SCIENTIFIC PROCEEDINGS.

ABSTRACTS OF THE COMMUNICATIONS.

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1 (257)

Cardiac insufficiency due to high arterial pressure.

By **HAVEN EMERSON.**

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Prof. Leo Loeb, of the University of Pennsylvania, first called my attention to the method of teaching the phenomena of pulmonary edema by administering massive doses of adrenalin to rabbits. His observations I repeated with similar, although not such severe results, in June, 1906, in one instance following the signs of mitral regurgitation and acute dilation of the heart through to a gradual recovery, and finally complete reestablishment of the normal heart sounds and heart rate in a rabbit.

During the winter of 1906–7 in the course of demonstrating the effects of direct and reflex vaso-motor phenomena to students, I had occasion to observe the effect of massive doses of adrenalin in cats, in which the carotid blood pressure and the intrapleural pressure variations were being recorded on a kymograph. The familiar phenomena of blood pressure, raised and maintained to 250–300 mm. of mercury, with a rapid respiration were observed, the usual slowing of the heart not appearing in these extreme conditions. In every instance there appeared shortly, small and then large moist rales over the entire chest, which gradually increased until the tracheotomy tube became flooded with serous froth. Often the systolic regurgitant murmur and the irregular rapid heart sounds were noticed preceding the edema.

If it had not been for a further observation I should not have ventured to bring this to your attention, but during June, 1907, I had occasion again to demonstrate the physical signs and physiological records in cats under heavy doses of adrenalin. To provide a more complete picture of the cardiac and pulmonary changes I used a cat under ether with the anterior chest wall removed and artificial respiration established. This cat, like another on an adjoining table, under normal respiration, showed the high blood pressure, and presently the moisture accumulating in the trachea. The heart in the cat with the chest opened presented the dilated right side of the heart, its deep venous hue and regurgitant murmur as usual, but presently these signs improved and the lungs from appearing deeply congested and sodden, became clear and pink, the serous exudate no longer obstructed the trachea, and the heart resumed its normal size, color, and rate. In the meantime the condition in the other cat was one of progressing edema.

In spite of the fact that cats, as well as rabbits, vary in their susceptibility to commercial adrenalin solutions, the changes in the picture above described, appeared too definite and too prompt to be due to any individual recuperative power or insusceptibility. I believe the change in the cat with the chest opened was due to the conditions of artificial respiration. Air was being forced into the lungs under pressure to distend them against their inherent elasticity and atmospheric pressure. Thus the pulmonary vessels may be considered as being subject to distinct positive pressure from without, recurring rhythmically with artificial respiration. It seems to me conceivable that this may have assisted the passage of blood from the right to the left heart, and in this way supplemented the right ventricle.

The effect of adrenalin in causing edema is not necessarily to be concluded a sufficient reason for avoiding its use therapeutically for relief from edema, since edema which has its origin in vascular relaxation, the vaso-paresis which occurs in the toxemia of pneumonia and diphtheria infections, as shown by Romburg and Pässler, is an entirely different clinical picture from the edema resulting from contracted arteries and failing heart, such as occurs in nephritics and severe arteriosclerotics.

In one instance adrenalin will improve the circulation by sub-

stituting its constricting action upon the blood vessels for the lack of vascular tone, and, as has been often observed clinically, will materially improve the existing pulmonary edema, while in the second instance, it would but aggravate the condition.

Although my observations are too few to base any general conclusions upon, I think they suggest that artificial respiration may be profitably tried in some cases of right heart failure, due to increased peripheral resistance, as soon as the mechanics of administering air can be perfected so as to avoid intubation or tracheotomy. And in this regard I may say that I have found that a person who is conscious can readily be supplied with air by artificial respiration by the use of an ordinary ether inhalation mask of the Bennett or Clover type. Whether this method will prove possible, in unconscious states or where there is general relaxation, as under anesthesia, I cannot say, but I think it is quite possible.

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Effect of potassium cyanide upon metabolism.

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Potassium cyanide was administered to two dogs in amounts sufficient to produce severe poisoning. One animal was kept upon a uniform, analyzed diet throughout the experiment (9 days) and the other was given no food during the experiment (5 days). In both animals an increase in the total nitrogen of the urine occurred on the day of poisoning which continued during the next following day. This increase was caused mainly by an increase in the amount of urea and by slight increase in the amounts of ammonia, total creatinin (sum of creatin and creatinin) and undetermined nitrogen (purin bases, uric acid, allantoin, amino-acids etc.). Neither experiment gave indication of marked percentage increase either of ammonia or of undetermined nitrogen, for the distribution of the nitrogen remained practically normal. Well marked absolute and percentage increase of creatin, and decrease of creatinin, was observed. Creatin was eliminated by the fasting dog throughout the whole period of observation.