carditis is difficult. Ideally, active infection should be eradicated first; the tissues hold sutures better and the hazard of infecting the valve replacement is decreased. Uncontrollable heart failure may require urgent surgery before completion of a full antibiotic course; however, early mortality rates approach 30%.[4]

We wish to draw attention to the possible risk of I.U.D. insertion causing endocarditis, which in our case resulted in the death of a young, previously asymptomatic patient. Endocarditis still has a mortality of some 30%.[5] We therefore suggest that I.U.D. insertion in patients at risk from endocarditis should be covered by antibiotics. Because of the Gram-negative organisms in the vagina a broad-spectrum antibiotic such as ampicillin should be used.

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Hyper-transaminasemia with Heparin Therapy

Many side effects seen with heparin therapy have been related to the coagulation system. Biological effects on intercellular enzymes,[1] bone electrolytes, antidiuretic hormone, and aldosterone,[2] however, have been noted. Recently we have noted a rise of serum transaminase levels beginning during heparin treatment. Since transaminase determinations are important in the differential diagnosis of myocardial infarction, liver disease, and pulmonary emboli, rises that might be caused by drugs are very important.

Patients, Methods, and Results

Transaminase levels were recorded before, during, and after heparin therapy in 14 inpatients. All patients received 10 000 units of heparin intravenously every six hours for a period of 10 to 21 days. Blood samples for the enzyme determination were taken from 60 to 90 minutes after the morning injection of intravenous heparin. Serum levels of GOT and GPT were estimated by the SMA 12/60 method, (normal levels with this method are 20 to 40 units for GOT and 20 to 45 units for GPT.) In 10 out of 14 patients the serum transaminase levels rose during heparin treatment (see table). These were definitely abnormal after heparin therapy started with the GPT being higher than the GOT and both falling to normal when treatment ended. The possiblity that heparin interfered with the SMA 12/60 determination of serum transaminases was excluded by performing the determination on paired blood samples from normal controls, to one of which heparin was added after blood was withdrawn. No difference was noted between these two samples.

Discussion

Serum transaminases rose in 10 out of 14 patients receiving intravenous heparin for their underlying disease. This rise was unrelated to dose or to the duration of therapy and in no patients were other diseases discovered that could explain this. It is not clear why this happens. Vavgornik[3] noted the rise in other serum enzymes, such as aldolase, sorbitol dehydrogenase, and leucine aminopeptidase in patients undergoing chronic hemodialysis with heparinization. No change in the GOT and GPT levels before and after dialysis were noted, but the levels during treatment were not tested.

The clinical importance of the rise in serum enzymes during heparin therapy is obvious. Heparin therapy is frequently given for thromboembolic phenomena, and the diagnosis of pulmonary infarction, hepatic damage, and myocardial infarction in these patients is important. The determination of transaminase levels is an established aid in the differential diagnosis of these conditions. The cause of this enzyme rise is not evident, and further studies must be carried out to determine this.

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Calcium Polystyrene Sulphonate: An Unusual Cause of Inhalation Pneumonia

Calcium and sodium polystyrene sulphonate are two potassium absorbing resins administered orally or by retention enema for treatment of hyperkalaemia. Histologically sodium polystyrene sulphonate has been seen on the surface of sections of gastric mucosa.[1] We report here an unusual case of pneumonia associated with the inhalation of the calcium resin.

Case Report

An elderly man was admitted to hospital with a brief history of severe chest pain and shortness of breath. Clinical and electrocardiographical examination showed left ventricular failure, pericarditis, and an acute myocardial infarction. The next day heart block occurred, and the patient underwent cardiac catheterization for maintenance by a pacemaker. Persistent hypotension was treated with intravenous isoprenaline. Oliguria failed to respond to large doses of frusemide and subsequent hyperkalaemia was treated with oral calcium polystyrene sulphonate (Calcium Resonium). Three days after admission the patient had a cardiac arrest and died.

Diagnosis and Maximum Enzyme Level

Age (Years)	Diagnosis	Maximal GOT Level	Maximal GPT Level	Day of Maximum Rise	Duration of Rise (Days)
29	Deep Thrombophlebitis	65	40	5	10
53	Cerebral Emboli	75	110	9	13
24	Deep Thrombophlebitis	70	155	9	13
32	Deep Thrombophlebitis	95	235	7	15
24	Deep Thrombophlebitis	70	120	9	9
33	Deep Thrombophlebitis and Pulmonary Emboli	90	120	12	21
50	Deep Thrombophlebitis	60	60	3	7
46	Pulmonary Emboli	70	135	7	15
33	Deep Thrombophlebitis	85	170	15	17
69	Pulmonary Emboli	70	100	3	13