

reduction technique, it did demonstrate the presence of antibody to poliovirus type 1. The subject involved was 12 years old, had been immunized by the three-dose ACIP schedule, and had received a booster seven years previously. Review of this subject's past medical history did not reveal any apparent immunologic disease.

Comment

It has been shown previously that neutralizing antibody to poliovirus declines over time and can be boosted with an additional dose of OPV.⁴ This study, however, demonstrates that adequate immunity was obtained from the present immunization policies for poliomyelitis and persisted at least six to nine years. Another booster at 12 years of age would therefore not offer increased protection at that age.

An additional booster at 12 years of age might, however, prolong the protection that exists at that time. This prolonged protection could eventually reduce the number of adults who because of their own inadequate immunity are at risk for contracting poliomyelitis from immunized infants.⁶ It is not known whether lack of immunity in this adult group results from inadequate immunization or declining antibody titers.⁷ Repeating this study on the same population in subsequent years may provide useful information in this regard.

Answering these questions about the duration of polio immunity is essential if a safe and effective national polio immunization policy is to be established. If polio immunity does indeed decline over time and additional boosters are needed, perhaps on a regular basis, then a switch to IPV might be indicated for the program as a whole. IPV is a safer vaccine than OPV, its only disadvantage being the need for regular boosters.

Acknowledgments

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An Extended Family With Giardiasis

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Asymptomatic infants with giardiasis may be the unsuspected cause of treatment failure in other extended-family members. The family physician must maintain a very high index of suspicion for

this problem. Giardiasis is often asymptomatic,¹ but little doubt remains that the organism can produce symptoms.² Waterborne,³ foodborne,⁴ and person-to-person⁵ transmission of *Giardia lamblia* has been observed. Galazka points out that the family physician must consider the source whenever giardiasis is identified in an individual.⁶ This case report describes individuals whose giardiasis was not cured until members of the extended fam-

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ily from different households were treated. *G lamblia* was recovered from members initially considered unlikely to be infected with the organism.

Case Report

The illustrative extended family (Figure 1) consists of three unmarried sisters, their five children, and another adult occasionally involved in the care of the children. The four adults maintained separate households, but contact among members of the extended family was frequent. They received their water from the municipal waterworks and had no pets. The children were not enrolled in any day-care center. The initial patient was one of the children.

Patient 6, a 3-year-old white boy, presented to the family practice center with a history of poor weight gain over a period of several months, intermittent diarrhea, occasional constipation, anorexia, and abdominal cramps, but no vomiting. The child weighed 25 lb (11.4 kg) and his height was 31½ in (83 cm), both of which were less than the fifth percentile. The remainder of the physical examination was unremarkable. Initial fecal studies revealed only *Entamoeba coli* cysts.

During the workup of this child the mother, patient 3, likewise presented to the clinic with a history of stomachache, bloating, nausea, and occasional bouts of diarrhea over the previous several weeks. She had recently traveled to Texas. She also admitted to significant recent emotional stress. Her physical examination and initial laboratory studies were unremarkable, and she was treated symptomatically with chlordiazepoxide and clidinium (Librax). After two weeks her symptoms were unchanged, and three stool specimens revealed cysts of *E coli* only. On the suspicion that *G lamblia* might also be present, duodenal mucus was sampled with an Entero-Test (HEDECO) capsule. *G lamblia* trophozoites were identified. Patient 6 (treated empirically) and patient 3 were treated with oral metronidazole for seven days in the usual recommended doses for children and adults. The child's diarrhea improved, and he gained weight. The mother's symptoms abated. However, symptoms returned in both patients after approximately three months.

It was noted that others in the extended family

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BRIEF SUMMARY

PROCARDIA* (nifedipine) CAPSULES

For Oral Use

INDICATIONS AND USAGE: I. **Vasospastic Angina:** PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

II. **Chronic Stable Angina (Classical Effort-Associated Angina):** PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk to such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug interactions: Beta-adrenergic blocking agents: (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy: Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

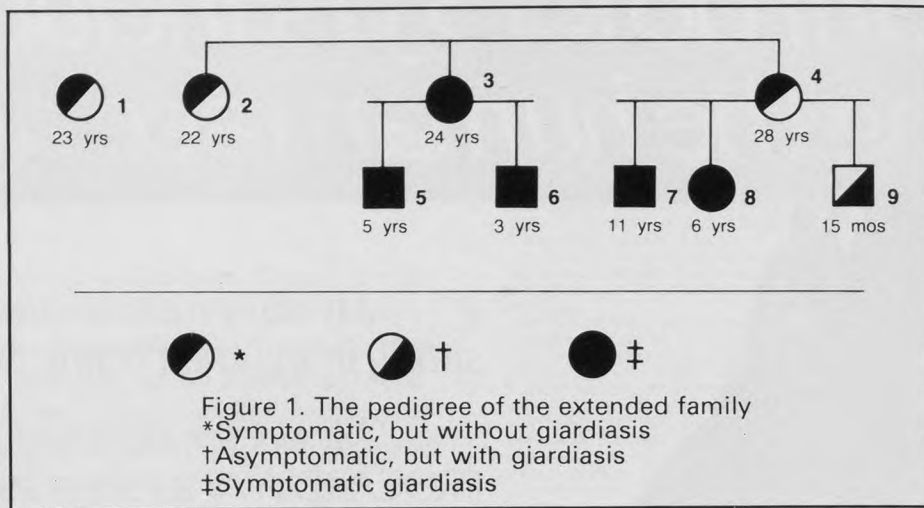
HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

More detailed professional information available on request.

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had gastrointestinal complaints, and because of suspected person-to-person transmission (and re-infection), the entire extended family was studied. At least three stool samples were examined from each of these nine people. Samples were preserved in 10 percent formalin and examined by the Kentucky State Laboratory within 24 hours. If the three specimens were negative, an Entero-Test was performed after informed consent was obtained in accord with the University of Louisville's Human Research Committee. Cysts of *G lamblia*, but no other protozoa, were identified in the stools of patients 6 and 7. In those people for whom stools were negative for cysts, trophozoites were found in the duodenal mucus of patients 5, 8, and 9. The false-negative rate for the stool samples was 75 percent. Those infected were treated with oral metronidazole in the usual recommended doses and remained asymptomatic for over one year.

Comment

When an individual is found to be infected with *G lamblia*, the physician should consider the source, even innocent-appearing asymptomatic infants. It is clear from the literature that the physician should consider community epidemics, other members of the household, sexual contacts, and even animal carriers as possible sources of the infection.⁶⁻⁸ This case illustrates that members of the extended family are another potential source. It is notable that the 15-month-old asymptomatic child with three negative stool samples was found to have trophozoites in his duodenal mucus. A

12-month-old relatively asymptomatic child was implicated as the source of a foodborne community epidemic in 1979.⁴ Thus, the physician should consider infants still in diapers as possible sources even if they are asymptomatic and have had multiple stool samples in which cysts of *G lamblia* could not be identified. The administration of the Entero-Test is not difficult in infants. Examination of duodenal mucus remains as the most exact way of diagnosing the infection⁹ until such time as serologic testing, now limited to research,¹⁰ becomes more widely available.

Acknowledgment

The HEDECO Company, Mountain View, California, provided the Entero-Test capsules, and the Kentucky State Laboratory examined study specimens.

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