

4352

Arterial Blood Pressure and Blood Flow in Skeletal Muscles as Influenced by Epinephrine.

CHARLES M. GRUBER.

From the Department of Pharmacology, Washington University School of Medicine, St. Louis, Mo.

In this research unanesthetized cats were used. The spinal cord was cut in each case in the thoracic region. All operative work was done under general ether anesthesia after which the anesthetic was discontinued for 5 to 24 hours before the injection of epinephrine. In these animals dilute solutions of epinephrine in small amounts injected in the same way caused in some animals a fall, in others no change, and in still others a rise in blood pressure.

Small doses of epinephrine invariably caused an increase in the rate of blood flow in skeletal muscles irrespective of the change in blood pressure. Large doses of epinephrine rapidly injected intravenously caused an increase in blood pressure accompanied by a decrease in rate of blood flow in skeletal muscle. Following the rise the blood pressure fell below the normal level and simultaneously with it a marked increase in the rate of blood flow through the muscles was observed.

These results upon unanesthetized cats confirm the findings in anesthetized and decerebrated ones—in that epinephrine produces a dilatation of the blood vessels in one region of the body at the same time that it causes vaso-constriction in other regions, and the blood pressure recorded is the stronger effect minus the weaker.

4353

Effect of Infections on the Thyroid Gland.

J. RABINOVITCH AND S. H. GRAY. (Introduced by Leo Loeb.)

From the Department of Pathology, Washington University School of Medicine, St. Louis, Mo.

Cole, Womack and Gray¹ and others^{2, 3} described certain pathologic changes in thyroids of persons who suffered from acute or

¹ Cole, W. H., Womack, N. A., and Gray, S. H., to be published, *Am. J. Surg.*

² Cole, W. H., and Womack, N. A., *J. Am. Med. Assn.*, 1928, xc, 1274.

³ See literature in references 1 and 2.

chronic infections, such as pneumonia or peritonitis. The glands were obtained at autopsy from individuals who succumbed to either an acute or chronic infection. The changes noted in the thyroid were of a two-fold nature, depending in each case on the acuteness or chronicity of the infection from which the patient died. Thus, where death was a result of pneumonia or acute peritonitis, the changes in the thyroid consisted mainly in desquamation of the epithelium lining the acini and softening of the colloid. When death of the individual was due to a more chronic type of infection, the thyroid did not show any epithelial desquamation, but rather a heaping up of the acinar cells with papillary projections into the alveoli, and increased scar formation. Very similar changes to those described above, were previously noted by Cole and Womack² in dogs in whom they had experimentally produced acute infections and toxemias.

We thought it of interest to carry out similar experiments in guinea pigs, because the thyroid gland of guinea pigs has been very carefully studied in the laboratory under normal as well as under various experimental conditions and we are therefore well acquainted with the changes which take place in the thyroid of guinea pigs under various conditions.

We used 26 guinea pigs, in 16 of which an acute infection was produced by injecting fecal material into the peritoneal cavity. The time of development of an acute peritonitis and the consequent death of the injected animal was found to vary according to the quantity of injected material and site of injection. If the site of injection was high up in the abdomen, viz., in the region of the diaphragm, where absorption is more rapid, the animal died much sooner than when injected in the pelvis.

Chronic peritonitis was produced in 6 guinea pigs, by injecting repeatedly small doses of fecal material intraperitoneally for periods of time ranging between 2 weeks and 4 months. Added to the series of acute infections, was one animal that died of pneumonia and another that succumbed to an acute necrosis of the subcutaneous tissue of the abdomen.

Controls consisted of thyroids removed from normal animals killed in chloroform. In a very large series, the thyroids were removed and fixed immediately after death. Four guinea pigs were returned to their cages after death and allowed to remain there at room temperature for 4, 6 and 18 hours before the thyroids were removed and fixed.

I. Control series. Thyroids removed from animals immediately after death due to inhalation of chloroform and fixed in Zenker's

solution, showed the following characteristics: The acini were of a variable size, ranging in dimension from small to intermediate and large. The lining epithelium was usually of a cuboidal type, although occasionally taller or lower than the average. The colloid was solid as a rule, but at times contained phagocytes that caused a partial or complete liquefaction and absorption of this substance. There was no evidence in these cases of any desquamation of the epithelium anywhere in the gland. In the interstitial tissue, there were occasionally lymphocytic or leucocytic infiltrations. The capillaries were sometimes congested and a moderate or even extensive increase in connective tissue was found in certain glands, particularly in the region of the larger vessels.

Glands removed from animals in which death occurred some time previously, showed some additional characteristics after death. The size of the acini and epithelium were the same as those described above. There was, however, present a definite but very slight desquamation of the epithelium, while the colloid was solid and showed a slight tendency towards softening. In animals in which the thyroids were removed 18 hours after death the desquamation of the epithelium was intense, and the colloid was somewhat softer in acini where desquamation was apparent.

II. The thyroid of infected guinea pigs. To this series belong 16 animals injected intraperitoneally with fecal matter, 10 of which were found dead in their cages; the thyroids in these instances were removed at an indefinite time following the death of the animal. The remaining six were found very acutely ill, and these were killed by chloroform and the thyroid removed and fixed immediately after their death. The thyroid of this latter series of animals displayed the following histological characteristics: 5 of these animals showed no desquamation of the acinar epithelium; the remaining one showed a definite desquamation. The size of the epithelium in every instance was cuboidal and the colloid hard, except in the animal where desquamation was noted, the colloid was soft.

In the large majority of cases the fecal matter was injected late in the afternoon. Ten of the injected animals were found dead early the next morning and were autopsied immediately. The time interval between death and autopsy could not have been more than a few hours. In 6 of the animals found dead, the thyroid acini showed marked desquamation of the epithelium; in 3 there was only a mild degree of desquamation, and in one desquamation was entirely absent. The epithelium in all these animals was cuboidal. The colloid, on the whole, was solid but there were occasional areas of

softening, particularly in the acini that showed desquamated epithelium.

III. The thyroid of guinea pigs chronically infected. In this series we had 6 guinea pigs which had received 3 to 7 doses of fecal injections over periods ranging between 3 weeks and 4 months. Four of these were killed by chloroform and 2 were found dead. Histologically, none of the thyroids removed showed any desquamation. The acini were small to medium in size; the epithelium was cuboidal and the colloid was hard. Three of these glands had a cellular appearance, probably due to the crowding of small acini and to the apparent filling of the interacinar spaces with epithelial cells. The number of mitoses, however, showed no increase above the normal. In one of these animals, scars were found in the interstitial tissue.

Discussion: Desquamation of the epithelium and softening of the colloid which had been described in the human and dog thyroids after acute infections, occurred only in one of the 6 acutely infected guinea pigs that we killed. This picture was noted however in 9 of the 10 acutely infected animals that were found dead. These changes occurred earlier and to a more marked degree in the latter group of animals than in our control animals which were examined some time after death. Whether these changes are due to specific effects of the infection upon the thyroid gland during life, or whether they were caused by postmortem autolysis which was hastened and intensified because of the presence of an infection and toxemia, we are as yet unable to determine definitely. However, the fact that only one of the 6 acutely infected animals, that we killed when very ill, showed this desquamation, seems to support the second view. It must be remembered, however, that different species of animals as well as individual animals of the same species may react differently to the same experimental procedures.

In the cases of chronic infection, we found no desquamation of the epithelium and the colloid was hard; 3 of these animals also showed very cellular glands, and one animal showed scar formation. These facts are in agreement with the findings noted in the human and dog. However, we also found similar cellular glands and scar formation in 4 of the 16 acutely infected animals. Furthermore, in going over a large number of thyroid removed from normal guinea pigs and from guinea pigs fed with various substances and in which we could find no evidence of infection, we noted a fair proportion of cases presenting this same cellular appearance as well as scar formation. The former was especially found in a series of animals

that were underfed⁴ and in which there was no increase in cell mitoses.

Summary: 1. In 22 guinea pigs an acute and chronic peritonitis was induced by injecting fecal material intraperitoneally. 2. In only one guinea pig examined before death was any desquamation of epithelium and softening of the colloid noted. In guinea pigs found dead, post mortem desquamation occurred earlier in experimental infections than in controls. 3. In chronic infections the guinea pig thyroid presented no deviations from the normal.

⁴ Rabinovitch, J., *Am. J. Path.*, 1929, v, 87.