Pulmonary Edema after Direct Current Countershock*

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Five DC countershocks totaling 1280 watt/sec were given to convert atrial fibrillation to sinus rhythm in a patient with post-commissurotomy mitral incompetence. Atrial fibrillation persisted, but the patient developed pulmonary edema 15 minutes later and was successfully treated. Most of the pathophysiologic mechanisms suggested previously to explain the occurrence of pulmonary edema following cardioversion apply to patients who were converted to sinus rhythm. In our case, direct injury of myocardial cells by the electric current seems to be the most likely mechanism. This is the first instance reported of pulmonary edema following direct current shock in which the rhythm did not revert to sinus.

Several cases of pulmonary edema have been reported following the application of direct current (DC) countershock for the conversion of atrial fibrillation (AF) to sinus rhythm. Four cases were described by Resnekov and McDonald1 in a series of 140 DC cardioversions. Lown2 reported five episodes of pulmonary edema in four patients. Other cases were reported by Honey et al.,3 Palohelme,4 Turner and Towers,5 Lindsay,6 and Sutton and Tsagaris.7

In all cases, pulmonary edema followed restoration of sinus rhythm. Indeed, the hemodynamic alterations caused by the resumption of orderly atrial contraction was confirmed by some authors to contribute to the pathogenesis of this condition.8,9

This report concerns a case in which the application of DC countershock was followed by pulmonary edema without restoration of sinus rhythm.

CASE REPORT

A 39-year-old man was admitted to the hospital complaining of breathlessness on exertion and palpitation. He gave a history of longstanding mitral stenosis for which mitral valvotomy had been performed three years previously. At operation, the valve cusps were found fibrosed and thickened, and there were multiple adhesions between the chordae. However, adequate commissurotomy was achieved. A few weeks after the valvotomy, atrial fibrillation was converted to sinus rhythm by a course of quinidine sulfate. A maintenance dose of quinidine was given for one and a half years and then the drug was stopped. A few days after discontinuation of quini-

dine, the patient again developed atrial fibrillation and right hemiplegia of obvious embolic origin occurred. He improved gradually and was maintained on anticoagulants, digitalis and diuretics. His condition remained stable until two months before admission, when he noted progressively increasing shortness of breath.

On examination there was a slight residual right hemiparesis. The blood pressure was 110/70. The apical heart rate was 110/min and irregular. The jugular veins were not distended and there was no edema in the lower limbs. The apex beat was in the fifth intercostal space at the midclavicular line. A grade 2 pansystolic murmur followed a faint first heart sound. The systolic murmur was well propagated to the axilla. The second heart sound was normally split with accentuation of its pulmonary component. A short mid-diastolic murmur was best heard at the apex. There was no third or fourth heart sound. The chest was clear and the liver was enlarged one fingerbreadth below the costal margin but was not tender. The electrocardiogram showed atrial fibrillation with rapid ventricular response and left ventricular hypertrophy. The chest x-ray film showed slight pulmonary congestion and left atrial and left ventricular enlargement.

Because it was decided to convert the rhythm to sinus rhythm, 0.2 gm quinidine was given three times daily for two days in addition to the maintenance dose of digoxin. Five DC shocks of 100, 180, 250, 350 and 400 watt/sec were then given. However, as they failed to induce sinus rhythm, the procedure was abandoned after the fifth shock.

Fifteen minutes after the end of the procedure, the patient started to complain of rapidly increasing shortness of breath and orthopnea and emitted frothy sputum. Auscultation revealed coarse crepitations all over the chest. Blood pressure was 140/90 mm Hg while the pulse rate was 110/min. Chest x-ray film (Fig 1) revealed a typical butterfly opacity extending from the hilus to the lung fields with mottling shadows. The patient was given the customary treatment of pulmonary edema and relief gradually ensued. Chest x-ray examination on the following day (Fig 1) showed clear lung fields. The patient's further hospital course was uneventful and he was discharged one week later.

DISCUSSION

The mechanism of production of pulmonary edema following DC countershock is not clear. Several pathophysiologic mechanisms can be suggested.

The electric current may have a deleterious effect on the myocardial cells. The effect may be more pronounced in the left ventricle due to presence of underlying pathology, e.g., mitral incompetence, cardiomyopathy, or myocardial ischemia. The electric current may also directly injure the endothelial cells lining the pulmonary capillaries. Thus, it may increase the permeability of the pulmonary capillary bed and facilitate the production of pulmonary edema at a lower left atrial pressure than would normally occur.

Damage to myocardium by direct current discharge has been shown to occur in animals.10,11 In humans the evidence is only presumptive and is based on serial changes of the electrocardiogram, elevation of serum enzymes, and clinical evidence of heart failure. ST elevation (current of injury) and/or T wave changes were reported in 12 cases by Oram and Davies,11 in seven cases by Szekely et al12 and in four cases by Turner and Towers.13 Individual cases were also reported by

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 Killip, Sussman et al, and Morris et al. These changes were transient lasting from a few seconds to two minutes. Elevation of serum enzymes after countershock was commonly reported (Resnekov and McDonald and Slodki et al). It is not known whether the enzymes were released from the myocardium or from other tissues traversed by the current, such as skeletal muscle and lungs. Clinical evidences of depression of the myocardial contractility after countershock (third heart sound, hypotension, shock, increase in cardiac size) were reported by Resnekov and McDonald and by Turner and Towers. ST and T wave changes were never reported after DC shock in patients who developed pulmonary edema, but elevation of serum enzymes was reported in a case by Resnekov and McDonald. If pulmonary edema is the result of the effect of electric current on cells, one would expect its incidence to depend on the dose of electricity used for cardioversion. Resnekov and McDonald noted that five out of seven patients who developed pulmonary edema or increase in cardiac size required energy levels between 300 and 400 watts/second, an energy that is greater than that needed to convert patients who did not develop these complications. In the present case, a total of 1280 watts/second was given. On the other hand, in several cases reported, a much larger energy was used without the occurrence of pulmonary edema. Thus, Kong and Proudft gave 140 DC shocks of between 80 and 200 watt/second each to a patient with recurrent ventricular fibrillation over a period of 69 hours without apparent damage to the heart. Marriott and Sandler reported a case in which 34 DC countershocks totaling 7500 watt/sec were applied during a period of 16 hours, in which no evidence of myocardial damage was found at autopsy. However, biochemical and electrophysiologic damage, not detectable histologically, cannot be excluded. In addition, cases of pulmonary edema were reported following the use of a small energy of 80 watt/sec (Honey et al). Logan et al and Rowland et al showed that following DC countershock, the right atrial contraction may be restored while that of the left atrium is delayed.

They pointed out that the output of right ventricle may temporarily exceed that of the left ventricle resulting in accumulation of blood in the pulmonary circulation and pulmonary edema. This explanation, although possibly valid for the previously reported cases, can be ruled out in the present case because sinus rhythm was not regained.

Pulmonary embolism was suggested by Lown as a possible cause for the development of pulmonary edema in four of his patients. Pulmonary edema and pulmonary embolism were found at autopsy in one patient that died after DC countershock (Navab and La Due). In the present case there were no clinical, radiographic or electrocardiographic signs of pulmonary embolism and follow-up did not show evidence of pulmonary infarction. No clinical or pathologic evidence of the occurrence of pulmonary embolism was described in other cases previously reported. Pulmonary embolism is more likely to occur after resumption of sinus rhythm since atrial contraction may dislodge parts of right atrial thrombi that may have formed during the presence of atrial fibrillation.

Prolonged recumbency during and following the application of DC shock has been suggested as a cause for pulmonary edema. In our case the patient was not orthopneic before the procedure and used to sleep all night on one pillow only. The whole period of recumbency that preceded, accompanied and followed attempt at cardioversion did not exceed one hour. In view of the patient's past tolerance to longer recumbency, it seems unlikely that this relatively short period would lead, unaided, to pulmonary edema.

REFERENCES

We report a young woman with rheumatoid arthritis whose solitary pulmonary lesion was composed of an alveolar cell carcinoma in intimate association with an apparent rheumatoid nodule. Rheumatoid disease is thus added to a growing and diverse list of disorders wherein pulmonary fibrosis can give rise to this neoplasm. A solitary lung nodule in a patient with rheumatoid arthritis cannot be categorically regarded as benign.

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Figure 1. Close-up view of chest roentgenogram showing nodule in right upper lung.