Cardioversion and “False Positive”
Technetium-99m Stannous Pyrophosphate
Myocardial Scintigrams

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SUMMARY The present studies performed in experimental animals demonstrate that electrical direct current cardioversion can produce skeletal muscle damage and increased technetium-99m stannous pyrophosphate (Tc-PYP) uptake in experimental animals; the electrically damaged skeletal muscle shows necrosis with extensive calcium deposition. In addition, the frequent administration of high energy cardioversion produces myocardial necrosis with calcium deposition, increased Tc-PYP myocardial uptake, and a positive Tc-PYP myocardial scintigram. The data indicate that, if diagnostic Tc-PYP myocardial scintigraphy is contemplated after cardioversion, paddle placement should be slightly removed from the anteroposterior projection of the heart on the external chest wall to avoid possible subsequent confusion between increased myocardial and skeletal muscle uptake of Tc-PYP. If multiple high energy cardioversion episodes are necessary, myocardial necrosis resulting from electrical injury may occur and be responsible for increased myocardial uptake of Tc-PYP with a resultant positive Tc-PYP myocardial scintigram.

IN AN EFFORT TO ESTABLISH more precisely the occurrence of myocardial infarction in patients admitted to the hospital with a suggestive clinical history, radioisotope imaging of the heart with technetium-99m stannous pyrophosphate (Tc-PYP) is routinely performed in patients admitted to the Parkland Memorial Hospital coronary care unit. Our experience with this myocardial imaging procedure during the past two years has confirmed its sensitivity in detecting and localizing areas of recent myocardial damage. The development of a positive Tc-PYP myocardial scintigram correlates well with standard serum enzyme and electrocardiographic markers of myocardial infarction.

In view of recent published clinical and experimental studies demonstrating both chest wall and myocardial muscle necrosis following transthoracic direct current (DC) countershock, we felt it important to determine the effect of cardioversion might have on the interpretation of a subsequent Tc-PYP scintigram; that is, might cardioversion cause sufficient chest wall muscle necrosis and Tc-PYP uptake to produce a false positive scintigram? Accordingly, we have performed an experimental study to determine whether skeletal and/or heart muscle takes up increased amounts of Tc-PYP after cardioversion in dogs.

Methods

Twelve dogs were studied in the experimental investigation. Each dog was anesthetized with intravenous nembutal (15 mg/kg), intubated and ventilated with a Harvard respirator. In two dogs the chest was opened through a left lateral thoracotomy and the heart exposed. In these two animals cardioversion paddles were applied directly to the heart and two consecutive direct current discharges, each of 10 watt seconds, were applied. The chest was then closed and the animals allowed to recover. In the remaining ten dogs cardioversion paddles were applied directly to the closed chest wall with one paddle positioned over the sternum and the other one over the cardiac apex. Five of the dogs received a single 200 watt second discharge, three received five con-
secutive 200 watt second discharges, and two were given ten 400 watt second discharges. Multiple discharges were separated by approximately five minutes.

In these dogs $^{99m}$Tc-PYP myocardial imaging was performed 24 hours following countershock utilizing previously published methods. At the completion of the myocardial imaging procedure, each dog was sacrificed with intravenous high dose pentobarbital; the chest was opened, and the heart removed. In the two dogs that had received ten 400 watt second discharges, $^{99m}$Tc-PYP myocardial imaging was repeated in the isolated hearts after the heart was removed from the dog's chest. In each of the dogs studied, myocardial tissue was removed from the right ventricle and the anterior and posterior regions of the left ventricle for histologic evaluation and adjacent samples were obtained for well scintillation counting for $^{99m}$Tc-PYP activity. The
Results

In the ten dogs that received countershock across the closed chest, there was marked \(^{99}\text{Tc}\)-PYP uptake in the skeletal muscle underlying the site of paddle placement and a positive \(^{99}\text{Tc}\)-PYP scintigram in the same region (fig. 1). Histological examination of skeletal muscle from the areas of increased \(^{99}\text{Tc}\)-PYP uptake demonstrated variable degrees of necrosis and calcium deposition which correlated with the level of increased \(^{99}\text{Tc}\)-PYP uptake (fig. 2). In the two dogs that received 10 watt second discharges twice directly to the epicardium, there was no increased myocardial \(^{99}\text{Tc}\)-PYP uptake by scintigram or by well counting and myocardial histology was normal. Similarly, in eight of the ten dogs that received transthoracic countershock, there was no increased myocardial uptake of \(^{99}\text{Tc}\)-PYP and no gross or microscopic myocardial lesions were found. However, two of these dogs demonstrated increased myocardial \(^{99}\text{Tc}\)-PYP uptake by well counting and microscopically myocardial necrosis with calcification was present. One of these dogs had received 200 watt seconds five times. This animal had a small area in the anterior right ventricle with a nine-fold increase in \(^{99}\text{Tc}\)-PYP uptake by well counting, and microscopically had a well circumscribed area of subepicardial necrosis and calcium deposition (fig. 3). The other dog that demonstrated increased myocardial uptake of \(^{99}\text{Tc}\)-PYP was one of the two that received 400 watt seconds \(\times\) 10. This dog had a positive \(^{99}\text{Tc}\)-PYP myocardial scintigram; the positive \(^{99}\text{Tc}\)-PYP myocardial scintigram was confirmed by also imaging the heart after it was removed from the dog’s chest (fig. 4). This animal had increased anterior RV uptake of \(^{99}\text{Tc}\)-PYP (107 times normal) and increased \(^{99}\text{Tc}\)-PYP uptake in the anterior LV (166 times normal). In both of these myocardial regions microscopic examination demonstrated extensive myocardial cellular necrosis and calcium deposition (fig. 3).

Discussion

Since the introduction by Lown and co-workers of DC cardioversion for the treatment of cardiac arrhythmias,\(^\text{16}\) physicians have been concerned that the application of electrical current across the chest might result in tissue injury. Turner and Towers in 1965 listed electrical damage to the heart and chest wall as a potential complication of cardioversion.\(^\text{9}\) These investigators suggested that cardioversion might produce heart and/or chest wall damage on the basis of the fact that transient ST-segment ECG changes and elevation of the serum SGOT occurred in some instances after cardioversion. Subsequent studies demonstrated that transient rises in serum enzymes commonly occur after cardioversion in both humans\(^\text{15}\) and experimental animals, and

![Figure 3](http://circ.ahajournals.org/)

**Figure 3.** Myocardial damage produced in dogs by multiple electrical discharges to the chest wall. **A** and **B**. Area of anterior right ventricle with nine times normal \(^{99}\text{Tc}\)-PYP uptake from a dog given five 200 watt second discharges shows subepicardial foci of necrotic muscle cells containing calcium deposits. **C** and **D**. Area of anterior right ventricle with 107 times normal \(^{99}\text{Tc}\)-PYP uptake from a dog given ten 400 watt second discharges shows massive myocardial necrosis and calcification. **A** and **C**, hematoxylin and eosin stains; **B** and **D**, von Kossa stains for calcium salts; all \(\times 132\).
recent CPK isoenzyme studies have indicated that the rise may be either in the myocardial (MB) or skeletal muscle (MM) fraction. In addition, marked necrosis of pectoralis muscle has been demonstrated at autopsy in patients subjected to the repeated use of transthoracic cardioversion. Furthermore, subepicardial and occasionally full thickness myocardial necrosis has been described in dogs receiving multiple DC countershocks — the amount of necrosis being directly related to the intensity of the shock and inversely to the time interval between shocks and to paddle size.

Realizing that skeletal muscle and myocardial injury may occur during cardioversion, and that the myocardial imaging agent \(^{99m}\)Tc-PYP is taken up in increased amounts by necrotic muscle cells containing increased calcium, we felt that it was important to define the effect of cardioversion on \(^{99m}\)Tc-PYP scintigraphy. The data obtained in these studies demonstrate that skeletal muscle necrosis and increased \(^{99m}\)Tc-PYP uptake occur in experimental animals after relatively little cardioversion energy has been applied to the chest wall and that myocardial necrosis with increased \(^{99m}\)Tc-PYP uptake by the damaged area of the heart occurs after relatively large amounts of electrical energy have been administered with cardioversion. Recently we also noticed gross evidence of skeletal muscle damage and increased \(^{99m}\)Tc-PYP uptake at the site of paddle placement after cardioversion in one patient admitted to our coronary care unit (fig. 5). This patient was cardioverted to convert ventricular tachycardia to sinus rhythm but the amount of electrical energy given was not recorded. Whether or not cardioversion produces myocardial necrosis and increased myocardial \(^{99m}\)Tc-PYP uptake and a positive \(^{99m}\)Tc-PYP myocardial scintigram in man is uncertain presently but the data obtained from the experimental animals suggest that if considerable electrical energy is applied over a short time period myocardial necrosis and increased \(^{99m}\)Tc-PYP uptake after cardioversion might also occur in man. This is a subject that will need additional prospective evaluation.

Thus, the results of the present study indicate that DC cardioversion produces necrosis of chest wall muscle and increased \(^{99m}\)Tc-PYP uptake, and at high energy levels, myocardial necrosis and a positive \(^{99m}\)Tc-PYP myocardial scintigram in dogs. The clinical use of cardioversion may also cause local necrosis and increased \(^{99m}\)Tc-PYP uptake in chest wall skeletal muscle, interfering in some instances with the proper interpretation of a subsequent \(^{99m}\)Tc-PYP myocardial scintigram, thus making the diagnosis of acute myocardial infarction difficult to establish scintigraphically in some patients.

Therefore, if \(^{99m}\)Tc-PYP myocardial scintigraphy is contemplated in patients as part of the diagnostic determination of whether a patient has had an acute myocardial infarction, the paddles during cardioversion should be placed away from the direct anteroposterior projection of the heart on the external chest wall. This should help to eliminate confusion regarding increased skeletal muscle versus increased myo-

![Figure 4. A \(^{99m}\)Tc-PYP scintigram obtained from one of the dogs that received 400 watt seconds ten times across a closed chest wall. Panels A, B, and C are anterior, left anterior oblique and left lateral views, respectively. Panel D represents myocardial imaging of the isolated heart after it had been removed from the dog's chest. Note the marked increase in \(^{99m}\)Tc-PYP uptake in skeletal muscle anteriorly and posteriorly (Panels A, B, and C) and also in the heart (Panels C and D).](http://circ.ahajournals.org/doi/abs/10.1161/01.CIR.54.3.402)
cardiac uptake of \( ^{99m} \text{Tc-PYP} \). Proper paddle placement should help to permit unequivocal interpretation of a positive \( ^{99m} \text{Tc-PYP} \) scintigram when definite myocardial necrosis is present.

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References


Figure 5. A \( ^{99m} \text{Tc-PYP} \) myocardial scintigram obtained from a patient cardioverted in our coronary care unit. Note the marked increase in skeletal muscle uptake high over the right precordium (Panel A), but when the patient is rotated it is difficult to be certain that there is any definite increased myocardial uptake of \( ^{99m} \text{Tc-PYP} \).
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