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Fetal Well-Being Assessed by Maternal Daily Fetal-Movement Counting

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Normal fetal movements are an indicator of fetal well-being, whereas reduced fetal activity may precede fetal death. In 1960 Bernstine¹ noted that decreased activity may reflect disturbance of placental function and indicate impending demise of the fetus. In 1972 Mathews² noted cases in which fetal death was preceded by a period of markedly reduced fetal activity. In 1973 Reinold³ reported that absence of spontaneous fetal movement places the fetus at high risk. Sadovsky and Yaffe⁴ observed that a definite decrease in fetal activity occurs before fetal death from chronic diseases such as toxemia. They considered the decrease in fetal movements to be an "alarm signal" of impending fetal death. Normal fetal activity has been associated with a good outcome and provides reassurance that delivery can be deferred to gain further fetal maturity.⁵

Women report 80 to 90 percent of the fetal movements that are picked up by electronic devices.⁶⁻⁸ Fetal movements are first felt by women at about the 18th week of pregnancy, increasing to a maximum between 29 and 38 weeks

and decreasing slightly just before term.⁴ Ehrstrom⁶ reported a 12-hour median of 86 fetal movements in the 24th week, 132 in the 32nd week, and 107 in the 40th week. There is a daily rhythm with maximum activity in the evening. Activity is relatively uniform in the morning and afternoon, with approximately seven fetal movements per hour.

An abnormal daily fetal movement count has been a better predictor of impending fetal death than placental lactogen levels or urinary estriol determinations.^{5,8,9} A record of fetal activity by a compliant patient has been shown to be a reliable alternative to antepartum fetal heart-rate testing for initial screening of fetal well-being.¹⁰

Several protocols have been developed and used for maternal counting of fetal movements.^{4,6,8-19} Of these protocols, patients have readily accepted and complied with the Cardiff "count-to-ten" system first developed by Pearson.¹⁶ In this system, patients note the time when ten movements have been felt for that day and report for evaluation if there have been fewer than ten movements in 12 hours. This protocol was demonstrated to be efficacious in the study by Liston et al.¹⁸

Case studies^{4,11} have demonstrated a decrease in fetal movements prior to fetal death from

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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 those 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 for 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 effects 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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chronic causes such as toxemia or hypertension. Studies in which women have been followed prospectively without interventions for decreased fetal movements have demonstrated that the fetus with decreasing fetal activity is at risk.^{5,14} Prospective studies in which interventions were done for decreasing fetal movements have also demonstrated the significance of the daily fetal-movement count.^{6,17-20}

There has been only one large prospective, controlled, randomized trial of formal fetal-movement counting. Neldam¹⁹ studied 2,250 pregnant women in a hospital-based obstetric service. One half were taught to count fetal movements methodically and inform the hospital if they felt decreased fetal activity. Of infants weighing more than 1,500 g, there were eight intrauterine deaths in the control group and no deaths in the maternal monitoring group. Only nine (0.4 percent) in the monitoring group reported a decrease in fetal movements. Five acute cesarean sections and two inductions were performed. One patient later had an elective cesarean section, and one had a spontaneous delivery. The difference in the stillbirth rate in mothers who counted fetal movements and those who were not specifically instructed to do so was statistically significant. Based on the local stillbirth rate, six would have been expected in each group. Neldam²⁰ recently reported on a continuation of his study involving a total of 3,111 women and again noted a statistically significant reduction in the stillbirth rate in the group of women monitoring fetal movements.

Liston et al¹⁸ studied high-risk women who were performing daily fetal-movement counts. If fewer than ten movements were felt in 12 hours, the patient reported for a "non-stress" test. If the non-stress test was nonreactive, an oxytocin challenge test was performed. In seven of the 11 cases of reported diminished activity, there was evidence of fetal compromise. Appropriate interventions were undertaken and there were no deaths in this series.

Conclusions

The following conclusions seem warranted on the basis of current knowledge. A decrease in fetal

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MATERNAL FETAL-MOVEMENT COUNTING

activity may indicate fetal compromise. There is evidence that maternal monitoring of fetal movements can identify fetuses at risk for poor outcomes and that medical interventions can lead to a lowered stillbirth rate. A practical regimen for evaluating a report of decreased fetal movements is performance of a non-stress test, to be followed, if abnormal, by an oxytocin challenge test. The resources necessary to perform these procedures are available to family physicians, but the overall impact of introducing maternal counting of fetal movements into primary care has not been studied.

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