sion which exists over the echocardiographic versus the anatomic presence of aortic-mitral continuity. The patient whose echocardiogram is shown in figure 5 had TAPVC. By angiography he clearly had aortic-mitral continuity (i.e., there was no subaoic conus). Neither the fact that the echocardiographic recording displays a break in the line between the posterior aortic wall and the anterior leaflet of the mitral valve on this particular sweep, nor the fact that the closed position of the mitral leaflets is slightly posterior to the posterior aortic wall means that there is aortic-mitral discontinuity. These findings are highly dependent upon the location of the transducer on the chest wall and, unfortunately, add a great deal of subjectivity to the interpretation of echocardiograms.

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Interaction of Sulfisoxazole and Warfarin

To the Editor:

Sulfonamides have been described as having the potential to enhance the response to coumarin anticoagulants. The primary mechanism for this possible interaction is displacement of coumarin from plasma protein binding sites. Potential for interaction has generally been attributed to long-acting sulfonamides because of their greater degree of protein binding. We recently observed a patient who experienced increased warfarin activity after completing a 14 day course of the short acting sulfonamide sulfisoxazole.

A 74-year-old man was admitted to the City of Memphis Hospital in February 1975 with hemoptysis, hematuria, and gingival bleeding. The patient had a previous diagnosis of organic heart disease, pulmonary embolism, and peptic ulcer disease. For several months the patient had been maintained on digitalis, diuretics, antacids, and warfarin (10 mg daily). Seventeen days prior to admission, a 14 day course of sulfisoxazole 500 mg every 6 hours was begun for a urinary tract infection. The prothrombin time (PT) at the beginning of sulfisoxazole therapy was 20 seconds; nine days later the PT was 28 seconds. On the last day of sulfisoxazole therapy, the patient noted hemoptysis, bleeding from the gums at the site of an old tooth fracture, and hematuria. Two days later, the patient was admitted to the hospital with the above complaints and a PT of 60 seconds. No other medication had been taken by the patient and liver function was within normal limits. Stool guaiac was negative. Phytonadione 15 mg was administered intravenously, and a normal PT was attained the following day.

To the best of our knowledge, there are no previously reported cases suggestive of the enhancement of the activity of warfarin by sulfisoxazole. We certainly can only speculate that sulfisoxazole was responsible for this episode, because rechallenge was not feasible. However, no other etiology of the problem could be determined.

It was somewhat puzzling that the potentiation of warfarin did not become clinically significant until approximately 14 days after initiation of sulfisoxazole therapy. However, Hobbs et al. reported a case of warfarin potentiation caused by protein displacement by oxypHENylbutazone that took 18 days to become clinically significant. Also, Eisen described a patient whose PT was 57 seconds 11 days after starting a 7 day course of phenylbutazone while on maintenance warfarin.

We report this case with the attitude that it is only a possible example of an interaction of warfarin and sulfisoxazole.

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Letter: Interaction of sulfisoxazole and warfarin.
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