Table 1. Acute Colitis Associated with Methyldopa.

Patient No.	Age/Sex	Daily Dose	Duration	Symptoms	Confirmatory Tests	Concomitant Drugs	Results of Rechallenge	Clinician's Diagnosis
		mg	days					
1	Unknown	1000	4	Bloody diarrhea	Proctoscopy, x-ray films	Triamterene-hydrochlorothiazide, allopurinol, probenicid	Positive after 3 days of methyldopa	Colitis
2	53/M	500	9	Gastrointestinal bleeding	Proctoscopy, x-ray films	Reserpine-hydralazine-hydrochlorothiazide	Not performed	Colitis
3	50/M	1000	1½	Bloody diarrhea	Proctoscopy	None	Not performed	Acute ulcerative colitis
4	58/F	750	90	Diarrhea	Sigmoidoscopy, biopsy	Doxepin, propranolol, conjugated estrogens	Not performed	Ulcerative colitis
5	65/F	500	42	Various	Biopsy	Hydrochlorothiazide	Positive after 12 days of methyldopa	Ulcerative proctitis
6	60/M	750	60	Bloody diarrhea	Biopsy	None	Positive after 4 days of methyldopa	Ulcerative colitis

comitantly with methyldopa could be interpreted as indicating an additional causal factor; however, the isolated rechallenges with methyldopa implicate this drug as the sole causally related agent. In addition, in Patient 3, the medication taken after methyldopa was a diuretic, presumably a thiazide, which was not associated with symptoms of colitis.

The recent *Journal* article on a proposed mechanism of action in methyldopa-induced autoimmune hemolytic anemia[3] persuaded us to look for cases of autoimmune hemolytic anemia in patients with ulcerative colitis. Indeed, although the combination is rare, a number of cases have been reported and were recently reviewed with additional case presentations by Altman et al.[4] Thus, it is possible that the mechanism of methyldopa-induced colitis may be similar to that proposed for hemolytic anemia in the article by Kirtland et al.

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PERICARDIAL MESOTHELIOMA AFTER EXPOSURE TO ASBESTOS

To the Editor: In her article on malignant mesothelioma in the July 24 issue,[1] Antman states that "No association between pericardial mesothelioma and asbestos exposure has been reported." However, Churg and Warnock have reported a case of malignant mesothelioma of the pericardium arising after direct application of asbestos and fiberglass to the pericardium.[2] The tumor was diagnosed 15 years after the epicardial surface had been dusted with 300 mg of asbestos in an attempt to improve cardiac circulation in a patient with angina pectoris. Another report, which appeared after Antman's review, described a primary pericardial mesothelioma that occurred in a 60-year-old man 30 years after he had been exposed to asbestos at a shipyard during World War II.[3] In both cases, amphibolic asbestos fibers were identified in tissues by means of electron diffraction and energy-dispersive x-ray analytic techniques. Pericardial mesothelioma may rarely occur as a complication of exposure to asbestos many years before the patient's clinical presentation.

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GLYCOSYLATED HEMOGLOBIN WITH INSULINOMA

To the Editor: In their letter in the issue of December 11 Freedman et al. suggest that in the evaluation of fasting hypoglycemia, determination of hemoglobin A_1 (HbA_1) is a more practical procedure than serial glucose estimations during a prolonged fast.[1] They base their conclusion on the case of a patient with an insulinoma whose low preoperative HbA_1 value returned to normal four weeks after resection of the tumor.

We have measured glycosylated hemoglobin preoperatively in 13 patients with surgically confirmed insulinoma by means of a chromatographic technique using prepoured columns (Isolab, Akron, Ohio). None of the patients was taking diazoxide. HbA_1 levels ranged from 5.2 to 6.6 per cent (mean, 5.9±0.5 per cent). None of the 13 patients' values were below the range seen in 30 nondiabetic subjects (4.5 to 8.9 per cent; mean, 6.5±1.2).

The use of HbA_1 in the evaluation of hypoglycemia has not been systematically addressed in the medical literature, although conflicting comments were made during discussion of a series of presentations on glycosylated hemoglobin.[2] Gabbay stated that hemoglobin A_{1c} levels were not below normal in a population with hypoglycemia but did not give specific data, whereas Sueldner noted that two patients with insulinoma had hemoglobin A_{1c} levels below normal.

Although we have recently demonstrated that [illegible] our mean plasma glucose levels were significantly lower than normal in 13 other patients with insulinoma under conditions simulating normal activity (78±3 *vs.* 97±4 mg per deciliter, $P<0.01$),[3] the degree of deviation from normal is much greater in patients with poorly controlled diabetes.[4] It becomes readily apparent, therefore, why HbA_1 levels may be quite elevated in a patient with uncontrolled diabetes and yet not be consistently below the normal range in a patient with an insulinoma.

We conclude that glycosylated hemoglobin levels are not consistently low in patients with insulinoma and that the HbA_1 test is not reliable in screening for this disorder. In reaching a diagnosis of in-