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Coronary Artery Spasms after Coronary Artery Bypass Surgery

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Despite the improvement in perioperative management and surgical techniques, myocardial ischemia and necrosis still may result following coronary artery bypass grafting (CABG) surgery.[1] One possible mechanism is the coronary artery spasm[2,3] as characterized by ST-segment elevation, arrhythmias, atrioventricular block, severe hypotension, and even cardiac arrest occurring after discontinuation of cardiopulmonary bypass. Recent case reports suggest that the condition may be effectively treated and possibly has no bearing on the long-term prognosis of the surviving patients.[4–6]

In the last 3 years, we observed and documented 10 cases of severe myocardial ischemia after CABG, which fit the clinical features of the coronary artery spasm and were able to confirm the dangers of the condition as well as the effectiveness of treatment with vasodilating drugs.

REPORT OF TEN CASES

Between 1979 and 1982, 397 patients underwent CABG surgery. Ten of these patients developed severe acute myocardial ischemia compatible with the diagnosis of coronary artery spasm immediately after surgery. Eight patients complained preoperatively of angina at rest (nocturnal angina in four patients), which contrasted with their good exercise tolerance. Seven patients suffered a myocardial infarction (nontransmural in four patients) an average of six (range 1–18) months preoperatively. Besides long-acting nitrates and nitroglycerin, six patients were taking nifedipine preoperatively, two patients were taking both nifedipine and beta-adrenergic blocking drugs, while two patients took beta-adrenergic blocking drugs only. With the exception of patients 1 and 2, who had an isolated left anterior descending (LAD) stenosis, the preoperative coronarography demonstrated a three-vessel disease in all other patients. In five patients, coronary artery spasms were observed during coronary angiography. The preoperative antianginal drugs were continued until the evening before surgery. Starting with the third patient, all patients receiving nifedipine preoperatively were given their last nifedipine dose with the premedication (morphine and scopolamine, im). Except for patient 2, all patients had a flow-directed (Swan-Ganz®) catheter inserted preoperatively. Leads V5 and II of the EKG were monitored continuously; other extremity leads were recorded intermittently.

Anesthesia was induced with flunitrazepam (range 0.6–2.1 mg), fentanyl (range 0.05–0.4 mg) and thiopental (range 40–200 mg) iv. Intubation of the trachea was facilitated by succinylcholine. For the maintenance of anesthesia, nitrous oxide (50%), fentanyl iv (in increments up to a total of 0.7–2.4 mg), and pancuronium iv were used. An additional dose of flunitrazepam (range 0.1–1.4 mg) was given on cardiopulmonary bypass in seven patients. In nine patients, a nitroglycerin infusion (range 0.25–2.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$ before and 1.0–3.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$ after the cardiopulmonary bypass) was started after induction of anesthesia and maintained throughout the operation.

Five patients required alpha-adrenergic agonists (phenylephrine 5.0–40.0 $\mu g \cdot min^{-1}$ for four patients and methoxamine 5.0 mg as a single bolus for the fifth) between induction of anesthesia and sternotomy. Eight patients were given propranolol (range 1.0–4.0 mg) and one patient oxprenolol (0.3 mg) iv prior to cardiopulmonary bypass.

St. Thomas' cardioplegia complemented by continuous pericardial cooling was used for myocardial protection. A mean of 3.1 ± 1 SD (range 1–4) grafts were inserted. The mean aortic cross clamp time was 59 ± 23 (range 26–99) minutes. In three patients a sympathetic denervation of the aortic root was performed in addition to CABG. During the period of ischemia, neither electrical nor mechanical activity of the heart was observed. Measurements of myocardial temperatures at onset and at end of aortic cross clamping confirmed an adequate myocardial hypothermia (table 1).

The intraoperative and postoperative hemodynamic data are presented in table 2. Prior to the induction of anesthesia, we found a normal hemodynamic function. Until the cardiopulmonary bypass, no signs of an increased myocardial oxygen demand were encountered in any of the patients. As the low blood pressure following induction in five patients responded promptly to alpha-adrenergic agonists, no significant hypotension occurred either. A profound reduction in ventricular function was observed immediately following the cardiopulmonary bypass. At that time, two patients had an elevated left ventricular filling pressure, while in eight patients it still was normal. Thus, the depressed ventricular function was not due to hypovolemia.

The circulatory collapse occurred in the period between the end of the cardiopulmonary bypass and the end of operation (seven patients) or 15–150 min postoperatively (three patients). It began with a progressive fall of arterial pressure. In seven patients the left atrial pressure increased (range 16–45 mmHg), but it remained normal (range 5–10 mmHg) in three patients. The right atrial pressure exceeded 10 mmHg in all but one patient. Eventually, either open chest massage or resumption of the cardiopulmonary bypass was necessary to restore the heart function in six patients.

The hemodynamic changes were preceded by ST-segment elevation in all patients. The ST-segments showed the pattern of acute myocardial infarction and appeared in inferior leads (II, III, avF) in five patients, in inferolateral leads (II, III, avF, avL and V5) in three patients, and in anterior leads (I, II, avL, V5) in two patients. Seven patients required sequential AV pacing because of a transient heart block.

The first patient of this series received massive iv doses of catecholamines but only a low dose of nitroglycerin and died after 8 h resuscitation. He was not receiving nifedipine preoperatively, nor did he receive it postoperatively. Six other patients were given iv adrenaline (range 0.4–4.0 $\mu g \cdot min^{-1}$) with no effect on the hypotension and low cardiac output. Instead of increasing the catecholamine dose as in the

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TABLE 1. Myocardial Temperatures in Patients Who Developed Coronary Artery Spasm*

	After Termination of the First Cardioplegic Infusion		Immediately before Declamping of the Aorta	
	n		n	
Septum	9	10 ± 1.0 (6–14)	8	16.6 ± 2.0 (7–24)
Posterolateral left ventricular wall	10	13.8 ± 1.6 (7–20)	9	14.7 ± 1.3 (10–21)
Inferior left ventricular wall	10	10.7 ± 0.9 (7–14)	8	15.6 ± 1.7 (10–22)
Anterior right ventricular wall	10	10.8 ± 0.9 (7–14)	8	17.2 ± 1.3 (11–21)

* Degrees centigrade, means ± SE (range).

first patient, we decided to employ the intraaortic balloon pump (table 3). However, the final lasting improvement in all nine surviving patients was brought about by either iv and sublingual nifedipine or intracoronary nitroglycerin (table 3, Figs. 1–3). The hemodynamics stabilized within 15–120 min after the start of the treatment. The severe depression of ventricular function still persisted even after the return of arterial blood pressure to within normal levels. Cardiac output values returned to normal by the first postoperative day, while the stroke indices remained decreased.

In patient 1, the autopsy revealed an acute anteroseptal infarction. All coronary arteries were patent. A thrombus was found in the distal end of the graft to the left anterior descendent artery. A new transmural infarction in the presence of patent grafts also was detected postoperatively in patient 8. The diagnosis was based on the occurrence of new Q waves in the EKG, a new perfusion defect in the thallium scan, a new akinetic area in the ventriculogram, and a peak CK-MB isoenzyme value well above the upper limit compatible with uncomplicated CABG surgery in our institution (60 U/l) (table 4). One additional patient (patient 6) developed new Q waves in the inferior leads with normal thallium scan, left ventricular wall motion, and CK-MB isoenzyme. The graft to the right coronary artery was closed in this patient. The further postoperative course in all nine surviving patients was uneventful.

DISCUSSION

In our documented group of patients, severe myocardial ischemia occurred following coronary artery bypass graft surgery. Several mechanisms, such as poor myocardial protection, excessive O_2 demand, and decreased myocardial O_2 supply, should be considered as causes for this unexpected ischemia.

Inadequate myocardial protection during the cross-clamping of the aorta is the most common cause of postoperative ischemia and cardiac failure. Its incidence has decreased after the introduction of cold cardioplegia, but there was no indication that our 10 patients were protected less effectively than the great majority of our patients with uncomplicated recoveries.

Inappropriate intraoperative anesthetic management leading to high sympathetic tone, hypertension, and tachycardia may cause ischemia due to excessive O_2 demand; hypotension may cause ischemia by decreasing the poststenotic coronary perfusion pressure. Our hemody-

TABLE 2. The Perioperative Hemodynamics in Patients Who Developed Coronary Artery Spasm (Means ± SE)

		Intraoperatively					Postoperatively			
	n	Before Induction	n	Before Bypass	n	After Bypass	n	After Recovery from Spasm	n	First Postoperative Day
Heart rate (beats/min)	10	60.6 ± 2.0	10	72.9 ± 2.6	10	89.2 ± 1.4	10	96.1 ± 2.2	9	96.4 ± 5.5
Mean arterial blood pressure (mmHg)	10	85.7 ± 2.8	10	85.6 ± 5.5	10	75.0 ± 2.9	10	95.3 ± 5.8	9	84.2 ± 4.0
Right atrial pressure (mmHg)	7	4.8 ± 1.1	10	6.9 ± 0.9	10	8.8 ± 0.8	10	9.4 ± 1.0	9	10.5 ± 0.9
Pulmonary capillary wedge pressure (mmHg)	7	10.1 ± 1.1	10	8.7 ± 0.8	10	12.2 ± 1.4	10	10.5 ± 0.7	9	11.1 ± 0.8
Cardiac index ($l \cdot min^{-1} \cdot m^{-2}$)	7	2.63 ± 0.15	9	2.11 ± 0.16	8	1.79 ± 0.15	8	1.77 ± 0.16	8	2.64 ± 0.16
Stroke volume index ($ml \cdot m^{-2}$)	7	43.0 ± 3.7	9	29.0 ± 2.4	8	20.0 ± 2.0	8	18.2 ± 1.7	8	27.0 ± 2.2
Systemic vascular resistance ($dyn \cdot sc \cdot cm^{-5}$)	7	1,251 ± 59	9	1,519 ± 159	8	1,505 ± 103	8	2,160 ± 290	8	1,151 ± 102

namic monitoring revealed no such conditions in any patient prior to or during the bypass. A primary decrease in myocardial O_2 supply appears to be the major problem. ST-segment elevation always preceded the hemodynamic changes, and in two patients the ST-segment changes were observed before the grafting procedure.

Transient ischemia may be caused by coronary air embolism. This usually occurs soon after clamping of the aorta with the resumption of effective heart contractions. Air then may be visible in the subepicardial vessels and grafts. It quickly resolves after measures aiming to increase the coronary perfusion pressure and rarely recurs. The late occurrence, persistence, and severity of ischemia, in spite of an earlier careful evacuation of air from the heart and the grafts, does not support air embolism as the mechanism of ischemia in our patients.

A failure in revascularization cannot satisfactorily explain the ischemia either. The early graft patency rate in those patients who were reinvestigated postoperatively was not different from our recently reported results with uneventful course.[7] Patients 1 and 6, who suffered intraoperative myocardial infarction indeed had occluded grafts but still patent native arteries to the infarcted area. Perhaps a coronary artery spasm caused irreversible ischemia and infarction and the graft thrombosis in these two patients was secondary to the poor or absent run off in the grafted region. A myocardial infarction due to coronary artery spasm in a patient with variant angina recently has been documented by means of coronary angiography.[9] On the other hand, until now no obvious relationship between graft occlusion and perioperative infarction could be identified[1] and, in a recent study, 83% of seventy patients with perioperative infarction had patent grafts to the area of infarction.[8]

An occlusion of the artery due to embolism of particulate matter or thrombus also could have been responsible for the ischemia. However, three patients reported in the literature showed no thrombotic occlusion during emergency angiography but a diffuse coronary spasm that abated after intracoronary injection of nitroglycerin[3,10] or endotracheal amyl nitrate.[11] We did not perform emergency coronary angiography in our 10 patients; patient 1 could not be stabilized enough to allow for transport to the catheterization laboratory, and in the other patients we obtained an immediate response to the treatment with vasodilator drugs, a result that one would not expect in a case of thrombotic occlusion of the coronary artery and that supports our conclusion that the ischemia was due to coronary artery spasms. This conclusion is underlined further by the fact that in five of the 10 patients, coronary spasms were demonstrated by preoperative angiography and that eight of 10 patients presented with pain at rest and/or nocturnal angina, suggestive of a vasospastic component of their preoperative ischemia.[12]

The mechanism responsible for the increased incidence of coronary spasm during the early postbypass and postoperative period is not known. Sympathoadrenergic stimulation with an unopposed alpha receptor mediated coronary vasoconstriction in patients receiving beta-adrenergic blocking drugs, respiratory alkalosis due to hyperventilation, cold, local trauma, release of vasoconstricting substances by platelets, and high local concentration of potassium have all been proposed.[2,3,12,13] Other factors are also conceivable: plasma–vasopressin increases during aortocoronary bypass surgery[14] and may provoke

TABLE 3. Treatment of Patients Who Developed Coronary Artery Spasm

Patient No.	Cardio-pulmonary Bypass	Open Chest Massage	IABP	Vasodilator Drugs	
				Nitroglycerin $\mu g \cdot min^{-1}$	Nifedipine mg
1	0	+	+	80	0
2	1×	+	+	430	0
3	0	+	+	150	2.0 iv
4	2×	0	+	240	0.8 iv
5	1×	0	+	140	1.0 iv
6	0	0	0	160	1.0 iv
7	0	0	0	340*	0.8 iv
8	0	+	+	400*	20.0 s. ling
9	0	0	0	340†	20.0 s. ling
10	0	0	0	200†	0.95 iv

* Additional nitroglycerin given intracoronary.
† Additional nitroglycerin given in the left atrial catheter.

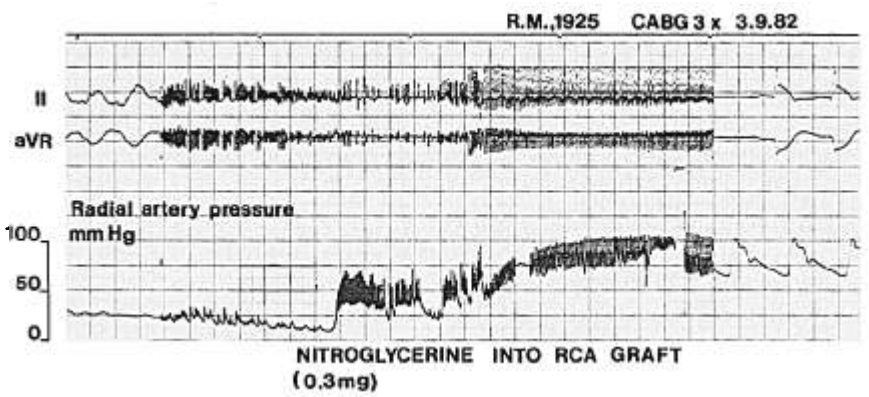

FIG. 1. Patient 8. From top to bottom, Leads II and aVR of the EKG and the radial artery pressure recording during an open-chest resuscitation in the intensive care unit following cardiac arrest that occurred 1 h after surgery. Nitroglycerin (0.3 mg) was injected into the graft to the right coronary artery and restored the spontaneous heart action.

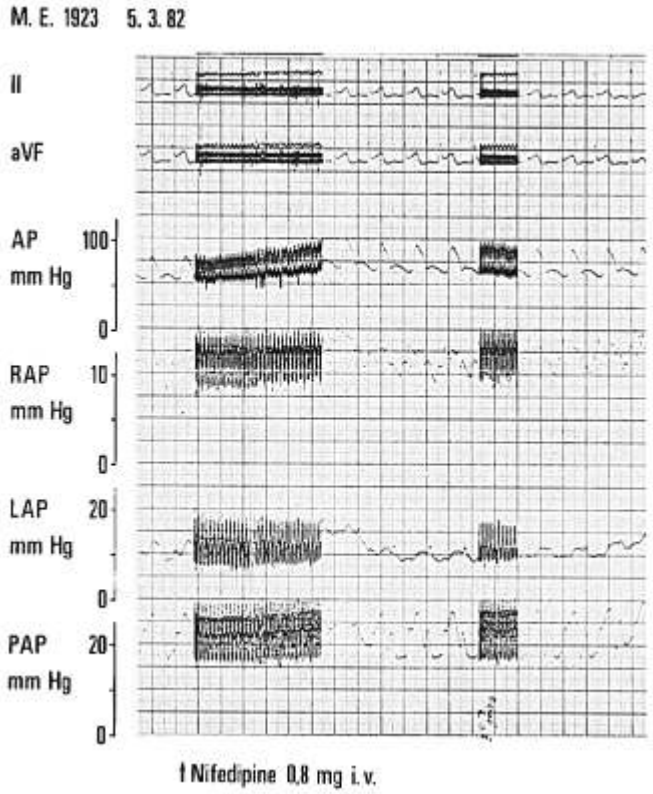

FIG. 2. Patient 6. From top to bottom, Leads II and aVF of the EKG, radial artery pressure (AP), right atrial pressure (RAP), left atrial pressure (LAP) and pulmonary artery pressure (PAP) were recorded. ST-segment elevation in the inferior leads, hypotension, and low cardiac index (1.5 $l \cdot min^{-1} \cdot m^{-2}$) occurred shortly after closure of sternum. During a slow injection of nifedipine (0.8 mg iv over 3 min) the ST-segment, the arterial pressure, and the cardiac index (2.2 $l \cdot min^{-1} \cdot m^{-2}$) improved significantly.

coronary artery spasm in arteries sensibilized by cold and mechanical injury. Histamine liberation with the possibility of an H_1-receptor mediated coronary vasoconstriction[15-17] may occur in the postbypass period due to allergic reactions to protamine and blood products. We observed no such reactions in any of our patients. A decrease of extracellular magnesium concentration increases the coronary vascular tone and potentiates the response of coronary arteries to vasoconstricting agents.[18] We did not measure plasma magnesium in our patients, but we found a significant increase of plasma magnesium following the use of St. Thomas' cardioplegia in a comparable group of patients submitted to CABG surgery.[19] A hypomagnesemia therefore was not likely to occur in our patients. Recently, thrombin, which readily is activated during surgery, was shown to be a potent vasoconstrictor of ischemic coronary arteries with damaged endothelium.[20] The decreasing heparin levels at the end of cardiopulmonary bypass and the subsequent heparin reversal by protamine could lead to a sudden increase of thrombin in the coronary blood and subsequent vasoconstriction. The endothelium of coronary arteries is highly susceptible to ischemic injury,[21] which may be unrelated to the more easily detected ischemia of the contractile myocardium. A disproportionate rewarming of the endothelium during aortic cross-clamping due to collateral blood flow leading to ischemic damage and to exaggerated vasoconstrictor responses of the coronary arteries cannot be excluded in our patients. Lastly, a rebound vasoconstriction involving coronary arteries and caused by a sudden withdrawal of short acting vasodilator drugs such as organic nitrates and nifedipine could be a factor.[22,23] However, our patients received their last nifedipine dose with the premedication, and the nitroglycerin infusion was started in all but one patient prior to the skin incision. Perhaps higher doses of these drugs would have protected our patients better. An established

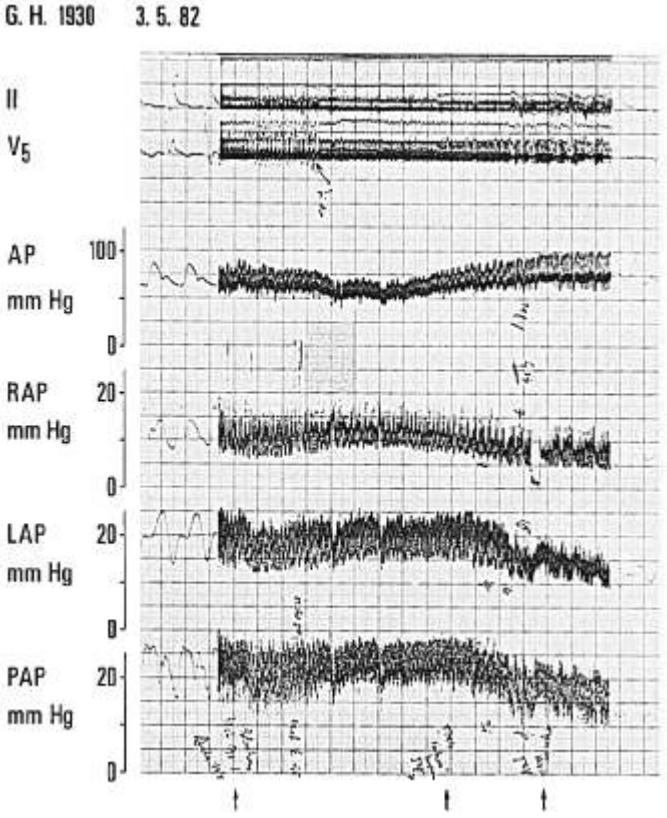

FIG. 3. Patient 7. The recorded parameters are the same as in figure 2. ST-segment elevation in inferior leads and hypotension occurred shortly after termination of cardiopulmonary bypass and were followed by cardiac arrest. Open-chest massage restored the spontaneous heart action, but the hypotension, high left atrial pressure, and low cardiac index (1.1 $l \cdot min^{-1} \cdot m^{-2}$) still persisted 15 min later. At the first arrow, the last increment of iv nifedipine (0.1 mg) of the total 0.8 mg was given. A test dose of adrenalin (10 μg iv) as a single bolus was injected but failed to increase the arterial pressure and provoked ventricular premature beats. At the second and third arrows, 0.05 mg nitroglycerin was injected into the grafts to right coronary and left anterior descendent arteries and resulted in a lasting improvement of the hemodynamics, characterized by an increase in both arterial pressure and the cardiac index (to 2.15 $l \cdot min^{-1} \cdot m^{-2}$) and a decrease of filling pressures.

TABLE 4. Postoperative Controls in Patients Who Developed Coronary Spasm after CABG

Patient Number	Number of Grafts	Thallim Scan	Peak CK-MB U/l	EKG (New Q Waves)	Coronary Angiography (Grafts)
1	1	—	126	—	—
2	1	Normal	35	0	—
3	3	Old defect	79	0	—
4	3	Normal	44	0	Patent
5	3	Old defect	49	0	Patent
6	3	Normal	51	+	1 closed
7	4	Old defect	53	0	Patent
8	4	New defect	145	+	Patent
9	4	Normal	53	0	Patent
10	4	Normal	45	0	—

coronary spasm responds poorly to iv nitroglycerin, even when given in doses as high as 1.0 mg · min^{-1} or in repeated 1.0-mg boluses.[24] Intracoronary nitroglycerin proved to be more reliable for the treatment of postoperative coronary spasm.[3,4,6,11] Nifedipine appears to be equally effective and resolved intraoperative spasm even when given sublingually.[5,25,26] This is of practical importance as long as iv nifedipine remains available only for clinical research purposes. Other vasodilators used successfully include intracoronary papaverine,[2] amyl nitrate aerosol intratracheally,[12] and iv phentolamine.[3]

Thus severe myocardial ischemia, especially if it occurs in patients with variant angina immediately after coronary artery bypass grafting, may be due to coronary artery spasm. This ischemia may seriously compromise cardiovascular function and eventually lead to cardiac arrest, but it may respond favorably to nifedipine and nitroglycerin. Coronary artery spasm also may be an important contributing factor to perioperative myocardial infarction.

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