CONCERNING THE MECHANISM PRODUCING AN ADRENALIN MYOCARDITIS

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Spinal shock expresses itself, as is well known, by severe reflex suppression mainly below the level of sectioning of the spinal cord. This permits the use of spinal section as a method for the temporary exclusion of nerve centers in the distal portion.

Our previous studies [1] have shown that in rats whose spinal cord had been severed at the C₄ - T₄ level and who had thus had induced in them spinal shock, injections of adrenalin during this period of shock will either not induce the development of myocarditis at all or, at most, induce it in only a light form. This permits the supposition that the inflammatory myocardial process is suppressed in these experiments because the spinal centers of cardiac innervation (T₄-T₃) are in the shock zone and, therefore, the reflexes associated with them are greatly hampered. Such a supposition is supported by the results of experiments with sectioning of the spinal cord at the lumbar level and with experiments in which decortication is performed: severing the lumbar cord does not prevent adrenalin myocarditis; decortication, which intensifies reflex activity, also makes the adrenalin myocarditis develop most severely. It still did not clarify the cardiac alterations accompanying these three indicated operations and did not reveal the relations between these changes and the development of adrenalin myocarditis.

The present study was undertaken in an effort to answer these questions, and we have repeated our previous studies, with some additional work and use of the ECG machine.

EXPERIMENTAL METHODS

The spinal cord was severed at the C₄ - T₄ level in 27 rats. ECG’s (the three standard and fourth chest leads) were taken before the operation and 24 and 48 hours after the operation. Then, the “myocarditis” dose of adrenalin (0.5 cc) was given intramuscularly and the ECG was repeated (40 minutes and 24 hours after the injection).

EXPERIMENTAL RESULTS

Of the 27 rats operated, 2 died within two days of the operation, 11 perished shortly after the adrenalin injection, and the remaining 14 were sacrificed 24 hours after the injection of adrenalin. The rat hearts were studied microscopically (frozen sections stained with Sudan III and hematoxylin). In 7 rats no signs of myocarditis could be found, in 6 rats the myocarditis appeared to be quite mild, and only in one rat was it marked. In the control material, the same dose of adrenalin given to 52 healthy rats induced myocarditis in 46 rats.

In our previous experiments (21 operated rats) myocarditis did not develop in 12 rats, and was only
mildly evident in 9. In the control experiments myocarditis developed as a rule (39 out of 43 rats receiving 0.5 cc adrenalin and 151 out of 153 receiving 0.8 cc).

This demonstrates that in two groups of experiments with sectioning of the spinal cord at level C₃ - T₁ the response with a development of a myocarditis to an injection of adrenalin was almost eliminated.

ECG's taken 24 and 48 hours after the operation (before injecting the adrenalin) showed considerable abnormalities. All rats had bradycardia of varying degree, and some had arrhythmias.

The normal rat heart rate is 480-500 beats per minute, in rats the spinal cord of which had been severed at the C₅-T₁ level the pulse varied from 100 to 400 beats per minute.

The ECG changes indicated severe metabolic disturbances and conduction myocardial disturbances (lowering or elevation of R wave, S-T interval shifts in the chest and, sometimes, in the standard leads, fragmentation of the QRS complex in the chest lead, biphasic or inverted T).

It should be noted that after the indicated operation the ECG waves in the rats altered much as after injections of large doses of adrenalin.

On the ECG taken 40 minutes after the adrenalin injection, there could be noted an acceleration of the pulse by 20-60 beats per minute while in the others there could be observed an aggravation of those changes noted after the operation. Twenty four hours after the injection the pulse either returned to normal post-operative levels or else the bradycardia became more pronounced. In rats with a developing myocarditis the ECG would reveal appearance or deeper inversions of the T wave. Rats not developing a myocarditis showed no further ECG alterations (Figure).

The appearance of the bradycardia after the spinal cord sectioning above the level of cardiac innervation is evidence in our experiments of the depressed state of the sympathetic centers in this zone.

Experiment with introduction of adrenalin after operation severing the spinal cord at level C₅ - T₁, rat No. 1062 (ECG taken in chest lead IV).

a) ECG before operation. Pulse is 480 per minute, R is 0.5 mv; b) ECG 24 hours after the operation. Pulse is 260 per minute, S-T interval shift, R is 1.1 mv, T is inverted; c) ECG 24 hours after injecting 0.5 cc adrenalin. Pulse is 170 per minute. T is isoelectric. No myocarditis was seen histologically.