Both drugs increased heart rate in cats, heart rate and cardiac contractile force in dogs, and heart rate and forearm blood flow in man.

Both drugs acted by stimulating adrenergic β -receptors, as their effects were antagonized by propranolol.

Isoprenaline was found to be about 40 times more active than orciprenaline in cats, 10 to 20 times more active in dogs, and 10 to 40 times more active in humans.

These observations suggest that orciprenaline may be of value in patients in whom side-effects have occurred from blockade of adrenergic β -receptors.

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Medical Memoranda

Initial Cardiac Tamponade in Acute Leukaemia

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Invasion of the pericardium may occur in acute leukaemia (Hayhoe, 1960), and occasionally primary leukaemic pericarditis (Parx, 1961) and haemorrhagic pericarditis (Kosteasph, Xenakes, and Loubros, 1961) have been described. A review of the literature, however, shows that pericardial involvement is extremely rare. To the best of our knowledge cardiac tamponade as an early major manifestation of acute leukaemia has not been described. Recently we saw this phenomenon, and details of the case are given below.

CASE REPORT

A man aged 26 was admitted to hospital with a 10-day history of fever, pain in chest, cough, and dyspnoea. On examination he was acutely orthopnoeic, B.P. 60/45 mm. Hg; pulse 134/min., slightly irregular; and there was pulsus paradoxus. Jugular venous pressure 12 cm. The liver was enlarged two fingerbreadths and tender. The apical impulse could not be felt and the area of cardiac dullness was increased. The heart sounds were feeble; no murmur or friction rub could be heard. Diffuse rales were heard in both lung fields. There were non-tender enlarged lymph nodes and the spleen was just palpable and without tenderness. Laboratory investigations showed haemoglobin 11 g./100 ml. (Haldane), W.B.C.

11,300/cu. mm. (lymphoblasts 35%, lymphocytes 55%, segmented neutrophils 10%), and platelets 90,000/cu. mm.

The diagnosis of acute leukaemia was confirmed by bone-marrow examination. X-ray examination of the chest revealed massive pericardial effusion. The E.C.G. showed sinus tachycardia with low-voltage S-T depression and flattening of T waves in all the leads. An emergency pericardiocentesis was performed and 120 ml. of haemorrhagic pericardial fluid was aspirated. Smear examination of aspirated fluid showed plenty of red blood cells and blast cells. He was treated with corticosteroids and mercaptopurine, and pericardiocentesis had to be repeated on three occasions. He had complete symptomatic and haematological remission and was sent home after 45 days.

He returned three months later with a relapse of acute leukaemia. This time he had haematuria, malaena, extensive leukaemic skin infiltrations, progressive anaemia, retinal haemorrhage, and purpura. There were, however, no cardiac signs or symptoms. He stayed in hospital for 60 days and was discharged after remission had been obtained. At the time of writing he was in remission on maintenance doses of corticosteroids.

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