MAE. Wick et al reviewed 15 cases of MAE, 14 of which showed pulmonary vascular involvement. However, in none of the cases reports did the premortem clinical signs and symptoms direct the physician to the lung for biopsy and diagnosis.

In the series by Wick et al reporting 15 patients, 11 had fever of unknown origin and two had hyponatremia. Massive infiltration of the intraadrenal blood vessels leading to adrenal insufficiency has been forwarded as a possible explanation for fever, hyponatremia, and hypotension in MAE patients. We feel the clinical evidence in our case does not suggest hypothalamic, but is consistent with SIADH.

SIADH is a well-known consequence of both malignant tumors and pulmonary disease. Infected lung tissue may synthesize ADH, most notably in tuberculosis. The consideration of pulmonary tuberculosis in our patient prompted the bronchoscopy. The absence of significant pulmonary infiltrates precluded a transbronchial biopsy. No evidence of tuberculosis was discovered at autopsy, but pulmonary vascular infiltration by the cells of MAE was found. The availability of a specimen of lung tissue might well have led to a premortem diagnosis of MAE and a trial of chemotherapy. We suggest that SIADH in the setting of fever may warrant the consideration of a transbronchial biopsy despite the absence of parenchymal infiltrates.

REFERENCES

1 Pfleger VL, Tappeiner J. Zur Kenntnis der systemisierten endothelomastose der cutanen blutgefäße (reticuloendotheliose?). Der Hautarzt 1959; 10:359-63
11 Dolman CL, Sweeney VP, Magil A. Neoplastic angioendotheliomatosis: the case of the mixed primary? Arch Neurol 1979 36:5-7
12 Fulling KH, Gernell DJ. Neoplastic angioendotheliomatosis. Cancer 1983; 51:1107-18

Electrocardiographic J Waves after Resuscitation from Cardiac Arrest*  

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A patient was monitored prior to, during, and after cardiac arrest with a Holter monitor and an electrocardiograph. The arrest occurred without any premonitory signs on the ECG. At the onset of the arrest, torsades de pointes ventricular tachycardia occurred, which quickly degenerated into ventricular fibrillation. After a successful second defibrillation, the patient developed Osborn waves, which subsided within a few minutes. (Chest 1990; 98:1294-96)

The report of the findings in a patient who was monitored with multilead ECGs during cardiac arrest and successful resuscitation is presented.

Case Report

A 53-year-old white man suffered a lateral wall myocardial infarct one month prior to this event. Coronary angiography revealed that the left anterior descending coronary artery had a 100 percent occlusion, the diagonal artery had a 95 percent occlusion, and the posterior descending branch had a 50 percent occlusion. There was a small area of hypokinesia in the inferior wall. The left ventricular ejection fraction was 65 percent. The patient weighed 79 kg and was receiving diltiazem (80 mg four times daily). He was brought for aortocoronary bypass surgery.

Following informed consent for perioperative monitoring, a Holter monitor (Marquette 8500) was used to record a modified CM lead with the positive electrode at V₃ and the negative electrode below the right clavicle in the midclavicular line. Modified leads 3 and 1 with electrodes on the torso were also monitored. A digital electrocardiograph (Marquette MAC-12) was also used to record modified limb leads with electrodes on the torso and at V₃ and V₇.

On the morning of surgery, the patient received 10 mg of diazepam orally at 6 AM and 1 mg of lorazepam intravenously at 7:12 AM and again at 7:17 AM, during the placement of an introducer in the right internal jugular vein. A few minutes later, he was noted to be cyanotic, unresponsive, and in ventricular fibrillation. Cardiopulmonary resuscitation was performed, and defibrillation was attempted with 200 J and was successful with 300 J. Sinus rhythm returned, and satisfactory blood pressure was obtained. Infusion of nitroglycerin was started at 1 µg/kg-min.

Central nervous system damage was expected to be unlikely due to the arrest. It was decided to proceed with surgery. Fentanyl (25 µg/kg) and pancuronium (0.1 mg/kg) were administered, and the patient was intubated. The cardiopulmonary bypass was started 65 minutes later and lasted 90 minutes. A myocardial infarct was ruled out on the basis of intraoperative inspection of the myocardium and postoperative cardiac enzyme levels and ECGs. No neurologic or other deficits were noted after surgery.

Periarrest ECG

This monitored cardiac arrest is different in several respects from those reported earlier.1,4 This patient had no previous history of

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cardiac arrest or syncope or ventricular tachycardia. He had a nearly constant sinus rate of about 90/min and only one PVC in the 30 minutes preceding the arrest. The frequency of PVCs had not increased in the hours preceding the arrest, and there were no other warning arrhythmias. The corrected Q-T interval was 399 ms, which had been his usual value for the previous few days. There were no ST-T changes preceding the arrest.

Figure 1 shows the Holter ECG record immediately prior to the cardiac arrest. There were four multiform PVCs, followed by torsades de pointes ventricular tachycardia degenerating into ventricular flutter-fibrillation. The first defibrillation resulted in four ventricular beats, again followed by ventricular fibrillation-flutter. A second defibrillation delivered within 45 seconds restored a sustained sinus rhythm.

Immediately after resuscitation, the heart rate was about 80 beats per minute and variable, with a peak heart rate of 130 beats per minute during the first three minutes. Figure 2 shows the ECG waveforms in leads V4 and V5 before the arrest and immediately after resuscitation. Marked J-point elevation, new Q waves, and deep S waves were present in the precordial leads V4, V5, and CM4. The J-point elevation has been called an Osborn wave or J wave or camel-hump sign.3

After three minutes, the Osborn waves subsided in the precordial leads but became evident in the inferior leads 2, 3, and aVF, which by now had an R amplitude slightly greater than the value before the arrest. The Osborn waves in the inferior leads subsided within five minutes. Immediately after resuscitation the ECG vectors were virtually perpendicular to the frontal plane, and the complexes in the frontal plane leads were small. Lead 1 showed ST depression, and lead 3 showed J-point elevation on the Holter record. The amplitudes of the ECG complexes in the frontal plane leads increased in one minute. The ST depression in lead 1 subsided within 15 seconds.

During the three minutes following the reestablishment of the sustained rhythm, the patient had eight PACs, two PVCs, and one ventricular couplet. During the following 30 minutes the patient had two PVCs of the old form and three PVCs of a new form. The variability in the ECG waveforms and the R-R interval was reduced and occurred episodically. At the onset of cardiopulmonary bypass, on pouring cold physiologic saline solution on the heart, repolarization changes similar to those seen immediately after arrest were observed.

**DISCUSSION**

The cardiac arrest in all likelihood was secondary to respiratory arrest caused by excessive sedation. If so, this is the first report of electrocardiographic monitoring during such an arrest.

The J waves observed immediately after resuscitation have been described in hypothermia,1 after separation from cardiopulmonary bypass with cold potassium cardioplegia,6 after subarachnoid hemorrhage and massive cerebral injury,2 after cerebral thrombosis with hypothermia and shock, and after brain death with hypothermia.4 This is the first report describing these waves after resuscitation from a cardiac arrest. These waves were transient, and they subsided earlier in the precordial than in the inferior leads.

The J-point elevation is unlikely to be caused by cardiac injury due to cardiac massage or electrical defibrillation, as this has not been described previously; however, it is possible that the changes were caused by ischemia during the arrest.

**REFERENCES**

2 Luna ABD, Coumel P, Leclercq JF. Ambulatory sudden cardiac
Utility of Angiograms in Patients with Catamenial Hemoptysis*

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Pulmonary endometriosis is the likely cause in patients with hemoptysis during menses. While we describe two cases of catamenial hemoptysis, which were localized with chest CT scanning, angiograms revealed normal appearance. We suggest that angiograms may have little value in the evaluation of patients with pulmonary endometriosis.

(Chest 1990; 98:1296-97)

CT = computed tomography

Bronchial and nonbronchial systemic arteries are now considered to be the main source of hemoptysis. But in pulmonary endometriosis, which is a rare cause of hemoptysis, we have found no reports on its angiographic findings. This report examines whether the procedure of diagnostic angiogram and therapeutic embolization is useful in pulmonary endometriosis.

CASE REPORTS

CASE 1

A 22-year-old woman (gravid 2, para 0) was admitted with recurrent hemoptysis coincident with the onset of her menses during the previous year. Her past history was unremarkable, except for two artificial abortions. She had expectorated one half cup of blood per day and hemoptysis occurred only during the first three days of menses each month. Her menses occurred every 25 days, lasting five days. She did not have any symptoms. The physical examination, including gynecologic examination, was normal. At the time of admission, a chest roentgenogram showed a faint infiltrate of the left lingula. Fiberoptic bronchoscopy revealed no endobronchial lesion, and bleeding was seen originating from the left lingular lobe bronchus. Chest CT scanning revealed an ill-defined opacity with a cystic lesion in the left lingular lobe (Fig 1). Bronchial (Fig 2) and pulmonary arteriograms performed on the third day of her menses revealed no abnormalities. Since the clinical diagnosis of pulmonary endometriosis was presumed, she received hormonal therapy with danazol (600 mg/day) for two months and had no recurrent hemoptysis. One month after the cessation of therapy, she had recurrent hemoptysis.

CASE 2

A 28-year-old woman (gravid 2, para 0) was admitted with intermittent hemoptysis during the previous three months coincident with the onset of her menses. Her past history was unremarkable, except for two artificial abortions. She did not have any symptoms. Gynecologic examination revealed no abnormalities. During hemoptysis, a chest roentgenogram remained normal and fiberoptic bronchoscopy revealed blood originating from the left upper division bronchus. Chest CT scanning revealed an ill-defined opacity 1.5 x 0.5 cm in size, which was presumed to be secondary to hemorrhage, in the left upper lobe. Angiography revealed no abnormalities in the bronchial, nonbronchial systemic and pulmonary arteries. Since the clinical diagnosis of pulmonary endometriosis was made, we advised her to begin therapy with danazol, but she refused because she wanted to become pregnant. At a six-month follow-up, she had had recurrent hemoptysis during menses.

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Angiograms and Catamenial Hemoptysis (Katoh et al)