teaching models, as well as on patients. I agree with Faber that combining the experience of several specialty services will provide a broad range of experience for residents, but I suggest that anesthesia be included in the list of specialties that require complete endoscopic training. Such experience not only will immensely improve the intraoperative care of the patient but will also enable the anesthesiologist to use his acquired expertise in respiratory care, a field in which so many anesthesiologists are involved and have responsibility.

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REFERENCES

Impairment of Atrioventricular Conduction by Methyldopa

To the Editor:

The account of Williams et al1 of impairment of atrioventricular conduction by clonidine prompts us to report a case in which methyldopa produced similar disturbances.

CASE REPORT

A 69-year-old man with blood pressure of 190/110 mm Hg was placed on therapy with methyldopa (250 mg twice daily). In his initial electrocardiogram, there was first-degree atrioventricular block (P-R interval, 0.21 second) and left axis deviation (−60°; Fig 1A). Two days after initiation of therapy with methyldopa, the patient developed atrioventricular block of Mobitz types 1 and 2 (Fig 1B and 1C). Therapy with the drug was discontinued, and atrioventricular conduction returned to the initial state within two days (Fig 1D). Ten days later, the patient was again challenged with administration of the same dosage of methyldopa and again developed Mobitz type-2 block within two days (Fig 1E), which again disappeared within two days after therapy with the drug was discontinued (Fig 1F). Ten months later, the patient developed complete heart block, and permanent pacing was instituted.

DISCUSSION

We believe that both clonidine and methyldopa, by decreasing sympathetic tone, can impair atrioventricular conduction in susceptible individuals.

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Figure 1. Electrocardiographic tracings from 69-year-old man with first-degree atrioventricular block and left axis deviation (A). After two days of therapy with methyldopa, atrioventricular block of Mobitz types 1 and 2 developed (B and C). When therapy was discontinued, initial state returned (D). Challenge with same dosage of methyldopa produced Mobitz type-2 block (E), which again disappeared with cessation of therapy (F).

REFERENCE

Digoxin and the Exercise Electrocardiogram

To the Editor:

Tonkon and associates1 have confirmed our findings2 that the effects of digoxin on the exercise electrocardiogram disappear at near maximal heart rates. This constitutes a new type of "walk-through" phenomenon, and physicians supervising exercise tests should be alert to the possible role of digitalis when "abnormal" ST-segment displacement disappears as exercise continues and heart rate increases.

In our study, which has not been published in detail, the effects of oral therapy with digoxin were evaluated in 11 normal young men during multistage bicycle exercise according to a protocol described elsewhere. Each subject was tested while receiving a daily dose of 0, 0.125, 0.25, and 0.5 mg of digoxin in a double-blind manner. Modified X and Y bipolar leads were used. The response of the S-T segment to exercise was analyzed using two sets of criteria, Bruce's STs criterion (mean ST-segment displacement of 1.0 mm or more at 50 to 69 msec after the nadir of the S wave) and the criteria of Lester et al.4 Therapy with digoxin had no effect on the heart rate or blood pressure at rest or on the maximal achieved heart rate.

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