Serum Glutamic Oxaloacetic and Pyruvic Transaminases in a Case of Cardiac Arrest Resuscitated by External Cardiac Massage

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During the course of a study of the effect of angiodynamics on the serum glutamic oxaloacetic transaminase (SGO-T), a unique opportunity developed for observing the effect on serum transaminases of closed-chest cardiac massage in cardiac arrest. A patient, in whom five control blood samples had been obtained, developed cardiac arrest and was resuscitated by external cardiac massage.

Case Report

The patient, a 39-year-old man, entered the hospital for consideration of cardiac surgery. He had rheumatic heart disease with predominant mitral and mild aortic valve involvement and was receiving digitalis. No history of angina pectoris was elicited. The admission electrocardiogram showed atrial fibrillation with nonspecific ST-T changes consistent with digitalis effect.

Left ventricular angiodynamics by the percutaneous retrograde femoral technic was attempted to define the ventricular-aortic gradient and the contour of the aortic and mitral valves. The patient was given prophylactic procaine penicillin parenterally; premedication consisted of 100 mg. of secobarbital. The femoral artery was punctured with minimal trauma and a polyethylene catheter (PE 240) was advanced easily into the left ventricle. Marked ventricular irritability immediately developed, and seconds later was followed by ventricular tachycardia. Despite prompt withdrawal of the catheter, no contrast agent having been given, marked cyanosis and hypotension ensued within 2 minutes. Unfortunately, electrocardiography and pressure recording were interrupted momentarily. A brief period of cardiac asystole (15 to 20 seconds) was followed by a period of bradycardia (20 to 30 beats per minute) for 5 minutes. Effective external cardiac massage, as denoted by an aortic pressure of 120 over 40 mm. Hg, was achieved and, combined with mouth-to-mouth artificial respiration, resulted in the patient’s recovery in 7 to 10 minutes. The procedure was discontinued and his subsequent course was uneventful. Serial electrocardiograms showed no change. He had no fever, leukocytosis, or elevation of the erythrocyte sedimentation rate. No electrical stimulation or drugs were used during the resuscitation.

Five control blood samples covering a 48-hour period preceding the procedure had been obtained from this patient as part of a study of the effect of angiodynamics on SGO-T. In addition to a sample immediately following resuscitation, specimens were taken at 8, 12, 24, and 32 hours from the beginning of the procedure and thereafter daily for a total of 6 days. Transaminase determinations were made as previously described. The normal activity range for SGO-T was 9 to 42 units and for serum glutamic pyruvic transaminase (SGP-T) 5 to 35 units. Results are shown in figure 1.

Discussion

A marked rise of SGO-T and a modest elevation of SGP-T appeared immediately after resuscitation. The highest observed levels (SGO-T, 181; SGP-T, 59) occurred 8 hours following the procedure with declining abnormal values over the subsequent 3 to 4 days.

Several factors may have accounted for the elevation of these enzymes:

1. Acute myocardial infarction. This possibility cannot be excluded but appears remote, since there was no history of angina pectoris, no change in serial electrocardiograms, no fever, leukocytosis, or elevation of the erythrocyte sedimentation rate. Myocardial infarction with enzyme elevation of this magnitude without clinical substantiation would be unusual.

2. Catheterization of the heart. Cardiac

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catheterization may be associated with elevation of SGO-T activity. However, elevations of the degree observed in this patient have not been reported.

3. Drugs. No agents known to cause SGO-T elevation were given to the patient.

4. Hypotension. Hypotension which has been reported to elevate SGO-T was present for 2 to 3 minutes. We are not certain how much, if any, of the enzyme elevation can be attributed to hypotension of such short duration.

5. Trauma to liver. Subcapsular hematoma and laceration of the liver have resulted from external cardiac massage. Were the enzyme elevation due to hepatic trauma, a predominant rise of SGP-T should have resulted. This was not observed.

6. Trauma to skeletal muscle. Although no rib fractures were sustained as a result of external cardiac massage, there was unquestionable trauma to the muscles of the chest wall. Since skeletal muscle trauma causes enzyme elevation, this factor is probably significant.

7. Contusion of heart muscle. Contusion of the heart could theoretically occur as a consequence of external cardiac massage although it has not been reported. Such trauma could elevate the transaminase levels.

The relative importance of each of these factors cannot be determined. We believe skeletal muscle trauma played a major role in this instance.

**Summary**

Serial serum transaminase determinations were made before and following cardiac arrest encountered during retrograde left ventricular catheterization and successfully managed by external cardiac massage. Prompt, significant elevation of serum glutamic oxaloacetic transaminase and slight rise in serum pyruvic transaminase occurred without evidence of myocardial infarction. Skeletal muscle trauma appeared the most likely cause.

**References**


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Giovanni Battista Morgagni, the Founder of Pathologic Anatomy

In 1707 we find Morgagni in a scientific pilgrimage through the major cultural centers of the Republic of Venice. In Venice he became closely associated with Gian Girolamo Zanichelli, a chemist and naturalist of good reputation, and with Giovanni Domenico Santorini, a distinguished anatomist of the Ospedale dei SS. Giovanni e Paolo.

From overwork, Morgagni’s health failed him for a time and he returned to his native town for a period of rest in 1709. Here, he took up the practice of medicine, and this new activity had much to do in directing his thoughts toward the correlation of the symptoms of common diseases with the underlying organic change.

It was not long before Morgagni made his return to the stage of academic life. The death of Professor Guglielmini had created a vacancy in one of the two chairs of theoretical medicine at the University of Padua. Professor Vallisneri was promoted to the first chair, and Morgagni was offered the second chair, apparently on the instigation of Lancisi, the arbiter and most celebrated professor of the time. On October 8, 1711, Morgagni accepted the appointment.

The two main medical departments at the Medical School were those of practical medicine and of theoretical medicine. In 1714, with the death of Bernardino Ramazzini (1633-1714), the founder of occupational medicine, the Chair of Practical Medicine remained vacant. Although Morgagni was attracted to this position, Lancisi, his wise protector, advised him to wait for the Chair of Anatomy that, in his own words, “was more appropriate to him and carried higher prestige.” One year later, with the death of Michelangelo Molinetti, this chair became vacant, and, on October 5, 1715, Morgagni was appointed ad anatomen ordinarium—the most important position in the School, at the annual salary of 2,200 golden zecchini. At the time of the appointment, Morgagni was thirty-three years old.

The Chair of Anatomy was held in the highest esteem. Actually, the teaching embraced both physiology and pathology, and the anatomy lecture was the synthesis of all practical and theoretical knowledge available at the time.

The teaching of anatomy that Professor Molinetti, the predecessor at the Chair, had limited to a few hours had been extended by Morgagni to include a full semester of seventy lectures that he delivered himself, year in and year out, to the end of his days, with the same enthusiasm, industry, and perseverance that characterized all his endeavors. In the last year of his life Morgagni was writing to Sénac:

I am absorbed completely by the education of this youth entrusted to me by this most magnificent Republic, and to whom I devote humbly my effort.

This effort was fully appreciated by Morgagni’s admirers and pupils who, on leaving the School (ateneo), carried to far lands not only the inspiration of a true intellectual message but also the treasure of a new investigative method.—C. G. Tedeschi, M.D. Giovanni Battista Morgagni, The Founder of Pathologic Anatomy; A Biographic Sketch On the Occasion of the 200th Anniversary Of The Publication Of His “De sedibus et causis morborum per anatomen indagatis.” The Boston Medical Quarterly 12:114, 1961.
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