cooling was attempted, the peripheral temperature fell as the central temperature rose to 41°C (105.8°F). Only after peripheral vasodilatation was achieved did the central temperature fall. In the report by Spitzer and Brock, the patient was a 25-year-old Welsh master butcher who underwent an aortic valve replacement. His postoperative course was complicated by high core temperatures (41.2°C; 106.2°F) low peripheral temperature (19°C; 66.2°F), neurologic deterioration, and hypovolemia. Treatment with volume expansion and chlorpromazine was totally successful, both in reducing the patient's temperature to normal and in reversing his neurologic deterioration.

In the patient described herein, we propose that the extremely high fever was caused by the following pathophysiologic cascade: bronchoscopic examination induced her junctional rhythm, either directly or in conjunction with a low-grade fever caused by one of the previously mentioned sources. The junctional rhythm caused a further decrease in the patient's cardiac output which, in turn, caused poor peripheral perfusion and vasoconstriction. As the temperature of her skin fell, the patient was able to transfer less heat to the environment. This resulted in a rise in core temperature. This increase in the central temperature demanded an increased cardiac output, a demand that the patient could not meet because of her severe mitral stenosis, aortic stenosis, and junctional rhythm. Florid left and right ventricular failure ensued, causing a further drop in cardiac output and peripheral perfusion, thus completing the vicious cycle. As might be predicted, surface cooling did not alter the deterioration in the patient's condition.

In patients with high core temperatures caused or perpetuated by peripheral hypoperfusion, therapy with volume expansion and pharmacologic vasodilatation is usually successful. In this patient, increasing the perfusion by giving volume expanders was unappealing in the face of florid pulmonary edema. So, too, was giving a vasodilator drug in the presence of uncorrected shock. The break in the previously outlined cycle came rather dramatically by achieving core cooling with iced lavage of the patient's stomach. Her pulse rate fell pari passu with her temperature, and as sinus rhythm reappeared, her blood pressure and output of urine increased.

In the occasional patient with hyperpyrexia and a fixed low cardiac output such as described herein, the options of administering volume expanders or vasodilator drugs, or both, may not be available. In such a case, core cooling may be valuable in interrupting the progressive cycle of fever and shock. In this case, iced gastric lavage was used, but cooling could also be performed via the peritoneal cavity (cold dialysis), colon (iced enemas), or blood (extracorporeal circulation).

REFERENCES

Myocardial Infarction Associated with Thyrotoxicosis*

A. J. Proskey, M.D.; Franklin Saksena, M.D.; and William D. Towne, M.D., F.C.C.P.

Myocardial infarction occurs rarely with thyrotoxicosis. A 34-year-old woman with thyrotoxicosis sustained a transmural myocardial infarction and subsequently on cardiac catheterization studies had no significant coronary arterial disease but only residual apical wall akinesia. Thyroid hormone may directly influence myocardial oxygen supply and demand and, by some unknown mechanism exclusive of major coronary arterial blood supply, cause a critical imbalance resulting in angina pectoris and myocardial infarction.

Although angina pectoris occurs occasionally in patients with thyrotoxicosis, myocardial infarction has been reported rarely. In both instances, underlying atheromatous coronary disease has been presumed present as the cause. Although the physiologic effects of thyroid hormone on the cardiovascular system are well documented, little is known about the potential deleterious effect of excessive thyroid hormone on the heart in the absence of apparent heart disease.

The purpose of this report is to describe the case of a young woman presenting with unstable angina pectoris and active thyrotoxicosis who sustained a transmural myocardial infarction and who, on subsequent cardiac catheterization studies, had no significant coronary disease but only apical wall akinesia as a result of the past myocardial infarction.

CASE REPORT

The patient, a 34-year-old woman, was admitted via the emergency room on June 14, 1975, because of severe retro-

*From the Division of Adult Cardiology, Department of Medicine, Cook County Hospital, Northwestern University, and Loyola University, Chicago.
sternal chest pain of several minutes' duration, associated with shortness of breath and diaphoresis. She had been experiencing the pain intermittently over the prior two weeks. Past history was not significant for hypertension, cigarette smoking, birth control medication, or a family history of heart disease or diabetes. On physical examination the blood pressure was 160/90 mm Hg, the pulse rate was 120 beats per minute, and the temperature was 37°C (98.6°F). The thyroid gland was enlarged to two times normal. There was a grade 2/6 systolic ejection murmur along the left sternal border. There was a fine tremor of the outstretched fingers, and the deep tendon reflexes were hyperactive.

On admission, all laboratory data were normal, except the electrocardiogram showed evidence of anterior wall ischemia and possible subendocardial injury, with T-wave inversion in leads V2 through V6 (Fig 1; June 14, 1975).

After admission to the intensive care unit, the patient continued to experience chest pain intermittently. Serial ECGs continued to show a similar pattern (Fig 1; June 20, 1975); however, serial determinations of serum enzyme levels remained normal. On June 20, 1975, the patient was started on oral therapy with propranolol (40 mg every six hours), in addition to intermittent sublingual administration of nitroglycerin and sedation. Studies of thyroid gland function revealed the following values: serum thyroxin iodine (T4), 17.5 μg/100 ml; and radioactive triiodothyronine (T₃) resin uptake, 38 percent. The 24-hour thyroid iodine uptake was 36 percent. The patient was given ten millicuries of radioactive ¹³¹Iodine on June 24, 1975.

On June 28, 1975, at 4:30 AM, the patient complained of chest pain and was given nitroglycerin sublingually. An ECG taken at that time (Fig 1; June 28, 1975) was diagnostic of acute anterior transmural myocardial infarction. One hour later, the patient experienced two successive runs of ventricular fibrillation which responded to electroshock and intravenous administration of lidocaine. On June 29, 1975, the ECG (Fig 1; June 29, 1975) showed evidence of an acute anterior and inferior transmural myocardial infarction. The serum concentrations of enzymes were elevated, as follows: serum glutamic-oxaloacetic transaminase, 173 international units (IU) per liter; lactic dehydrogenase, 1,208 IU/L; and creatine phosphokinase, 452 IU/L. The patient had also developed a fever, and because of the possibility of thyrotoxic storm, she was started on a regimen of hydrocortisone, sodium iodide, and propylthiouracil, in addition to propranolol. Subsequently, her condition improved gradually and uneventfully, and she was discharged on July 24, 1975. In October 1975, the patient was found to have become hypothyroid and responded to administration of L-thyroxine in gradually increasing low doses.

On Jan 7, 1976, the patient was readmitted for a cardiac catheterization. The ECG showed evidence of prior myocardial infarction (Fig 1; Jan 7, 1976). On cardiac catheterization, right-sided and left-sided pressures were normal, with a cardiac index of 3.1 L/min/sq m. Left ventricular angigram showed apical akinesia but with otherwise normal appearing contractility. The left ventricular ejection fraction was 0.55. No mitral regurgitation was noted. Coronary arteriograms revealed no evidence of any disease in any of the major vessels or branches (Fig 2). On discharge, the patient was asymptomatic and euthyroid and was taking L-thyroxine (0.1 mg/day).

**DISCUSSION**

Transmural myocardial infarction associated with thyrotoxicosis without evidence of permanent organic coronary arterial obstruction could result from (1) tem-

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explain the phenomenon of myocardial infarction with normal coronary arteries, such as an abnormality of hemoglobin-oxygen dissociation or small vessel disease, cannot be excluded completely. However, since the frequency and real causes of temporary coronary occlusion that probably precipitate myocardial infarction in cases not related to arteriosclerosis remain obscure, it is reasonable to conclude that an associated condition, such as thyrotoxicosis, could in itself be the responsible mechanism under certain circumstances.

Although adrenergic blockade, as by the use of propranolol in our patient, can ameliorate in part the cardiac responses in thyrotoxicosis that are presumably mediated by catecholamines through the sympathetic nervous system, residual hyperkinetic cardiac action is still apparent. Therefore, although the cardiac effects of thyroid hormone are mediated in part via the sympathetic nervous system, it has a direct effect on the myocardial contractility independent of catecholamines. As is possible in our case, there may be no mechanism to blunt the cardiac effect of excess thyroid hormone directly on the myocardium. Since the myocardium participates in the increased oxygen consumption characteristic of all tissues in thyrotoxicosis, it seems possible that excessive thyroid hormone could directly influence myocardial oxygen supply and demand, and that by some unknown mechanism exclusive of major coronary blood supply, cause in certain cases, such as ours, a critical imbalance resulting in angina pectoris and myocardial infarction.

**REFERENCES**