exclude a haemolytic cause and the apparent absence of the gallbladder suggests a wide cholecysto-enteric fistula and obstructed cystic duct. Cholelithiasis is being seen with increasing frequency at Baragwanath Hospital, as this case illustrates.

REFERENCES

1. Bennion LJ, Grundy SM. Risk factors for the development of cholelithiasis in man II. *N Engl J Med* 1978; **299:** 1221-1227.
2. Becker BJP, Chatgidakis CB. Carcinoma of gall bladder and cholelithiasis on the Witwatersrand: autopsy survey of racial incidence. *S Afr J Clin Sci* 1952; **3:** 13-22.
3. Gelfand M. The incidence of gall stones in Zimbabwe. *Cent Afr J Med* 1981; **27:** 47-48.
4. Bremner CG. The changing pattern of disease seen at Baragwanath Hospital. *S Afr J Surg* 1971; **9:** 127-131.
5. Day EA, Marks C. Gallstone ileus: review of the literature and presentation of 34 new cases. *Am J Surg* 1975; **129:** 552-555.
6. Kasahara Y, Umemura H, Shiraha S, *et al.* Gallstone ileus: review of 112 patients in the Japanese literature. *Am J Surg* 1980; **140:** 437-440.
7. Rigler LG, Borman CN, Noble JF. Gallstone obstruction: pathogenesis and roentgen manifestations. *JAMA* 1941; **117:** 1753-1759.
8. Balthazar EJ, Schechter LS. Air in the gall bladder: a frequent finding in gallstone ileus. *AJR* 1978; **131:** 219-222.

Coronary artery bypass surgery in a patient with symptomatic ventricular arrhythmia

A case presentation and review of the literature

J. Z. PRZYBOJEWSKI

Summary

A 42-year-old White man suffered from recurrent symptomatic ventricular tachycardia but no angina pectoris. Cardiac catheterization demonstrated a normally contracting left ventricle and coronary angiography delineated significant atherosclerotic obstructions in the left circumflex (LC) coronary artery and the first diagonal branch of the left anterior descending (LAD) coronary artery. Coronary artery bypass graft (CABG) surgery was carried out on the anterolateral and mid-lateral branches of the LC coronary artery as well as the first diagonal branch of the LAD coronary artery. Frequent postoperative Holter monitoring as well as maximum-exercise stress testing has failed to show any recurrence of the ventricular arrhythmia, and the patient has remained asymptomatic and medical therapy has been discontinued. Some 30 months after operation left ventricular cine angiography demonstrated normal contractility. Selective coronary arteriography indicated that the CABG to the anterolateral branch of the LC coronary artery was occluded at its proximal aortic anastomosis. However, the CABGs to the mid-lateral branch of the LC and LAD coronary arteries were still patent. Repeat serial resting ECGs failed to show any evidence of postoperative myocardial infarction. It is concluded that CABG surgery was responsible for eliminating the episodes of life-threatening ventricular tachycardia, presumably by correcting myocardial ischaemia. The role of CABG surgery in the control of medically unresponsive and dangerous ventricular arrhythmias is reviewed.

S Afr Med J 1984; **65:** 573-578.

Cardiac Unit, Department of Internal Medicine, Tygerberg Hospital and University of Stellenbosch, Parowvallei, CP
J. Z. PRZYBOJEWSKI, M.B. CH.B., F.C.P. (S.A.)

Case presentation

The patient was a 42-year-old non-smoking White man in whom hypertension had been diagnosed in 1978. Methyldopa had been prescribed for the hypertension, but soon afterwards he decided to take up marathon running, usually covering a distance of 20 - 30 km 3 times a week, and he felt so healthy that he decided to stop taking the methyldopa. His blood pressure remained normal and he therefore remained off all medication. He remained asymptomatic, apart from occasional effort-induced palpitations, until early May 1980, at which time he felt unusually exhausted after a 42 km run. During that night his wife found him in an unrousable state with stertorous breathing. The general practitioner noted the patient to be cold and sweating with a low blood pressure and a pulse rate of 42/min; he also thought the patient was experiencing pulmonary oedema. Atropine and furosemide were immediately administered and he was admitted to a peripheral hospital.

The patient could not remember anything of the incident and when he came to his senses was aware of a slight and transient epigastric discomfort. A consultant physician found no evidence of congestive cardiac failure. The patient was well perfused with a blood pressure of 140/90 mmHg. His pulse was slow at 42/min, but was regular. There was a short grade 1/6 ejection

systolic murmur at the apex which did not radiate. The remainder of the examination was unremarkable. A resting ECG documented sinus bradycardia of 40/min, few unifocal ventricular extrasystoles, a P-R interval of 0,21 second (borderline first-degree atrioventricular block) and a mean frontal axis of +75°. Slight ST-segment elevation was present in the inferior leads, this feature being far more prominent in leads V1 - V3. In addition, asymmetrical T-wave inversion was present in leads aVL and V4 - V6. The possibility of myocardial ischaemia and even of a possible acute non-transmural myocardial infarction (MI) was therefore considered. An ECG taken the next day showed much the same features, except that asymmetrical T-wave inversion was now only seen in lead V6 and there was a suggestion of left ventricular hypertrophy by voltage criteria. Further daily ECGs showed persistence of these features. Serial 'cardiac enzyme' levels were all normal.

During the first 48 hours of ECG monitoring in the peripheral hospital several episodes of asymptomatic ventricular tachycardia were noted, some of long duration, and the patient was therefore given quinidine sulphate 400 mg 3 times daily. On the third night of his admission the nursing staff noted an episode similar to that described by his wife at home, with ventricular tachycardia, but this quickly reverted spontaneously to sinus rhythm. At no time was either ventricular fibrillation or complete heart block noted. No further ventricular arrhythmias were experienced and the patient was transferred to the Intensive Coronary Care Unit at Tygerberg Hospital on 27 May 1980. The referring physician thought the ECGs were consistent with the 'athletic heart syndrome' and that the patient had 'ischaemic heart disease aggravated by marathon running' and that he was 'at high risk for arrhythmia'. On clinical examination he looked healthy and had a regular pulse of 48/min and a blood pressure of 120/80 mmHg. There were no murmurs and examination of the cardiovascular and other systems was negative. A resting ECG delineated persistent left ventricular hypertrophy by voltage and early ventricular repolarization changes in the anteroseptal leads; a chest radiograph demonstrated a normal cardiac shadow and clear lung fields. M-mode echocardiography failed to show any evidence of hypertrophic cardiomyopathy or mitral valve prolapse. Side-room investigations were negative and the serum biochemical values were normal apart from an elevated serum cholesterol level of 8,04 mmol/l (normal 3,8 - 6,5 mmol/l). The possibility of an acute MI was excluded and the patient was given a submaximal treadmill exercise test. He managed to reach his target heart rate of 162/min and to exercise for a total of 12 minutes. Ventricular bigeminy was recorded 2 minutes into exercise but at no time did he complain of angina or feel faint. No ST-segment deviations were witnessed and the blood pressure response was satisfactory.

At this stage the clinical presentation was far from clear and because of the possibility of an 'angina equivalent' state the patient was subjected to full cardiac catheterization and selective coronary angiography on 29 May 1980. All intracardiac pressures and indices of left ventricular function were normal. Left ventricular cine angiography delineated a normally contracting chamber with no evidence of hypertrophic obstructive cardiomyopathy, mitral valve prolapse or mitral insufficiency. Selective coronary angiography established the presence of significant obstructive atherosclerosis. Left coronary angiography demonstrated significant obstructive lesions in the left circumflex (LC) coronary artery just proximal to a large, graftable anterolateral branch, two proximal lesions of the graftable mid-lateral branch of the LC coronary artery and a significant obstruction of the first graftable diagonal branch just distal to its origin from the left anterior descending (LAD) coronary artery (Fig. 1). The right coronary artery (RCA) was dominant and only minor internal luminal irregularities were visualized. Collateralization was not evident. The patient experienced no angina or arrhythmias during catheterization.

The final diagnosis was therefore that of significant double-vessel obstructive coronary atherosclerosis with good left ventricular function and the possibility of life-threatening ventricular arrhythmias secondary to myocardial ischaemia. In addition, the case was thought to represent an 'angina equivalent'. Oral quinidine gluconate 324 mg 3 times daily and sotalol 80 mg/d was given, together with a small oral dose of isosorbide dinitrate. After much discussion with the cardiac surgeons it was decided to perform saphenous vein aortocoronary artery bypass grafting to the first diagonal branch of the LAD coronary artery as well as the anterolateral and midlateral branches of the LC coronary artery. This operation was carried out successfully on 20 August 1980 using cold cardioplegia.

The patient was followed up at the Cardiac Clinic and remained asymptomatic and normotensive; resting ECGs persistently showed a sinus bradycardia of approximately 40/min with early ventricular repolarization changes most strikingly seen in the anteroseptal leads. Numerous 24-hour Holter

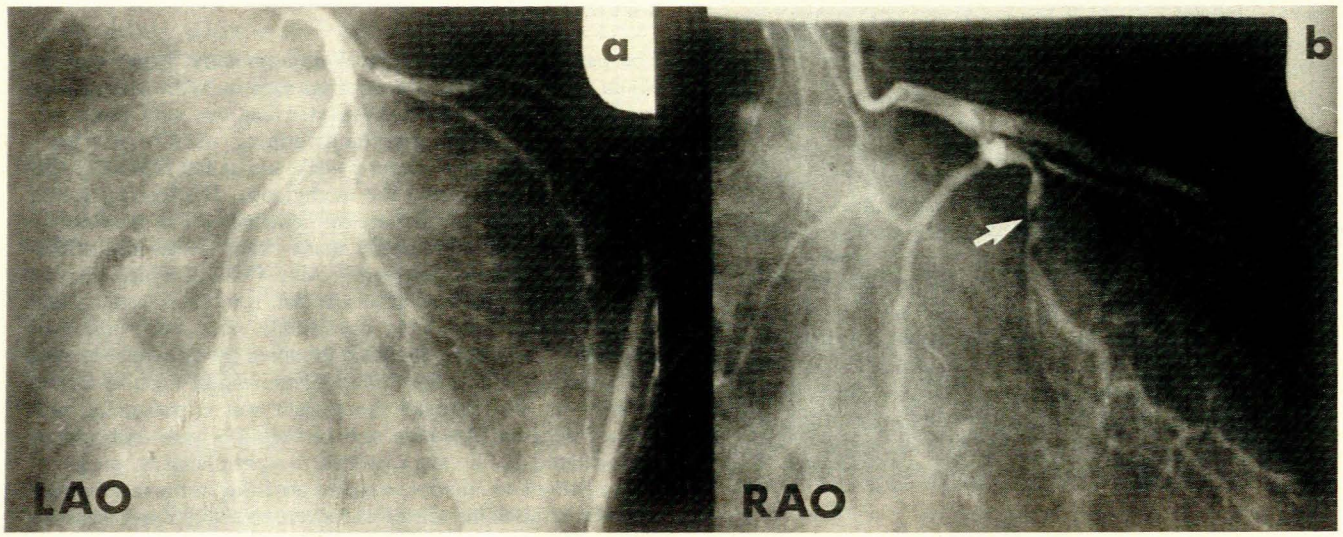

Fig. 1. Left coronary cine angiograms in (a) the left anterior oblique (LAO) view, and (b) the right anterior oblique (RAO) view. Diffuse internal luminal irregularities are present. Significant lesions (arrowed) **are seen in the LC coronary artery as well as the first diagonal branch of the LAD coronary artery.**

monitoring periods failed to show any episodes of ventricular tachycardia or fibrillation, and the only abnormality noted was infrequent unifocal ventricular extrasystoles. The patient has continued to participate in marathon running and has made a habit of telephoning me after such events to state how well he is feeling! A treadmill stress test performed recently was negative.

Approximately $2^1/_2$ years after coronary surgery full cardiac catheterization and selective coronary angiography were undertaken. All the intracardiac pressures and indices of left ventricular function, including a left ventricular cine angiogram, were within normal limits. The RCA still demonstrated insignificant lesions and did not provide any collateral supply to the left coronary circulation. The original atherosclerotic obstructions of the left coronary artery system had become more significant, as would be expected. The grafts to the first diagonal branch of the LAD coronary artery (Fig. 2) and midlateral branch of the LC coronary artery were patent, but the graft to the anterolateral branch of the LC coronary artery was occluded at the proximal aortic anastomosis.

Discussion

The place of coronary artery bypass graft (CABG) surgery[1] using autologous saphenous vein has been firmly established in the symptomatic relief of stable angina pectoris,[2] unstable angina pectoris[3] and the angina produced by left mainstem coronary artery narrowing.[4] Since there is overwhelming evidence supporting its effectiveness in these groups of patients, the latter form the majority of those operated upon for ischaemic heart disease (IHD). Claims are also being made of increased survival after CABG surgery,[5,6] and this is particularly striking in those patients with triple-vessel and left mainstem coronary artery disease.[4,7] A further claim is of reduction in the risk of sudden death, although the exact mechanism by which this is achieved is speculative.[8] The possible role of CABG surgery in preventing acute MI in subsets of patients with stable angina pectoris, unstable angina pectoris, variant (Prinzmetal's) angina pectoris, left mainstem coronary artery stenosis and evolving acute myocardial infarction is even more debatable. Many have strongly supported the concept of improvement in left ventricular function and have advocated the use of CABG surgery (sometimes in association with left ventricular aneurysmectomy[9]) in resistant congestive cardiac failure.[10] The aim of this article, with the help of the illustrative case, is to review the available literature on the controversial role of CABG surgery in the control of dangerous and life-threatening ventricular arrhythmias.

Studies supporting the effectiveness of CABG surgery for ventricular arrhythmias

That improvement in coronary blood flow by surgery might dispel ventricular arrythmias was suggested by Najafi *et al.*[11] These authors reported on a 31-year-old woman who developed severe unstable angina pectoris accompanied by life-threatening ventricular fibrillation some 8 months after triple valve replacement. Repeat surgery identified an obstructive non-atheromatous plaque occluding the origin of the left mainstem coronary artery. This was assessed as being iatrogenic and was corrected by an autogenous saphenous vein patch angioplasty. Immediately after reoperation the patient no longer complained of angina pectoris and no further ventricular arrhythmias could be documented. Nevertheless, some 2 months later she was readmitted on account of 'arrhythmias secondary to hypokalaemia', but without recurrent angina. At this stage aortic cine angiography delineated normal coronary ostia and arteries, attesting to the success of the second operation. In this case, therefore, hypokalaemia could have been incriminated in the pathogenesis of ventricular fibrillation.

Hutchinson *et al.*[12] described the successful treatment of a 47-year-old man suspected of having IHD on account of chest pain and angina/ventricular fibrillation precipitated by a Master two-step exercise test. This patient had total occlusion of the RCA and a most significant obstructive lesion of the left mainstem. Several episodes of ventricular fibrillation were then documented after coronary arteriography and the patient underwent CABG surgery to the left mainstem and diagonal branch of the LAD coronary artery; 'an immediate improvement occurred in the quality and character of the ventricular fibrillation'. This report was soon followed by that of Nakhjavan *et*

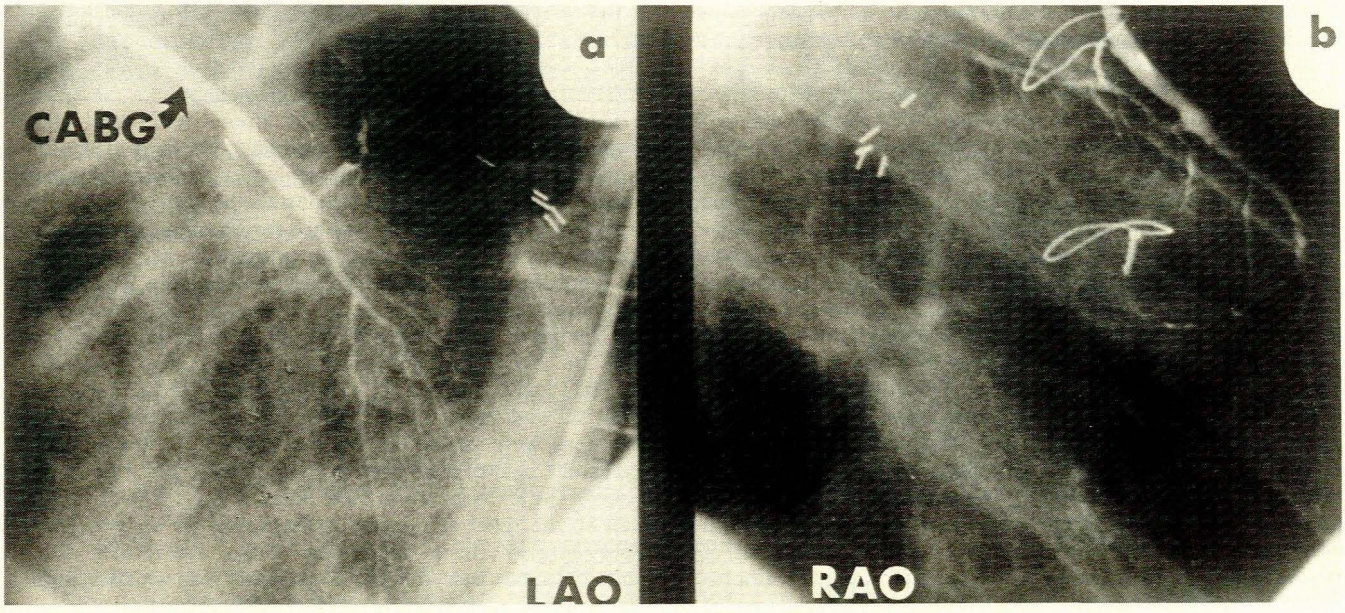

Fig. 2. Cine angiograms of the patent CABG to the first diagonal branch of the LAD coronary artery in (a) the LAO and (b) the RAO projections.

al.;[13] 7 weeks after an acute subendocardial MI a 49-year-old woman experienced repeated episodes of angina accompanied by multiple unifocal ventricular extrasystoles degenerating into ventricular tachycardia and resulting in syncope. Medical therapy with a variety of anti-arrhythmic drugs proved unsuccessful, as did temporary cardiac pacing with overdrive suppression, and the patient needed repeated electrocardioversion. Coronary angiography delineated a most significant left mainstem lesion, an LC coronary artery free of haemodynamically significant obstruction, and a normal RCA. Emergency CABG surgery on the LAD coronary artery abolished the unstable angina and ventricular tachycardia. It is not quite clear why the dominant LC coronary artery was not bypassed, as would be expected with a left mainstem lesion.

These 3 patients had all had unstable angina, significant left mainstem obstruction and dangerous symptomatic ventricular arrhythmias not associated with the acute phase of MI. Ecker and co-workers[14] were the first to document success with CABG surgery in the setting of ischaemic symptomatic ventricular tachycardia and in the absence of angina pectoris and a left mainstem obstruction. Their patient was a 61-year-old man who despite anti-arrhythmic therapy presented with recurrent ventricular tachycardia and syncope 2 months after an acute MI. Coronary angiography revealed significant obstructions of the RCA and the anterolateral branch of the LC coronary artery. Repeated overdrive pacing and electrical cardioversion were therefore employed, but the patient sustained an acute inferior MI followed by numerous symptomatic episodes of ventricular tachycardia. In view of this, emergency CABG surgery was carried out on the RCA and anterolateral branch of the LC coronary artery, and a small diaphragmatic left ventricular aneurysm was plicated but not excised. This abolished the ventricular tachycardia and the patient was discharged on short-term anti-arrhythmic drug therapy. Some 3 months postoperatively cardiac catheterization demonstrated a patent CABG to the RCA, but that to the LC coronary artery was occluded. Importantly, ECG and ventricular cine angiography failed to show any new MI. All medication was discontinued and the patient remained asymptomatic. Holter monitoring only detected infrequent unifocal ventricular extrasystoles. Ecker *et al.*[14] therefore hailed this as 'a new method of treatment of refractory ventricular tachycardia', making the assumption that it was ischaemic in origin.

The use of emergency CABG surgery in patients with medically uncontrollable symptomatic ventricular tachycardia and/or fibrillation as a complication of 'impending infarction' (unstable angina), as well as those presenting with these arrhythmias in isolation, was assessed by Lambert and co-workers.[15] Four patients in the former category and 5 in the latter were encountered. All 9 patients had significant obstructive coronary artery disease, varying numbers of arteries being affected. The results of CABG surgery appeared most satisfactory in that only 1 patient with recurrent ventricular arrhythmia relapsed and required insertion of a permanent pacemaker for adequate control. In the discussion to Lambert *et al.*'s[15] paper, Green (p. 527) comments on 6 of his own patients with recurrent ventricular tachycardia or ventricular bigeminy which resolved completely after CABG surgery.

Graham and co-workers[16] further supported the efficacy of CABG surgery for the abolition of ventricular arrhythmias, and reported on 9 patients — 8 had had either recurrent ventricular tachycardia or fibrillation resistant to intensive medical therapy and 1 had junctional tachycardia. Seven of the patients also had recurrent syncope due to arrhythmia, but only 4 complained of angina pectoris. In addition to obstructive coronary atherosclerosis, 6 of the 9 patients had a left ventricular aneurysm; in each case this was resected and accompanied by a minimum of one CABG. Three patients only underwent a CABG to two or more coronary arteries. Only 1 of the patients died within the first 24 hours after operation, whereas the remaining 8 were entirely free of any ventricular arrhythmias at an average of 13 months later. These authors therefore concluded that 'myocardial revascularization with saphenous vein grafts and/or left ventricular aneurysmectomy can be effective therapy for refractory ventricular tachyarrhythmias'.[16]

In a classic paper Mundth *et al.*[17] reported on a series of 10 patients who developed medically refractory ventricular tachycardia and fibrillation after MI; 7 were operated upon in the 'acute' phase (within 6 weeks of the MI) and the remaining 3 in the 'chronic' phase (at 21 weeks). Six patients in the 'acute' group underwent an 'infarctectomy', 2 undergoing an additional CABG operation; the remaining patient in this group received a CABG only. Four patients in this category were so ill that they required intra-aortic balloon pumping, which tended to decrease the episodes of ventricular arrhythmias. The 3 patients in the 'chronic' category underwent isolated CABG surgery since none of them had a definitive left ventricular aneurysm. Four of the 7 in the 'acute' group survived, 3 being asymptomatic and free from ventricular tachycardia and fibrillation. The 3 patients in the 'chronic' group all survived with abolition of the ventricular arrhythmias, there being only 1 late death at 18 months. In view of these results the authors were of the opinion that 'surgical therapy may be effective in the management of medically refractory ventricular irritability both in the acute postinfarct phase and in chronic coronary artery disease'.[17]

Potentially lethal ventricular arrhythmias occurring during exercise and demonstrated by stress ECG testing are generally accepted as indicating a poor prognosis and an increased risk of sudden death.[18] It is believed that the mechanism is a 'circus movement phenomenon' created by the 'boundary phenomenon' arising between ischaemic and non-ischaemic myocardial tissue.[19] Another postulate is that of increased automaticity.[20] Both these mechanisms are believed by some to be eradicated with the improvement of blood supply to the ischaemic myocardium, such as is possible with CABG surgery. Cline *et al.*[21] reported on 2 men aged 43 years and 50 years, both of whom developed ventricular tachycardia and fibrillation during the early stages of an exercise test and who were receiving anti-arrhythmic treatment. The younger man suffered from angina as well as presyncope and syncope, and was found to have a significant obstruction in the LAD coronary artery in which a CABG was urgently inserted. The older patient had had an inferior MI some 3 years previously and was found to have triple-vessel coronary artery disease requiring triple CABG surgery. In both these patients the ventricular tachycardia and fibrillation were not reproducible after CABG and their symptoms of angina and syncope completely disappeared without additional drug therapy. This report was followed by that of Bryson and co-workers[22] who reported the success of CABG surgery in 3 young men who had had severe angina pectoris and syncope, and in whom ventricular tachycardia and fibrillation was reproduced on pre-operative exercise testing. Postoperatively stress tests failed to elicit ventricular arrhythmias and they remained asymptomatic off all therapy at the 2-year follow-up. None had a left ventricular aneurysm, although 2 of the 3 had had a previous MI. Postoperative cardiac catheterization showed that all the CABGs were patent and no new infarctions were visible on left ventricular cine angiography. Bryson and co-workers[22] were quite emphatic that 'the termination of the arrhythmias in these patients appears to be related solely to the relief of myocardial ischaemia by aortocoronary bypass surgery'. In an addendum to their article they documented a further case, that of a 37-year-old man who developed ventricular fibrillation very early during a stress test, and in whom obstructions of the left mainstem and LAD coronary artery were successfully bypassed with subsequent disappearance of the ventricular fibrillation on stress testing.

It has generally been accepted that premature ventricular ectopic beats are exceptionally common after an acute MI, and that the danger of deterioration of this arrhythmia into ventricular tachycardia and fibrillation is one of the main reasons for the existence of intensive coronary care units. This complication of acute MI has also stimulated the heated debate concerning the use and misuse of 'prophylactic' anti-arrhythmic therapy during this period. Surgical intervention for intractable ventricular tachycardia and fibrillation as represented by 'infarctectomy' with or without CABG surgery is far more controversial. Nevertheless, some impressive results have been reported in the literature. Kenaan *et al.*[23] documented 11 consecutive patients with intractable ventricular tachycardia treated surgically, 3 having had an acute MI, 4 long-term post-infarction left ventricular dysfunction and the remaining 4 definite left ventricular aneurysms. Of the 3 with acute MI, 2 had only CABG surgery whereas the remaining 1 had an infarctectomy. One of the former patients died after a re-infarction early after operation, whereas the other 2 survived and remained asymptomatic and free of ventricular arrhythmias. Four of the remaining 8 patients underwent aneurysmectomy; 1 of these had an additional CABG, another 2 had plication of an aneurysm with internal mammary implantation, and in the remaining 2 CABG surgery was undertaken in one instance and double internal mammary implantation in the other. One of the patients with a CABG died 10 days postoperatively after a fresh MI. Therefore 9 of the total of 11 patients survived and the conditions of 8 were markedly improved. Only 2 required long-term anti-arrhythmic therapy for recurrent ventricular arrhythmia. An interesting finding was that it took some days or weeks to abolish the ventricular arrhythmias, particularly if resection was undertaken.

Alexander and co-workers[24] reported on a 51-year-old woman with unstable angina accompanied by recurrent drug-resistant symptomatic ventricular fibrillation in whom coronary angiography demonstrated an isolated severe left mainstem coronary artery lesion. This patient underwent emergency CABG surgery to the LAD coronary artery with resultant disappearance of the ventricular arrhythmia as determined by repeated Holter monitoring. Repeat catheterization revealed a patent CABG with normal left ventricular contractility. This was therefore a further example of significant left mainstem coronary artery obstruction responding quite dramatically to emergency CABG surgery.

The successful control of medically refractory recurrent ventricular tachycardia and fibrillation, causing syncopal episodes in 2 young men with Prinzmetal's variant angina, was well demonstrated by Nordstrom *et al.*[25] Both these patients had significant multivessel coronary obstruction treated by CABG surgery, the grafts being shown to be patent post-operatively. The authors therefore concluded that 'coronary artery surgery may be an effective method of therapy for ischaemic ventricular tachycardia when medical therapy fails'.[25]

Other researchers have published data further supporting the benefits of CABG surgery in controlling ventricular arrhythmias. Myerburg *et al.*[26] reported on 13 patients who had survived an episode of unexpected ventricular fibrillation. All had complained of angina pectoris before the episode, from which they were resuscitated by a medically supervised fire rescue team. Eight had had ECG evidence of a definite or highly probable previous MI. All 13 underwent coronary angiography; 11 had graftable lesions but only 8 agreed to CABG surgery. These 8 survived for 8 - 32 months, 6 being totally asymptomatic and 2 still experiencing angina pectoris. These results were a marked improvement on the 1-year survival rate of some 30% in patients suffering unexpected ventricular fibrillation in hospital and in whom CABG surgery was not carried out. Ricks and co-workers[27] documented 21 patients with medically refractory symptomatic ventricular arrhythmias with varying severity of coronary artery involvement, 18 of whom had additional left ventricular aneurysms. Five patients had an aneurysmectomy with or without associated CABG surgery within 1 month of an acute MI, and only 1 of these survived. The other 16 patients, of whom 3 underwent only CABG surgery without aneurysmectomy, were operated on more than 1 month after the infarction and only 3 died, 7 weeks after the operation. Nevertheless, 3 of the long-term survivors have experienced repeated ventricular tachycardia requiring electroversion. These results suggest that surgery 1 month after infarction may help to control medically resistant ventricular tachycardia and fibrillation.

In order to improve the outcome of left ventricular aneurysmectomy with or without CABG surgery, Moran *et al.*[28] studied 30 patients who had had drug-resistant ventricular tachycardia from 1 week to 5 years after MI. Five were inoperable and 21 had a definite left ventricular aneurysm. Five underwent pre-operative electrophysiological studies and intra-operative epicardial mapping in an attempt to determine the focus and course of the ventricular arrhythmias, but no irritable focus could be precisely located pre-operatively or at operation, nor could they determine whether re-entry or automaticity was the basic pathophysiological mechanism involved. Nevertheless, 4 had survived from 12 to 27 months postoperatively, 3 being totally asymptomatic. Among the remaining 20 patients who did not undergo intra-operative epicardial mapping, there was a total of 13 early and late deaths. Moran *et al.*[28] therefore concluded that intra-operative epicardial mapping was of importance in these cases. Despite this, Gallagher[29] was rather cautious in stating that 'revascularization of patients presenting with serious ventricular arrhythmias refractory to medical therapy and associated with significant lesions in the coronary vasculature deserves further clinical trial'. This statement is supported by Wellens *et al.*,[30] who were not impressed with the long-term drug treatment of ventricular tachycardia associated with IHD in spite of the use of programmed cardiac stimulation in assessing the efficacy of the various anti-arrhythmic agents available, and suggested the 'careful evaluation of the long-term efficacy of surgical therapy of symptomatic ventricular tachycardia'.

Studies refuting the effectiveness of CABG surgery for ventricular arrhythmias

In contrast to publications on the success of CABG surgery with or without left ventricular aneurysmectomy in controlling medically refractory ventricular tachycardia and fibrillation, several authors believe that this surgical intervention is of no additional value when compared with medical therapy. Mathur *et al.*[2] studied 72 patients who were randomized into medical and surgical (CABG) treatment groups. They found that 4 patients in the surgical group in fact experienced more frequent exertional ventricular tachycardia and that CABG surgery had obviously failed in these patients. Giacchino *et al.*[31] reported on 15 patients with intractable recurrent ventricular tachycardia secondary to IDH, all of whom had a left ventricular aneurysm; 11 had an aneurysmectomy with or without added CABG surgery. Despite this, 10 of the 15 patients were dead within 18 months of investigation. Giacchino *et al.*[31] therefore concluded that 'patients with severe coronary artery disease, marked LV dysfunction, and recurrent ventricular tachycardia have a high mortality with surgical or medical management'. De Soyza and co-workers[32] compared 43 patients treated medically for ventricular arrhythmias with 40 comparable ones treated by CABG surgery 1 year previously. Graft patency was shown to be 90% at that time. Holter monitoring was utilized to show that there was no significant difference in the frequency of ventricular arrhythmias in the two treatment groups. Tilkian *et al.*,[33] analysing 140 patients complaining primarily of angina pectoris and found to have exercise-provoked ventricular arrhythmias, randomized these patients to medical and surgical (CAGB) treatment groups.

They found that 1 year later the frequency or severity of exercise-induced ventricular arrhythmias had not decreased in spite of a decrease in myocardial ischaemia as shown by improvement in exercise tolerance, diminished ST-segment depression on effort testing, relief of angina pectoris and patency of the CABGs. Quite unexpectedly, complex ventricular arrhythmias sometimes arose after successful CABG surgery. Tilkian *et al.*[33] therefore concluded that 'aortocoronary bypass surgery should not be expected to decrease ventricular arrhythmias induced by exercise'. Guinn and Mathur,[34] in a similar study comprising 116 patients, could detect no statistical difference between medically and surgically treated groups, stating that 'coronary artery bypass surgery does not prevent or reduce the occurrence of PVCs or dangerous arrhythmias'. Using Holter monitoring, Graboys *et al.*[35] assessed 58 patients who had undergone CABG surgery for angina and could find no reduction in premature ventricular ectopic frequency or complexity even as late as 3 months postoperatively. Leutenegger *et al.*[36] studied the effect of CABG on ventricular arrhythmia in 27 patients with unstable angina pectoris by means of Holter monitoring pre-operatively and 100 days after CABG surgery. These workers found no statistically significant improvement in these arrhythmias and were therefore of the opinion that 'at the present time ventricular arrhythmias alone constitute no indication for bypass surgery'.[36] De Soyza *et al.*[37] analysed by means of a similar method the effect of CABG surgery on ventricular arrhythmias in 57 patients operated upon primarily for unstable angina.[38] None of these patients had ventricular tachycardia or fibrillation, and none of them underwent postoperative catheterization in order to assess the patency of the CABGs. Nevertheless, 80% had relief of their angina without any postoperative evidence of MI. De Soyza *et al.*[37] again concluded that CABG surgery had no significant beneficial effect on the frequency or complexity of premature ventricular contractions. Price *et al.*,[39] in an analysis of 62 patients receiving CABGs for angina pectoris, emphasized the fact that ventricular arrhythmias tend to be more frequent within the early (12 - 36 hours) postoperative period but decrease in frequency in the post-hospital (6 - 8 weeks) phase. This latter study has an important bearing on the conclusions arrived at by all the previous studies employing Holter assessment of ventricular arrhythmias.

Conclusion

From the evidence presented, there is indecision about the exact role of CABG surgery, with or without additional left ventricular aneurysmectomy/infarctectomy, in the control of ventricular arrhythmias of varying severity. However, in the light of the experience gained from our case a plea is made to consider CABG surgery outside of its usual indication in medically uncontrollable angina pectoris.

I wish to sincerely thank Miss H. Weymar of the Cardiology Unit, Tygerberg Hospital, for preparing the manuscript and the ECGs. Thanks are also due to Mr Chris Wilberforce, Head of the Department of Photography, for his painstaking preparation of the photographs. Finally, due appreciation is shown towards Dr C. Viviers, Chief Medical Superintendent of Tygerberg Hospital, for permission to publish.

REFERENCES

1. McIntosh HD, Garcia JA. The first decade of aortocoronary bypass grafting, 1967-1977: a review. *Circulation* 1978; **57:** 405-431.
2. Mathur VS, Guinn GA, Anastassiades LC *et al.* Surgical treatment for stable angina pectoris: prospective randomized study. *N Engl J Med* 1975; **292:** 709-713.
3. Scheidt S. Unstable angina: medical management or surgery. *Cardiovasc Med* 1977; **2:** 541-553.
4. Takaro T, Hultgren HN, Lipton MJ, Detre KM. The Veterans Administration cooperative randomized study of surgery for coronary occlusive disease: II. Subgroup with significant left main lesions. *Circulation* 1976; **54:** suppl III, 107-117.
5. Cosgrove DM, Loop FD, Sheldon WC. Results of myocardial revascularization: a 12-year experience. *Circulation* 1982; **65:** suppl II, 37-43.
6. Lawrie GM, Morris GC. Survival after coronary artery bypass surgery in specific patient groups. *Circulation* 1982; **65:** suppl II, 43-48.
7. McConahay DR, Killen DA, McCallister SC *et al.* Coronary artery bypass surgery for left main coronary artery disease. *Am J Cardiol* 1976; **37:** 885-889.
8. Hammermeister KE, De Rouen TA, Murray JA, Dodge HT. Effect of aortocoronary saphenous vein bypass grafting on death and sudden death: comparison of nonrandomized medically and surgically treated cohorts with comparable coronary disease and left ventricular function. *Am J Cardiol* 1977; **39:** 925-934.
9. Mundth ED, Harthorne JW, Buckley MJ, Diusmore R, Austen WG. Direct coronary arterial revascularization treatment of cardiac failure associated with coronary artery disease. *Arch Surg* 1971; **103:** 529-534.
10. Chatterjee K, Swan HJC, Parmley WW, Sustaita H, Marcus H, Matloff J. Depression of ventricular function due to acute myocardial ischemia and its reversal after aortocoronary saphenous vein bypass. *N Engl J Med* 1972; **286:** 1117-1122.
11. Najafi H, Escamilla HA, Clark JG. Acute coronary insufficiency and life-threatening cardiac arrhythmias eight months after triple heart valve replacement. *Surg Clin North Am* 1970; **50:** 1119-1127.
12. Hutchinson JE, Kemp HG, Schwartz MJ. Emergency treatment of cardiac arrest in CHD with coronary bypass graft. *JAMA* 1971; **216:** 1645.
13. Nakhjavan FK, Morse DP, Nichols HT, Goldberg H. Emergency aorto-coronary bypass: treatment of ventricular tachycardia due to ischemic heart disease. *JAMA* 1971; **216:** 2138-2140.
14. Ecker RR, Mullins CB, Grammer JC, Rea WJ, Atkins JM. Control of intractable ventricular tachycardia by coronary revascularization. *Circulation* 1971; **44:** 666-670.
15. Lambert CJ, Adam M, Geisler GF *et al.* Emergency myocardial revascularization for impending infarctions and arrhythmias. *J Thorac Cardiovasc Surg* 1971; **62:** 522-528.
16. Graham AF, Miller C, Stinson EB, Daily PO, Harrison DC. Surgical treatment for life-threatening ventricular arrhythmias (Abstract). *Am J Cardiol* 1973; **31:** 136.
17. Mundth ED, Buckley MJ, DeSanctis RW, Daggett WM, Austen WG. Surgical treatment of ventricular irritability. *J Thorac Cardiovasc Surg* 1973; **66:** 943-951.
18. Radstein M, Wallach L, Gubner RS. Mortality study of the significance of extrasystoles in an insured population. *Circulation* 1971; **44:** 617-625.
19. Han J. The concepts of reentrant activity responsible for ectopic rhythms. *Am J Cardiol* 1971; **28:** 253-262.
20. Moe GK, Mendez C. Physiologic basis of premature beats and sustained tachycardias. *N Engl J Med* 1973; **288:** 250-254.
21. Cline RE, Armstrong RG, Stanford W. Successful myocardial revascularization after ventricular fibrillation induced by treadmill exercise. *J Thorac Cardiovasc Surg* 1973; **65:** 802-805.
22. Bryson AL, Parisi AF, Schechter E, Wolfson S. Life-threatening ventricular arrhythmias induced by exercise: cessation after coronary bypass surgery. *Am J Cardiol* 1973; **32:** 995-999.
23. Kenaan G, Mendez AM, Zubiate P, Gray R, Kay JH. Surgery for ventricular tachycardia unresponsive to medical treatment. *Chest* 1973; **64:** 574-578.
24. Alexander S, Makar Y, Ellis H. Recurrent ventricular fibrillation: treatment by emergency aortocoronary saphenous vein bypass. *JAMA* 1974; **228:** 70-72.
25. Nordstrom LA, Lillehei JP, Adicoff A, Sako Y, Gobel FL. Coronary artery surgery for recurrent ventricular arrhythmias in patients with variant angina. *Am Heart J* 1975; **89:** 236-241.
26. Myerburg RJ, Ghahramani A, Mallon SM, Castellanos A, Kaiser G. Coronary revascularization in patients surviving unexpected ventricular fibrillation. *Circulation* 1975; **59:** suppl III, 219-222.
27. Ricks WB, Winkle RA, Shumway NE, Harrison DC. Surgical management of life-threatening ventricular arrhythmias in patients with coronary artery disease. *Circulation* 1977; **56:** 38-42.
28. Moran JM, Talano JV, Euler D *et al.* Refractory ventricular arrhythmia: role of intraoperative electrophysiological study. *Surgery* 1977; **82:** 809-815.
29. Gallagher JJ. Surgical treatment of arrhythmias: current status and future directions. *Am J Cardiol* 1978; **41:** 1035-1044.
30. Wellens HJJ, Bär FWHM, Vanagt EJDM, Brugada P. Medical treatment of ventricular tachycardia: considerations in the selection of patients for surgical treatment. *Am J Cardiol* 1982; **49:** 186-193.
31. Giacchino R, Moran JF, Schreiber R, Johnson S, Gunnar R. The problem of recurrent ventricular tachycardia in the presence of angiographically proven coronary artery disease (Abstract). *Circulation* 1975; **51/52:** suppl II, 11-110.
32. De Soyza N, Murphy ML, Bissett JK, Kane JJ. A comparison of ventricular arrhythmia in coronary artery disease patients randomized to surgical and medical therapy (Abstract). *Clin Res* 1976; **24:** 2A.
33. Tilkian AG, Pfeifer JF, Barry WH, Lipton MJ, Hultgren HN. The effect of coronary bypass surgery on exercise-induced ventricular arrhythmias. *Am Heart J* 1976; **92:** 707-714.
34. Guinn GA, Mathur VS. Ambulatory, nocturnal and exercise arrhythmias in coronary artery disease: a prospective randomized study to assess the influence of aorto-coronary bypass surgery (Abstract). *Am J Cardiol* 1977; **39:** 270.
35. Graboys TB, Lown B, Collins JJ, Cohn LH. Does coronary revascularization reduce the prevalence of ventricular ectopic activity? (Abstract). *Am J Cardiol* 1978; **41:** 401.
36. Leutenegger F, Giger G, Fuhr P *et al.* Evaluation of aorto-coronary venous bypass grafting for prevention of cardiac arrhythmia. *Am Heart J* 1979; **98:** 15-19.
37. De Soyza N, Thenabadu N, Murphy ML, Kane JJ, Doherty JE. Ventricular arrhythmia before and after aorto-coronary bypass surgery. *Int J Cardiol* 1981; **1:** 123-130.
38. Horowitz LN. Ventricular arrhythmia before and after aorto-coronary bypass surgery (Editorial). *Int J Cardiol* 1981; **1:** 131-132.
39. Price JE, Vismara LA, Amsterdam EA *et al.* Evaluation of ventricular arrhythmias post-coronary bypass surgery: decreased prevalence following hospital discharge determined by ambulatory ECG monitoring (Abstract). *Am J Cardiol* 1977; **39:** 269.