embolization into the pulmonary circulation, as seen on radiologic examination of the chest. She recovered and survived for ten years, dying later of an intercurrent and unrelated infection.1 Conrad et al5 also reported a similar case, with recovery after a week of acute illness. Studies by Schultz and Beskind6 and by Buxton et al7 have also described cases of embolization of mercury when the metal was used as an anaerobic seal and mixing agent in syringes used in collection of blood for measurement of levels of oxygen and carbon dioxide. In the series of Buxton et al,8 of a total of 1,063 cardiac catheterizations and blood gas analyses, nine cases of accidental embolization occurred.

The clinical syndrome of embolization of metallic mercury varies, depending presumably upon the quantity of mercury injected and the number of vessels occluded in the various involved organs. Embolization to the lungs is seen roentgenographically in most cases; and in some, including our patient, metallic mercury passes through the pulmonary vascular bed into the systemic circulation. Conrad et al5 and Buxton et al7 believe that the passage of metallic mercury from the right to the left side of heart occurs in the lungs through precapillary shunts or directly through the pulmonary capillaries. The magnitude of the symptoms vary from none, as in our case, to fatal involvement of multiple organs. The acute manifestations reported in the literature are nausea, vomiting, anorexia, bloody diarrhea, tremors, muscular weakness, and apprehension. Signs include stomatitis, tender and swollen gums, peripheral neuritis, and psychologic disturbances.

Few cases of pulmonary embolization of metallic mercury have been reported in which pulmonary function has been studied. The patient of Conrad et al5 was dyspneic and showed a decreased vital capacity (VC) and maximum breathing capacity on the fifth day of hospitalization. These values improved in studies performed six weeks later, although the mercury could still be seen on the x-ray film. In the report by Celli and Khan,4 their patient who received mercury intravenously to increase his athletic prowess was also dyspneic and demonstrated mild hypoxemia on admission; when first studied two weeks after admission, their patient showed a decrease in VC and diffusing capacity but no obstruction of the airways. By 20 weeks, both of these measurements had improved but were still abnormal. Our patient was not dyspneic and revealed no abnormalities in any of the multiple measurements performed one week following the injection and none 12 months later. Thus, it appears that in the dyspneic patient, a reversible restrictive pattern is present, and in the non-dyspneic patient, no physiologic disturbances are present.

The fate of injected metallic mercury has only been speculative. Follow-up of many of these cases has shown the gradual disappearance of metallic mercury from the tissues on radiographic observations. Buxton et al8 believe that slow ionization of metallic mercury occurs through biologic oxidation, forming mercuric salts which are largely excreted via the colon, kidneys, and salivary glands. This is believed to account for the symptoms of metallic mercurialism;8 however, actual chemical analysis of mercury and its salts in urine, feces, and saliva of the reported cases has not been performed. Although we attempted to perform such measurements on our patient, it was unfortunately impossible, due to his continuing severe psychiatric problems.

We suggest that the lack of symptoms and physiologic abnormalities in our patient over an 18-month follow-up period may reflect the fact that either (1) the total amount of mercury injected was small or (2) ionization, formation of salts, and excretion of the metal are not taking place or are occurring at sufficiently slow rates so as not to produce tissue levels of the mercury salts that are toxic and lead to symptoms or measurable biochemical abnormalities.

References


Successful Management of Massive Pulmonary Embolism Occurring during Cardiopulmonary Bypass for Mitral Valve Replacement*

Joseph C. Cleveland, M.D., F.C.C.P.; Ira M. Lebenson, M.D., F.C.C.P.; and Jack W. Friedman, M.D., F.C.C.P.

Massive pulmonary embolism occurred during cardiopulmonary bypass in a patient requiring mitral valve replacement. Successful management of this unusual problem is described.

Systemic venous thromboembolism occurring during cardiopulmonary bypass is a rarely encountered clinical entity. Previous reports have not been published in the thoracic surgical literature. Since the problem is

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potentially disastrous and fatal, the following report
describing the successful management of our case
seems appropriate.

CASE REPORT
A 65-year-old white woman was seen at Carle Clinic on
October 24, 1975, with an arterial embolus to the left leg.
She had longstanding mitral valve disease. Her entire left leg
below the knee was cold, white, painful and pulseless. Arterial
embolectomy was performed (J.C.C.). Good arterial circulation
was restored to the leg.

Cardiac catheterization was recommended after recovery
from the embolectomy (she was NYHA class 3). Abnormalities
demonstrated by complete right and left heart study on
November 5, 1975, showed severe calcification of the mitral
valve with a 19 mm Hg gradient across it and atrial fibrilla-
tion.

Mitral valve replacement was recommended and accepted.
On December 9, 1975, operation was performed via median
sternotomy incision. Initial heparin dosage was 3,000 units of
beef lung heparin per kilogram of body weight. Activated
clotting time of >600 seconds was present prior to institution
of cardiopulmonary bypass and was maintained throughout
bypass until protamine was administered. Venous return to
the pump oxygenator was accomplished by 2 wire-wound
caval cannulae connected to a Y-connector and thence to the
pump. Arterial return was by a No. 20 Fr arterial infusion
catheter through the left common femoral artery. Hypo-
thermia (30°C) was established. The left atrium was opened
with an incision parallel to the septum.

There was no thrombus in the left atrium. The mitral valve
was heavily scarred and calcified. It was excised along with
the chordae tendineae and tips of the papillary muscles.
Sutures of No. 0 white Dacron on small Teflon pledgets were
placed circumferentially around the annulus as horizontal
mattress sutures. The prosthesis chosen was a No. 25 Bjork-
Shiley model, which had been passed to the surgeon.

At this point, 25 minutes into cardiopulmonary bypass, the
perfusionist announced a sudden, severe decrease in venous
return to the pump oxygenator. The operating table was
raised and tapes around the cavae loosened but produced no
change. The possibility of retrograde aortic dissection was
considered to be the most likely problem, so the femoral can-
nula was removed and inserted in the ascending aorta. No in-
crease in venous return occurred.

Next, the vena cava cannulae were pulled back into the
right atrium. During this entire episode, blood flow of about
900-1,000 ml/min could be accomplished, but no higher
flow rate occurred because of lack of return. Cooling to lower
temperature was begun with the small flow available. The
right atrium became very distended and both caval can-
nulae were occluded by old thromboembolic material. The
cannulae were then removed from the atrium and, with
them, a small amount of this material. The venous cannulae
were cleared of the material and the venous lines flushed with
saline solution and reilled. The right atrium was vigorously
massaged to breakup the remaining thromboembolus. The
venous cannulae were reinserted into the cava and cardi-
opulmonary bypass re instituted. Venous return was normal.

Timing of the incident showed a period of ten minutes with
flow no higher than 1,000 ml/min and 3 minutes with the
pump turned off while emergency cannula clearance and re-
priming were done. The patient's temperature was 28°C to
28°C during this time. After satisfactory perfusion was re-
instituted, the sutures were placed in the sewing ring of the
prosthetic valve, the valve seated, and sutures were tied and
cut followed by left atrial closure. The patient was re-warmed
to 35°C and the heart spontaneously defibrillated. Separation
from the pump oxygenator was accomplished easily. Re-
versal of heparin with protamine and sternal closure was ac-
complished in a routine fashion. Total cardiopulmonary by-
pass time was 100 minutes including the episode described
above. Chest x-ray film taken immediately after operation is
shown in Figure 1.

In the intensive care unit, the patient was hemodynamically
stable, but was totally flaccid. She was unconscious. An
emergency EEG and neurologic consultation were obtained.
The EEG showed diffuse slowing and a sleep pattern. The
neurologist's opinion was that the patient had global brain
damage due to anoxia related to the incident described.
The patient slowly regained consciousness. She began to
move her right side after several days. After one week, her
endotracheal tube was removed, and she maintained satis-
factory spontaneous respiration. She began to recognize family
members. After ten days in the intensive care unit, she was
transferred to the rehabilitation unit because of left hemi-
paresis. Slow but steady improvement occurred, and she was
discharged home (by wheelchair) on January 24, 1976. A
chest x-ray film taken shortly before discharge is shown in
Figure 2. Her discharge medications were digoxin .125 mg
daily and warfarin (Coumadin) 2.5 mg daily.

The patient is now fully ambulatory, able to manage her
housework and capable of a satisfying lifestyle. She is NYHA
class 1. There is no neurologic deficit.

DISCUSSION
The rarity of this problem blunted its consideration
when perfusion difficulties arose. The more common
problem of retrograde aortic dissection was thought
responsible for the sudden volume loss. This is the most

Figure 1. Portable chest x-ray film taken immediately after
operation demonstrating changes related to massive embo-
lim to the right upper lobe pulmonary arterial tree.
serious problem associated with femoral cannulation and is usually fatal.\textsuperscript{1,2} Thus, the change of cannulation sites from femoral to aortic was rapidly performed. However, the blood volume was not lost, but the inferior vena cava cannula was totally blocked and occluded by thromboembolic material. The increased circulation related to cardiopulmonary bypass may well have caused this large thromboembolus to break loose from its attachment, probably in an iliac vein.

Hopefully, report of this isolated clinical occurrence will ease the management problems of the next cardiac surgery team which experiences a similar problem. However, the use of moderate hypothermia (28-30°C) appears to be the main reason the patient subsequently achieved a very gratifying result. The routine use of moderate hypothermia lowers oxygen consumption by 25 to 40 percent and thus allows a margin of safety when circulation from the pump must be temporarily decreased or stopped.\textsuperscript{3}

When a diagnosis of a blocked cannula becomes obvious, the only reasonable course is to clear it or change it, as was done here.

The decision to allow the major portion of the embolus to proceed to the distal pulmonary arterial tree was one of expediency in that it allowed us to re-establish cardiopulmonary bypass as soon as possible. The decision to leave the embolic material there was made on the basis of what appeared to be a normal PA pressure by palpation, satisfactory arterial blood gases after separation from bypass, and hemodynamic stability. The ability of the pulmonary arterial system to clear itself of emboli is once again demonstrated in this patient.\textsuperscript{4,5}

![Chest X-ray film taken 40 days after operation prior to hospital discharge](image)

**Figure 2.** Chest x-ray film taken 40 days after operation prior to hospital discharge. Complete clearing of the abnormality seen in Fig 2 is demonstrated.

**Symptomatic Osseous Sarcoidosis with Findings on Bone Scan\textsuperscript{*}\textsuperscript{8}

Harold M. Silver, M.D., F.C.C.P.;* Ali Shirkhoda, M.D.;† and David B. Simon, M.D., F.C.C.P.;‡

Twenty-one years after the onset of sarcoidosis, a 51-year-old woman experienced pain in the lower portion of her back, which proved to be the result of sarcoidosis involving the pelvis. The pelvic abnormality consisted of osteoclastic and osteoblastic lesions. A bone scan showed several other areas of increased uptake, and the diagnosis was confirmed by bone biopsy. The patient improved with treatment with steroids, but the findings on the x-ray film and the bone scan did not change. Sarcoidosis may cause obscure, but symptomatic, osseous lesions.

When a patient who had minimal sarcoidosis for more than 20 years developed pain in the back, the combination of radiographic studies, a bone scan, a needle biopsy, and ultimately open biopsy proved necessary to determine the cause as sarcoidosis. Bone scans and x-ray films, as well as the clinical sequence, were of interest; and this is believed to be the first abnormal bone scan to be reported in osseous sarcoidosis.

**Case Report**

In March 1956, a 32-year-old black woman complained of "bumps" around the nose, forehead, and eyes, which had been present for the past three years. There were no respiratory symptoms, and the chest x-ray film was said to have been normal in February 1955.

The skin revealed several firm nodules measuring 3 to 4 mm each in the borders of the nostrils. There were slightly elevated nodules infiltrating the medial canthus bilaterally. The findings from the remainder of the physical examination, in-

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