pleural effusion, such as rheumatoid arthritis, ADA activity may be high; nevertheless, further experience is needed.

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Acute Hepatotoxic Effect of Disopyramide

To the Editor:

Disopyramide is a quinidine-like antiarrhythmic drug. Because of its strong anticholinergic action, gastrointestinal disturbances are the most frequent side-effects. We report a case of hepatocellular toxicity following oral disopyramide treatment.

A 46-year-old woman with atrial fibrillation (AF) was admitted to our department for cardioversion. From the age of 16 years she suffered from mitral stenosis due to rheumatic heart disease. At the age of 43 years, because of paroxysmal AF, DC cardioversion was performed and quinidine was given to maintain the achieved sinus rhythm. After two months, however, AF reappeared and digoxin therapy was started. Because of two episodes of peripheral thromboembolism, coumadin was instituted and open mitral commissurotomy performed. Six months after the operation, while still on anticoagulant therapy, it was decided to attempt a repeat cardioversion, hoping that the better hemodynamic state could maintain sinus rhythm.

On admission, AF was noted on ECG. On physical examination, the first sound was strong and a diastolic murmur, grade 2-3/6, at mitral area was heard. The echocardiogram was compatible with mitral residual stenosis and the left atrium diameter was 4.5 cm. Routine laboratory findings were normal. The DC cardioversion was done, after oral quinidine 0.2 gr x 6 was instituted the day before. Sinus rhythm was achieved, but AF reappeared after one hour. The following day a second trial of cardioversion was attempted by oral disopyramide, at a schedule dose of 300 mg every six hours on the first day. One hour after the second dose, sinus rhythm appeared on the monitor, but the patient began to complain of strong pains in the right hypocondrium, radiating to the back with nausea and vomiting; physical examination revealed a moderately enlarged and sensitive liver, but there was no sign of cardiac failure. Disopyramide was stopped and six hours after, the laboratory findings revealed: serum aspartate transaminase (SGOT) 127 IU (normal range 0-22 IU), serum alanine transaminase (SGPT) 127 IU (normal range 0-25 IU), lactic dehydrogenase (LDH) 67 IU (normal range 170-330 IU), prothrombin time (PT) activity 32 percent of normal. The peak of normal results of liver function tests was reached after three days:

SGOT 443 IU, SGPT 770 IU, LDH 1992 IU; PT activity was still 30 percent of normal five days after the cessation of coumadin therapy.

Two days after disopyramide therapy was stopped, pain and hepatomegaly had disappeared, but the biochemical abnormalities returned to normal ranges after two weeks. Renal function tests, blood cell count, serum total lipids, cholesterol, bilirubin, alkaline phosphatase, creatinine kinase, glucose and electrolytes had always been in the normal range. Test for hepatitis antigen was negative. Disopyramide challenge test and liver needle biopsy were not performed, because they were not regarded by us as ethically and medically justifiable.

The strictly temporal relation between the onset of clinical symptoms and laboratory abnormalities after disopyramide administration and their return to normal after the drug was discontinued, strongly suggest that the hepatotoxicity was due to disopyramide. Quinidine, too, could be a cause of the liver dysfunction, but our patient had taken it in the past without any sign of intolerance.

Intrahepatic cholestasis has been previously described as a side-effect of disopyramide, but to the best of our knowledge, acute toxic reaction on hepatic cells has not been reported. The drug must therefore be considered a potential hepatotoxic agent.

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Erratum

To the Editor:

Regarding our paper entitled "Aristotle's triventricular heart and the relevant early history of the cardiovascular system," that was published in Chest in October, 1983, pages 462-468, several eagle-eyed friends pointed out two very unfortunate printing errors.

In the abstract at the beginning of the paper, two lines from the bottom, in the left hand column, the text presently reads: " . . . by Rufus and Ephesus . . . ." This should be: by Rufus of Ephesus . . . .

The more important error, however, occurs on page 466, in the right hand column in the middle paragraph. The text presently reads: "The connecting passages from the left atrium to the lungs that also are obscure (small) must be the bronchial arteries or the patent ductus arteriosus (or both)." This sentence obviously does not make sense. Going back to the original manuscript, I have discovered that it should be as follows:

"The connecting passages from the left atrium to the lungs that also are obscure (small) are obviously the pulmonary veins. The connecting passages from the left ventricle to the lungs, that also are obscure (small), must be the bronchial arteries, or the patent ductus arteriosus (or both)." Thus, 19 words were inadvertently omitted, which is why this part of the discussion makes no sense.

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