Nodal bradycardia induced by tocainide

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Summary
A case of tocainide-induced nodal bradycardia in standard recommended dose is reported. There was no recurrence when the drug was subsequently reintroduced in a reduced dosage. It is suggested that in the elderly, tocainide should be used in a lower dosage than normally recommended.

KEY WORDS: tocainide, nodal bradycardia.

Introduction
Tocainide (2 amino-2',6'-propionoxylidine hydrochloride) is a new anti-arrhythmic drug that is chemically similar to lignocaine but, in contrast, is effective in the treatment of ventricular arrhythmias when given orally (Nyquist et al., 1980). Tocainide administered every 8 hours suppressed ventricular ectopic beats by more than 70% in 11 of 15 patients in a placebo-controlled multi-dose study (Winkle et al., 1976). The recommended oral dose has been a minimum of 1200 mg daily in 2 or 3 divided doses. Side effects reported so far have been few, but include lightheadedness, nausea, vomiting, bradycardia, hypotension and left ventricular failure (Bastian et al., 1980). To our knowledge, there has been no report of nodal bradycardia caused by tocainide. We report such a case of severe nodal bradycardia on tocainide in a dose of 1200 mg daily without a recurrence on a reduced dose of 800 mg daily.

Case report
A 79-year-old woman was admitted in January 1982 with polymyalgia rheumatica which was treated with oral corticosteroids. She was also found to have multiple ventricular ectopics mostly in bigeminal rhythm. Her renal function was normal. She was started on oral tocainide in a dose of 400 mg, three times daily, and on the following day her electrocardiogram (ECG) showed complete disappearance of the ventricular ectopics. After 4 days on tocainide, she developed severe epigastric discomfort followed by several bouts of vomiting. A repeat ECG showed severe nodal bradycardia at a rate of about 48 per min with the ventricular ectopics still completely suppressed. Serial ECG's and cardiac enzymes did not suggest any evidence of myocardial infarction. Her blood urea and liver function tests remained satisfactory.

It was therefore assumed that the untoward effects were due to tocainide which was stopped. On the next day of cessation of therapy the ECG came back to normal sinus rhythm, but within a few days the ventricular ectopics reappeared. She was later re-started on tocainide in a dose of 400 mg twice daily which again caused complete suppression of ventricular ectopics, but this time without any untoward effect.

Discussion
Our study confirms the previous reports that tocainide is very effective in suppressing ventricular arrhythmias (Graffner et al., 1980). However, our patient developed epigastric discomfort, severe vomiting and nodal bradycardia on starting tocainide in the recommended oral dose of 400 mg, 3 times daily. These features completely disappeared on cessation of therapy and it can be safely assumed that these were side effects due to tocainide. There was no recurrence of nodal bradycardia or any other untoward symptom on reintroduction of the drug in a reduced dosage. It therefore appears that these side effects are most probably dose related. We suggest that in the elderly, tocainide should be used in a dose of 400 mg twice daily rather than 3 times daily to avoid any untoward side effect. However, further study will be needed to evaluate our proposition.

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References
Clinical reports

A prospective randomised trial of tocainide in patients following myocardial infarction. American Heart Journal, 100, 1017.


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