determine the onset of papillary necrosis. The chronic inflammatory reaction, which is apparent in the interstitial tissue at two weeks, is even then extending beyond the site of the original lesion and may indeed constitute the real basis of the renal damage incident to the abuse of analgesics.

Some of these hypotheses are being tested in further experiments.

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Medical Memoranda

Abscess Formation in an Acute Cardiac Infarct

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Abscess formation in the myocardium is a rare condition. Saphir (1941, 1942) found 32 (0.6%) cases among 5,626 necropsies. In every case it was associated with generalized pyaemia.

Myocardial abscess in an area of acute infarction is even less common. Cossio and Berconsky (1933) were the first to describe such a case in a patient with pneumococcal pneumonia and meningitis. Tedeschi *et al.* (1950) described two cases in association with staphylococcal pulmonary infection. Miller and Edwards (1951) recorded an *Escherichia coli* abscess secondary to chronic pyelonephritis. Tennant and Parks (1959) described a *Clostridium perfringens* abscess derived from an infected gall bladder. Katz (1964) was unable to determine the site of origin in his two cases of infarction, while Horeau *et al.* (1966) did not specify any source of infection in their two cases. Korns (1966) reported a case of *C. perfringens* infection in an infarct, derived from the small bowel.

In the following case the patient developed a myocardial abscess superimposed on a fresh cardiac infarction.

CASE REPORT

A 50-year-old man was admitted to the coronary care unit on 6 February 1968 with acute cardiac infarction. Serial E.C.G. changes showed an acute diaphragmatic surface lesion confirmed by serial enzyme changes. Progress was fair until an episode of ventricular tachycardia occurred on the third day. This responded to intravenous lignocaine (Xylocaine 2%) therapy. On this day he also complained of pleuritic pain in the left infracostal area and dull pain in the left shoulder. No obvious cause for the pain was found, and he responded to symptomatic measures. From the fourth to the eighth day he had an intermittent pyrexia, which was attributed to heart muscle necrosis. Antibiotics were not given. During this time he had been on continuous intravenous cannulation and had developed a local thrombophlebitis. On the ninth day he developed congestive cardiac failure and cardiogenic shock. He died four hours later despite attempts at resuscitation. His monitor showed asystole.

Post-mortem examination of the heart showed an extensive fibrinopurulent pericarditis. There was a multiloculated abscess about 2 cm. in diameter located within an area of recent infarction involving the diaphragmatic and septal walls of the left ventricle. There was severe atheroma of the coronary vessels, and the right coronary artery was occluded by fresh organized thrombus. Other features were an enlarged thyroid and a pin-head (1-mm.) subcapsular abscess in the upper pole of the left kidney. Severe pulmonary congestion was present. Death was due to " pump " failure caused by cardiac infarction, with secondary abscess formation. *Staphylococcus aureus*, coagulase-positive, was grown in abundance from the abscess. No culture or histological examination was performed on the area of cannulation.

COMMENT

Abscess formation is rare in an area of cardiac infarction. In all there are 10 reported cases in the literature. In at least six of these it was associated with pyaemia. In our patient with a large myocardial and a pin-head renal abscess no source of infection was found. Routine cannulation (Plextrocan) after his admission to the coronary care unit caused slight tenderness of a cephalic vein, with some swelling and redness, but this common finding was not regarded as significant.

Systemic bacteraemia has been reported with long-term intravenous therapy. Smits and Freedman (1967) reported 10 cases of septicaemia, five due to *Staph. aureus*, in patients on prolonged venous catheterization. Hassal and Rountree (1959) also reported a case of staphylococcal septicaemia secondary to cannulation, and it is likely that this was the source of the infection in our case. Another possibility was infected parenteral fluids, but culture of specimens of lignocaine derived from the same batch showed no contamination.

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