

CHANGES IN THE PULMONARY CIRCULATION OF RABBITS WITH FRACTURED TARSAL BONES (Roentgenologic study)

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Physicians have long been interested in the fact that diseases of the internal organs often arise in patients injured in the extremities [1-9]. However, the mechanism of such illness has been little studied.

This article presents data on the physiological mechanism of changes in the pulmonary circulation arising after trauma to the extremities of animals.

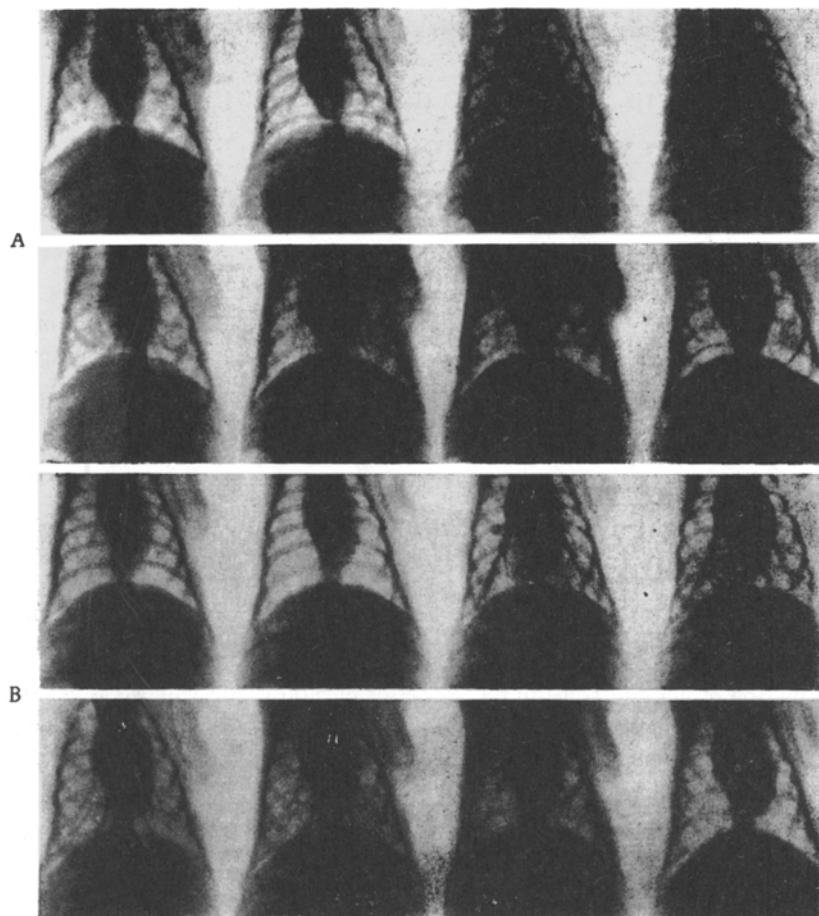
METHOD

The method of contrast angiography was used for registering changes in the circulatory rate and comparison of the width of the pulmonary vessels after various influences on the living organism. For this purpose a device for obtaining serial photographs 10 × 30 cm in size was added to the x-ray apparatus (URDD-110 k4 model 1-a). Animals were strapped in the horizontal position on the bench. Under local anesthesia (5 ml of 0.5% novocaine solution) the jugular vein was exposed and opened between two ligatures. A sterile chlorvinyl catheter full of contrast material was placed in the vein in the direction of the heart. After a survey x-ray, five ml of 35% cardiostast solution was injected and at the same time a series of eight roentgenograms was made. Angiography was performed five times in each animal: up to an at the moment of inflicting trauma, then at 2, 4 and 6 h after the trauma. In addition six rabbits were studied 24 h after the beginning of the experiment. Fractures of the tarsal bones were inflicted with an 150 gm weight lowered from a height of 30 cm. The interval between two successive administrations of cardiostast always exceeded 15-20 min, which permitted the effect of the contrast material on the blood vessels to reach a minimum.

In studying the roentgenograms we determined the time of filling of the right atrium and right ventricle with contrast material, as well as the time the blood was moving to the pulmonary artery, veins and left ventricle. Sixteen rabbits were studied in a total of 42 angiocardigrams.

RESULTS

An analysis of our data showed that in all animals after trauma the passage of contrast material from the pulmonary arteries to the pulmonary veins was prolonged by 2-3 sec, and that a delay occurred in the initiation of left ventricular filling with cardiostast (see figure). Upon measurement of the x-rays the width of the pulmonary arteries after trauma was in all cases detected to be greater than the main trunks of the pulmonary arteries by 1-2 mm. While the width of the opening of the small branches of the pulmonary arteries (IV-Vth order) appeared 0.5-1 mm smaller than before trauma, their filling with cardiostast was slower by 2-3 sec. The aperture of the pulmonary veins was unchanged. It must be emphasized that in most of the rabbits after trauma the cupola of the diaphragms was noted to be high. Delay in contrast material in the pulmonary artery system upon repeat examination 2, 4 and 6 h after trauma was not so clearly expressed as at the time the trauma was inflicted; however, on review of the x-rays and angiocardigrams, small and confluent areas of hemorrhage and pulmonary edema, increases in the pulmonary vascular markings and widening of the fine branches of the pulmonary veins were observed. Twenty-four hours after the beginning of the experiment, congestive phenomena in the lesser circulation were observed in 2 out of 6 animals. Pulmonary edema at this must be considered on the basis of the x-rays, disappeared after 8-10 days.



Angiocardiograms of rabbit No. 14, before (A) and after (B) fracture of the tarsal bones (read from left to right).

To elucidate the mechanisms which govern the disturbance of the pulmonary circulation, the tarsal fractures were performed in 10 animals after intramuscular administration of 0.5 ml of morphine, simultaneously giving careful novocaine infiltration of the soft tissues at the trauma site. In all ten cases with anesthesia we were unsuccessful in recording a post-traumatic slowing of the lesser circulation and congestion in the lung parenchyma was absent. Diaphragmatic tone did not change. Only in 3 animals on the post-trauma pulmonary angiogram did the network of small arteries and veins appear more prominent because of a certain amount of congestion.

Evidently, fracture of the tarsal bones of the hindlegs of animals, evoking intense pain, reflexively evokes changes in the pulmonary circulation. These are characterized by retardation of the blood flow and changes in pulmonary vascular tone.

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