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Pressor Reactions and Bacterial Localization in Central Nervous System.

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When we produce pressor episodes in an animal body, some region of the vascular bed must be relatively anoxemic. In such regions, particularly those in which vascular anastomoses are poor, or terminal vessels exist (skin, central nervous system, special sense organs, etc.) stimulation promptly follows, the endothelium of the capillaries becomes more permeable (Landis)[1] and we may assume that bacterial adhesion can here occur to a greater extent than under normal conditions of circulation.

In general, the capillaries of the brain and the central nervous system are less permeable than those of the splanchnic bed and consequently they are less "sticky". Bacterial localization in the brain, considering the volume of the blood flow, is relatively infrequent—abscess formation certainly not common.

We were interested in the localization of bacteria in the central nervous system and for this purpose used single as well as repeated injections of pitressin (betahypophamine) to bring about periods of transient anoxemia in some of the tissues of the central nervous system. Increased stickiness and permeability of the endothelium of the blood vessels at the foci of anoxemic stimulation might enhance the localization of bacteria here as well as elsewhere.

The throats of 15 dogs were sprayed with a suspension of *B. megatherium* and *Staph. aureus* with the purpose of tracing the passage of bacteria through intact mucous membrane to the bloodstream and ultimately to the brain. The animals were divided into 3 groups of 5 each.

1. In the first (control) group the animals were destroyed 40 minutes after spraying of throat. Microörganisms were very rarely observed in the sections of the brain.

2. In the second group, pitressin was injected intravenously 10 minutes after spraying and 40 minutes later the animals were destroyed. The number of microörganisms in sections of the brain did not exceed those found in the control group.

[1] Landis, E. M., *Am. J. Phys.*, 1927, **81**, 124.

3. In the third group, the animals had received repeated injections of pitressin at 3 and 4 day intervals. One animal received 9; one, 12; two, 17; and one, 25 injections. Ten minutes after spraying an additional intravenous injection of pitressin was given and 40 minutes later the animals were destroyed. In this third group the microörganisms were found in appreciable numbers, with the cocci forming definite groups. *B. megatherium* was not found at all in the tissues of the first 2 groups of animals, but in the third group a few single rods were found.

We were aware that the staining of bacteria in the tissues not infrequently leads to confusion and the method is open to the criticism that an object, presumably bacterial, may actually be an artifact. For this reason we gave 5 rabbits from 5 to 9 injections of pitressin intravenously at intervals of from 3 to 4 days. On the day of the last injection a suspension of *B. megatherium* and *Staphylococcus aureus,* previously stained and thoroughly washed, was injected intravenously. After one hour the animals were bled to death.

The tissues of the hippocampus major and the spinal cord were examined in frozen sections, without the use of any stain and the findings were quite similar to those of the dogs observed in the third group, namely, a considerable degree of bacterial localization in the tissues of the central nervous system.

Four normal rabbits that had received a single injection of stained bacteria, but without injection of pitressin, were treated in a similar way. No organisms were observed in the nervous tissue.

The repeated spasm of the blood-vessels of the central nervous system that follows the injection of pitressin apparently ultimately results in alterations in the tissues that can be demonstrated objectively. These changes are now being studied microscopically.

These results suggest that pressor effects may lead to the localization of bacteria in regions where vascular spasm has been particularly pronounced. The mechanism involved is probably associated with the production of focal areas of tissue (and capillary) stimulation in the regions that have been anoxemic. Here the capillaries become more permeable and adhesive, they dilate, the current is slowed and the opportunity for localization of bacteria in transit is greatly enhanced.