chest pain. The resting electrocardiogram had always been normal. He smoked 20 cigarettes daily. Physical examination was unremarkable, his blood pressure being 130/80 mm Hg. An exercise electrocardiogram and thallium perfusion scan result were normal. In spite of atenolol (100 mg daily) and verapamil (360 mg daily) his symptoms became increasingly frequent and effort tolerance declined. Coronary angiography, performed in June 1983, showed normal vessels with a dominant right coronary artery; left ventricular function was normal.

In August 1983, while still receiving atenolol and verapamil, he was readmitted after further chest pain and syncope. He showed complete heart block with a ventricular rate of 40/min. After pacing for 24 hours the heart reverted to sinus rhythm; there was no evidence of a myocardial infarction. One month later the electrocardiogram was normal both at rest and on exercise. The results of thallium-201 scintigraphy was normal at peak exercise, five hours after exercise, and during a cold pressor test.

He is currently having no treatment and still has intermittent chest pain, not always related to exertion. His bundle electrocardiography showed a normal H-V interval of 40 milliseconds. Rapid atrial pacing failed to induce either chest pain or Wenckebach phenomenon. Corrected sinus node recovery time was normal. An M mode echocardiogram was normal.

Comment

The cause of this patient's chest pain remains uncertain. He had no evidence of obstructive coronary artery disease or of myocardial infarction. Coronary artery spasm has not been excluded, although abnormalities might have been expected in electrocardiograms taken during pain or the cold pressor test. He has no evidence of cardiomyopathy or myocarditis. The most likely cause of the transient episode of complete heart block was the concurrent administration of atenolol and verapamil. Without such treatment atrioventricular conduction is normal. For ethical reasons we are unwilling to restudy conduction after further administration of these agents.

Despite the theoretical risks of giving two drugs, both with negative chronotropic and inotropic effects, some authors have suggested that this combination of drugs is safe in selected patients—that is, those without conduction disorders or left ventricular dysfunction. The manufacturers have no report of such an occurrence, and combination treatment has been shown to be more effective than either drug used alone. As this case illustrates, however, even these criteria may not prevent the development of an iatrogenic complication.⁴ When using these agents together caution is necessary, and the regimen of Bala Subramanian would be appropriate.²

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Infantile colic and parental smoking

Infantile colic is very common and usually presents as paroxysmal screaming with clenched fists, legs drawn up, and a red face. The belief that crying occurs mainly in the evening was not confirmed in population surveys.¹ The symptoms are thought to result from vigorous gastrointestinal contractions, but almost nothing is known about the cause.^{2,3} Brazelton⁴ claimed that paroxysmal crying that occurs in the early evening is a distinct entity from colic. We describe the relation of evening and postprandial colic to parental smoking.

Subjects, methods, and results

The study group comprised 253 infants examined at the routine paediatric consultation at 3 months of age. Mothers were interviewed and four digestive symptoms recorded—namely, regurgitation, vomiting, flatus, and crying after meals. If the mother answered "yes" to the question on crying she was asked "When your baby cries, does he or she seem to be in pain and is he or she agitated?" Crying for two hours or more in the early evening was also recorded. Other questions concerned parity, birth weight, method of feeding (breast, bottle, mixed), conditions of delivery, and socioeconomic state, education, and smoking habits of the parents. Proportions were compared by χ^2 tests and log linear model analysis used for studying several factors simultaneously.

Half the mothers (127) reported that their infants cried after feeding, 72 occasionally and 55 regularly; 109 seemed to be in pain and were agitated. We classified them into three grades of postprandial colic: grade 0 (not crying), 126 infants; grade 1 (occasional crying or regular crying without pain), 75 infants; grade 2 (regular crying with pain), 52 infants. There were no differences between the sexes. Postprandial and evening colic occurred independently. Postprandial colic was related to other digestive symptoms, but evening crying was not.

Parental smoking was associated with digestive symptoms (table) and particularly with postprandial colic but not with evening colic. In the 215

Parental smoking and digestive symptoms

	No of smoking parents			p Value*
	0	1	2	
No of infants	136	86	31	
No (%) with postprandial colic:				
Grade 0	90 (66)	33 (38)	3 (10)	0.001
Grade 1	37 (27)	23 (26)	9 (29)	
Grade 2	9 (7)	24 (28)	19 (61)	
No (%) with regurgitation after meals	48 (35)	44 (51)	15 (48)	0.06
No (%) with vomiting after meals	19 (14)	17 (20)	7 (23)	NS
No (%) with flatus after meals	67 (49)	52 (60)	22 (71)	0.04
No (%) with evening crying	91 (67)	55 (64)	18 (58)	NS

NS = Not significant.

* Tests for homogeneity.

families in which the mother did not smoke and the father either also did not smoke or smoked 1 to 10, 11 to 20, or more than 20 cigarettes a day the prevalence of postprandial colic (grade 1 or 2) was 32%, 45%, 69%, and 91% respectively ($p < 0.001$). When neither parent smoked (136 infants) the prevalence was 32% and when only the mother smoked (seven infants) it was 57%.

Postprandial colic was less frequent in breast fed infants and those in the higher socioeconomic classes. Parents who smoked were less likely to breast feed and had a lower educational level. Postprandial colic, however, remained strongly related to each of the three factors—parental smoking, infant feeding, and socioeconomic state—after controlling for the two others. Parity, birth weight, and the conditions of delivery were not associated with either postprandial colic or evening crying.

Comment

Postprandial crying with apparent pain and agitation was related to other digestive symptoms and may be regarded as characteristic of infantile colic. The effect of parental smoking was striking. Many adult non-smokers suffer discomfort in an environment polluted by smoke or the odour of stale tobacco, and young babies are probably more susceptible because of their sensitive olfactogustatory systems. It may be hypothesised that gastrointestinal contractions are triggered by olfactory or gustatory stimulation through a vagal reflex mechanism.⁵

Whatever the true cause of infantile colic it may benefit families to know that parental smoking may be a factor.

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