A 55-yr-old male undergoing varicose vein surgery with an extradural block complained of chest pain. E.c.g. changes and subsequent examination of the heart including coronary angiography suggested the diagnosis of Prinzmetal's variant angina. The therapeutic implications of this condition in patients undergoing anaesthesia and surgery are discussed.

Continuous monitoring of the e.c.g. during surgical procedures has become the accepted standard of practice. While early detection of arrhythmia remains the primary purpose of such electronic surveillance, deviation of the ST segment from the isoelectric line may be observed. We report a case in which, it is postulated, coronary vasospasm compromised myocardial perfusion and was responsible for the e.c.g. and haemodynamic changes observed.

CASE REPORT
A 55-yr-old black male was admitted to hospital for stripping of varicose veins. Five years before admission he was noted to be mildly hypertensive; this was well controlled with hydrochlorothiazide 50 mg twice daily. On admission arterial pressure was 150/90 mm Hg. The remainder of the physical examination was normal. The patient gave no personal or family history of ischaemic heart disease. The chest x-ray was normal and the electrocardiogram revealed mild left ventricular hypertrophy based on voltage criteria. Renal function was marginal, with the blood urea nitrogen being 2.9 mmol litre\(^{-1}\) and the creatinine 141 \(\mu\)mol litre\(^{-1}\); serum sodium was 137 mmol litre\(^{-1}\), potassium 4.5 mmol litre\(^{-1}\) and chloride 100 mmol litre\(^{-1}\). The patient was premedicated with pentobarbitone 100 mg and pethidine 50 mg i.m. 1 h before arriving in the operating room.

After giving 5\% dextrose in lactated Ringer's solution 500 ml i.v., a lumbar extradural block was performed at the L3–4 interspace. Twenty-five millilitre of 1.5\% lignocaine with 1 in 200,000 adrenaline was injected to give a block extending to T6. Arterial pressure decreased to 120/70 mm Hg over a 10-min period, and then stabilized. Twenty minutes after the commencement of surgery the patient suddenly complained of retrosternal chest pain. The arterial pressure was 90/60 mm Hg and the electrocardiogram showed elevation of the ST segment, widening of the QRS complex and occasional ventricular premature contractions (fig. 1). Nitroglycerin was given sublingually with immediate relief of pain and return of the ST segment to the baseline (fig. 2). The remainder of the procedure was uneventful. After operation serial electrocardiograms and cardiac enzyme measurements were within normal limits. The patient had no further anginal attacks during his hospital stay.
Following discharge he was referred to the cardiology clinic. A stress electrocardiogram revealed short runs of ventricular tachycardia without deviation of the ST segment.

On cardiac catheterization, left ventricular contraction and haemodynamic function were normal. A coronary angiogram (fig. 3) showed mild narrowing of the left anterior descending and circumflex arteries and a 45% stenosis of the first diagonal branch of the left anterior descending coronary artery; the minimum diameter was 1.2 mm as determined by quantitative angiography (Brown et al., 1977). The same angiographic views were repeated after administration of propranolol 0.2 mg kg⁻¹ i.v. (fig. 4) over a 15-min period, followed by three 0.3-mg doses of adrenaline (fig. 5) subcutaneously. The stenosis in the diagonal branch constricted to 0.5 mm (representing a five-fold increase in flow resistance). The circumflex and left anterior descending arteries were virtually unaffected. Nitroglycerin 0.6 mg, given sublingually, dilated the constricted segment to 1.4 mm (fig. 6). The patient did not experience any chest pain during the procedure, despite the severe spasm. This degree of vasoactivity is greater than is usually found in classical angina and a diagnosis of Prinzmetal's or variant angina was made.
DISCUSSION

In 1959 Prinzmetal and others described 32 patients whose anginal symptoms differed markedly from those of classical angina pectoris. Although the location and character of the pain was similar to classical angina pectoris, it occurred when the patient was at rest or engaged in minimal physical activity. In other words, the pain was not precipitated by any of the factors that are usually associated with increased myocardial oxygen demand.

The electrocardiographic changes during an attack of Prinzmetal’s or “variant” angina are diagnostic and consist of ST segment elevation in the lead overlying the ischaemic region associated with ST segment depression in the reciprocal leads. This is in marked contrast to classical angina in which the ST segments are depressed during the attack and reciprocal changes do not occur. The chest pain is promptly relieved by sublingual nitroglycerin and within 1–2 min ST segments return to the baseline. Between the periods of pain the electrocardiogram is normal and the serum transaminase concentrations are unchanged or minimally increased.

The attack of angina in our patient was not preceded by cardiovascular disturbances such as tachycardia, hypertension or severe hypotension. The arterial pressure had stabilized for approximately 20 min following the initial decrease from 150/80 to 120/70 so it is tempting to attribute the chest pain to coronary vasospasm. This in turn would have caused myocardial ischaemia with resulting hypotension and arrhythmia. Despite the associated decrease in arterial pressure, the anaesthetist elected to treat the ST elevation with a coronary vasodilator rather than resorting to the more conventional approach of treating the complication with a vasopressor. This therapy was highly efficacious.

One can only speculate as to the origin of the spasm. It has been shown that spinal or extradural blockade denervates the sympathetic nervous system two to three segments above the level of somatic block. Above the level of sympathetic blockade, compensatory vasoconstriction is seen, presumably in an effort to offset the reduction in peripheral resistance caused by the block. This reflex sympathetic activity would involve the cardiac sympathetic nerves causing coronary vasoconstriction and consequent ischaemia.

Prinzmetal suggested that the pathophysiology of the condition is increased tonus (spasm) of a large coronary vessel which is already partially occluded by atherosclerosis. This has subsequently been demonstrated by coronary angiography performed during the attack (MacAlpin and Kattus, 1967; MacAlpin, 1970; Silverman and Flamm, 1971; Dhurandhar et al., 1972; Oliva, Fotts and Pluss, 1973; Gensini, 1975).

Levin, Wolk and Summers (1974) found that 27% of the patients had normal coronary angiograms. It is now recognized that patients with Prinzmetal’s angina may have totally normal coronary angiograms.

The factors initiating coronary artery spasm have not been fully elucidated. The coronary arteries are supplied with both alpha and beta receptors, the former mediating vasoconstriction and the latter vasodilatation (Zuberbuhler and Bohr, 1965; Bohr, 1967; Macraven et al., 1971). It is known that alpha receptors predominate in the larger coronary vessels and the injection of adrenaline or noradrenaline in the patient who is beta blocked will induce profound vasoconstriction.

Yasue and others (1976) studied four patients with Prinzmetal’s angina. These patients were given propranolol 40 mg and 3 h later adrenaline 0.4–0.5 subcutaneously. With the beta receptors blocked, the unopposed alpha activity of adrenaline was well demonstrated. Within a short time all patients experienced angina. This was associated with an increased arterial pressure and a reduction in heart rate. On a subsequent occasion the regimen was repeated and coronary angiography was performed during the attack. Severe spasm accompanied by ST segment elevation was demonstrated in all four patients. Both the spasm and the pain were rapidly relieved by nitroglycerin.

Is coronary vasoconstriction solely a result of sympathetic stimulation, or does it follow some parasympathetic–sympathetic interplay? Levy (1971)
postulated that increased vagal activity results in stimulation of the preganglionic sympathetic fibres with release of noradrenaline at the post-ganglionic nerve ending.

In an earlier paper Yasue, Touyama and Shimamoto (1974) postulated that the parasympathetic nervous system played a pivotal role in initiating the attacks. They studied 10 patients with Prinzmetal's angina. Three of the patients had anginal symptoms at the time of the investigation, while the remaining seven had been symptom-free for more than 3 months. All 10 patients were given 10 mg of metacholine subcutaneously. In the three with symptoms, an attack of Prinzmetal's angina was precipitated, associated with ST segment elevation, while the symptom-free patients were not affected. When the three patients were given atropine, the subsequent injection of metacholine did not induce an attack. One of the patients was given phenoxybenzamine on the night before receiving a metacholine test and this prevented the attack. Administration of propranolol aggravated the symptoms.

Further evidence supporting the importance of the parasympathetic nervous system comes from studies on patients during rapid eye movement (REM) sleep (Nowlin et al., 1965; Murao et al., 1972). Variant angina is known to be associated with REM sleep. Acetylcholine is known to trigger REM sleep, while atropine has been shown to prevent it.

**Management**

Nitroglycerin is the mainstay of treatment. Its effect, when given either sublingually or i.v., is almost instantaneous. Endo and others (1975) described 35 consecutive patients whose symptoms, e.g., changes and arrhythmia were relieved by nitroglycerin. The value of atropine seems to be preventive rather than therapeutic and it should be used as part of the premedication in a patient with known Prinzmetal's angina.

Propranolol is contraindicated in the management of variant angina. By blocking the beta receptors, unopposed alpha activity of endogenous catecholamines can either precipitate or intensify coronary artery spasm. Calcium antagonists such as verapamil and nifedipine prevent calcium flux, thus relaxing the coronary vessels and preventing their responses to vasoconstrictive influences.

In a study of 12 patients with variant angina given nifedipine in appropriate doses, Goldberg and others (1979) reported that pain disappeared in 11 of the patients. None exhibited hypotension or tachycardia. Nifedipine withdrawal is associated with an aggravation of anginal symptoms and should not be discontinued before operation.

Patients in whom ventricular arrhythmia develops during an attack often fail to respond to conventional anti-arrhythmic drugs such as lignocaine or procainamide; calcium blockers are often effective (Heupler and Proudfoot, 1979).

Since the pathophysiology is primarily one of spasm, it is not surprising that excision of the stenotic lesion is ineffective (Endo et al., 1975), whereas a bypass graft of the spastic segment has been effective in two patients in this institution.

**REFERENCES**


**ANGINE (VARIANTE DE PRINZMETAL) PENDANT UNE ANESTHESIE EXTRADURALE**

**RESUME**

Un homme de 55 ans, subissant une intervention chirurgicale pour des varices après un blocage extradural, s'est plaint de douleurs dans la poitrine. Les variations de l'e.c.g. et l'examen ultérieur du cœur, y compris une angiographie coronaire, ont laissé penser à une angine de la variante de Prinzmetal. Les implications thérapeutiques de cet état sur des malades subissant une anesthésie et une intervention chirurgicale sont débattues dans cet article.

**PRINZMETAL-ANGINA WAHRHEND EXTRADURALER ANÄSTHESIE**

**ZUSAMMENFASSUNG**


**LA VARIANTE PRINZMETAL DE ANGINA, DURANTE ANESTESIA EXTRADURAL**

**SUMARIO**

El examen de un paciente masculino de 55 años de edad en proceso quirúrgico al respecto de varices y con un bloqueo extradural se quejó de dolor en el pecho. Los cambios en el electrocardiograma (e.c.g.) y el subsiguiente examen del corazón, incluyendo la angiografía coronaria, sugirieron el diagnóstico de Prinzmetal, que es una variante de angina. Se discuten, seguidamente, las repercusiones terapéuticas de esta condición en pacientes sometidos a anestesia y a proceso quirúrgico.