nourished middle-aged male. Emphysematous bullae lay on the external surfaces of both lungs. Small amounts of fibrin were scattered over the pleural surface covering the left lung. Much of the left lower lobe and part of the left upper lobe were consolidated, red-grey, and airless. There were no deposits of fibrin upon the pleura covering the right lung. The right upper, middle, and lower lobes were severely congested, but consolidation was not complete.

Microscopically, the consolidation of the left lung was found to be due to a confluent haemorrhagic bronchopneumonia. In the most severely affected parts of the left lung the alveolar walls were damaged or distorted, and the capillaries distended with red cells, many of which had escaped into the alveolar spaces. Within the alveoli in this part of the lung were large numbers of polymorphonuclear leucocytes. Many small Gram-negative diplococci lay both inside the polymorphonuclear leucocytes and free within the alveoli; no other organisms were seen. The Gram-negative diplococci were most frequent in those parts of the lungs which were consolidated; a few were present in the left upper lobe and in the right lung.

The liver was pale and fatty; there was centrilobular congestion. The spleen was small and firm, with a well-preserved purple-red cut surface. Within many splenic leucocytes were Gram-negative diplococci. Both kidneys were the site of severe congenital cystic disease. Small scattered haemorrhages were present in the cortex of both adrenals, and among the disrupted cortical cells were many Gram-negative diplococci. The bone-marrow showed normal active haemopoiesis. The remaining organs showed no significant abnormality.

The cause of death was therefore considered to be confluent left haemorrhagic bronchopneumonia due to infection with Gram-negative diplococci, and accompanied by septicaemia with small adrenal cortical haemorrhages.

Discussion

The case reported is one of bronchopneumonia with septicaemia which proved fatal within 24 hours. The predominant organism isolated in culture from the sputum was *A. anitratus* (Schaub and Hauber) Brisou. Organisms with identical morphology were also seen in tracheal pus and intracellularly in histological sections from the lung, spleen, and adrenals. It seems, therefore, that the fatal infection in this patient was due to *A. anitratus*. Schaub and Hauber (1948) showed that this bacterium could be pathogenic for mice and guinea-pigs. In humans, such proof has been lacking. *A. anitratus* has been isolated in patients with urinary and with pulmonary infections, but its significance in these has not been clear (Schaub and Hauber, 1948). It has been found in the blood of patients suffering from fever and diarrhoea (Stuart *et al.*, 1949). Waage (1953) isolated it in a case of cerebral abscess but could not determine whether it was a pathogen or a saprophyte. It has also been found in patients with conjunctivitis (Moor, 1949). In none of these papers was there convincing evidence that *A. anitratus* had behaved as a pathogen. The case we have described above shows that *A. anitratus* may cause a severe and fatal illness with septicaemia and adrenal apoplexy.

It is obscure why this organism has not been clearly recognized before in cases of pneumonia. The fulminating onset in our patient was similar to that of certain cases of influenza so often met at the time, and an actual infection with the influenza virus may have favoured invasion by *A. anitratus*. No virus studies were done in our patient, but the widespread massive invasion by *A. anitratus* that we have demonstrated shows that this organism was ultimately responsible for the pneumonic, septicaemic, and adrenal manifestations. Chronic bronchitis and severe polycystic disease of the kidneys may also have been predisposing factors. *A. anitratus* has been described as particularly sensitive to the tetracyclines and less sensitive to streptomycin and sulphathiazole (Brooke, 1951; Lund, 1954). The organism we have described was, however, sensitive only to sulphathiazole.

Summary

A case is described of pneumonia and septicaemia, fatal within 24 hours. *Achromobacter anitratus* (Schaub and Hauber) Brisou was isolated from the sputum, and a morphologically identical diplococcus was seen in sections of the lung, spleen, and adrenals. Proof that this organism may behave as a pathogen has not previously been established.

We are indebted to the Central Public Health Laboratory, Colindale, for identification of the strain.

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Western Australian Medical School has started a scheme under which fifth-year students will accompany general practitioners on their rounds in Perth to learn more about disorders encountered in family practice but uncommon in hospitals. Thirty students will train under the scheme during the next year.