Tension and Distensibility of Carotid Sinus Wall, Pressoceptors and Blood Pressure Regulation

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Local carotid sinus application of noradrenaline induces a stimulation of the pressoceptors at different constant physiologic and nonphysiologic intrasinusal pressure ranges. Tension and resistance to stretch of the arterial wall where the pressoceptors are located, are the fundamental factors affecting, at different pressure ranges, those receptors which regulate and moderate reflexly the systemic arterial pressure.

Experiments showed that the state of tone and tension, and thus the distensibility and resistance to stretch of the arterial wall where the pressoceptors of the carotid sinus are located, are the primary factors affecting these pressoceptors which regulate and moderate reflexly the systemic arterial pressure.

Drugs such as adrenaline, noradrenaline, vasopressin, diacetyladrenaline thiosulfonic acid, ephedrine, synephrine and hydroxyphenylaminopropanol or Aramine, applied locally to the carotid sinus areas, induce a contraction of the arterial wall, decrease their distensibility, increase their resistance to stretch and thus increase the stimulation of the pressoceptors and provoke a progressive and prolonged reflex fall of the systemic arterial pressure.

Drugs such as papaverine, Priscoline, sodium nitrite, and potassium chloride in doses relaxing smooth muscles, applied locally to the wall of the arteries of the carotid sinus, induce the opposite reactions and cause a reflex rise of systemic arterial pressure.

The action on blood pressure of locally applied adrenaline, noradrenaline and Aramine to the carotid sinus may be reversed by local carotid sinus application of an adrenolytic drug.

Experiments of Landgren, Neil and Zotterman confirmed the observations with adrenaline, noradrenaline and Vasopressin, and also showed that local application of these drugs to the carotid sinus elicits a very definite increase in the pressoceptor impulse traffic. This increase is most conspicuous in the smaller fibers. The effect of adrenaline is prevented by the previous application of an adrenolytic drug. Sodium nitrite administered locally to the carotid sinus wall causes a marked reduction of the pressoceptor fibers activity.

Landgren further showed that at a constant intrasinusal blood pressure of 100 mm. Hg, sodium nitrite causes a dilation of the isolated carotid sinus wall of cats, while adrenaline application causes a contraction. This author concluded from his experiments performed on the isolated carotid sinus preparation in vitro of cats, that local application of adrenaline to the carotid sinus causes an increased distensibility of the arterial wall in the region of the physiologic blood pressure variations (90 to 180 mm. Hg), and this in spite of the diminution of the absolute values of the carotid sinus diameter and volume.

As compared with the control, adrenaline causes a decreased distensibility of the isolated carotid sinus wall in the pressure region below 100 mm. Hg. After local sodium nitrite application, on the other hand, the distensibility of the carotid sinus wall is increased at low pressures, but is decreased or unchanged in the region of the physiologic blood pressure variations. The small pressoceptor spikes, however, increase in frequency after local application of adrenaline on the carotid sinus at all levels.
intrasinusal pressure ranges. The local application of adrenaline to the sinus wall also renders the pressoceptors eliciting the large spikes more sensitive to blood pressure changes within the limits of the physiologic variations. If the intrasinusal pressure falls below the physiologic values, the discharge of large pressoceptor spikes ceases as well before as after adrenaline application, and there is no impulse activity of large pressoceptor spikes during the lower intrasinusal constant pressures. The small pressoceptor spikes therefore seem to be of greatest importance for the adrenaline effect on the carotid sinus wall. According to Landgren, if adrenaline, on the other hand, is assumed to exercise its effect by stimulating the smooth muscle coat in the carotid sinus wall, there must be a direct relation between the tone of the contractile elements and the discharge of small pressoceptor spikes.

These experimental observations and conclusions of Landgren incited us to perform a series of experiments in order to investigate the reflex influences on systemic blood pressure of local application of noradrenaline on the carotid sinus, in vivo, of dogs, during constant physiologic and nonphysiologic intrasinusal pressures.

**METHOD**

Dogs were anesthetized with morphine (1 mg. per kilogram, subcutaneously) and chloralosane (100 mg. per kilogram, intravenously).

The vagi-aortic nerves were cut in order to limit the reflex regulation of blood pressure to the carotid sinuses. The systemic blood pressure was registered with a mercury manometer from a femoral artery. Both carotid sinuses were prepared according to a modified technic described by Lim and Chang and Heymans, Donatelli and Shen.

The common carotid arteries were cut between ligatures. Sounds provided with a small balloon prepared from a jugular vein, were inserted into the cephalic ends of the common carotid arteries, the small balloon being introduced up to the dilatation of the carotid sinus areas. The common carotid arteries were then tied to the cannulas (fig. 1).

The balloons, the cannulas and plastic connecting tubes were filled with saline and connected with a pressure device. The intrasinusal pressure was registered.

With this method, a constant pressure may be maintained in both carotid sinuses and intrasinusal pressure variations may also be elicited. By injection of 2 to 4 ml. of 1 per 1000, in isotonic saline solutions, into the external conjunctival space surrounding both carotid sinus areas, L-noradrenaline hydrochloride was applied to the wall of the arteries of the carotid sinuses.

**EXPERIMENTS**

The vasomotor and systemic blood pressure responses to different variations and constant levels of pressure in the carotid sinuses were first registered. The experiments showed a decrease of systemic pressure in response to an increase of pressure in the carotid sinuses and a rise of systemic arterial pressure following a decrease of pressure in the carotid sinuses (I, fig. 2). At a constant pressure in the range of
physiologic levels into the carotid sinuses, the systemic arterial pressure was maintained reflexly at normal levels (II, fig. 2). Noradrenaline was then applied locally (II, fig. 2) on each carotid sinus area, while the intrasinusal pressure was maintained at constant physiologic level.

The local application of noradrenaline on the wall of the arteries of the carotid sinus induces a progressive marked reflex fall of systemic arterial pressure (II, 3, fig. 2), although the intrasinusal pressure was maintained at constant physiologic level. Furthermore, decreasing the pressure in the carotid sinuses no longer induced a reflex rise of systemic arterial pressure (III, fig. 2). About 55 minutes after the local carotid sinus application of noradrenaline, the systemic arterial pressure remained at a low level and the hypertensive reflexes normally induced by lowering intrasinusal pressure were still absent or very slight.

The systemic arterial pressure and the hypertensive reflexes returned progressively, but slowly, to normal conditions. This very pro
longed influence of locally applied noradrenaline to the wall of the carotid sinus must be related to the restricted circulation of the blood in the carotid sinus preparations.

The same experimental observations of reflex fall of systemic arterial pressure, were made after local application of noradrenaline to the carotid sinus at different intrasinusal constant pressures in the range of 120 to 180 mm. Hg, and at higher or lower intrasinusal constant pressures (fig. 3).

That the reflex fall of the systemic arterial pressure, after local carotid sinus application of noradrenaline at different constant intrasinusal pressures is due to a stimulation of the carotid sinus pressorceptors, is shown by the fact that section of the carotid sinus nerves while the systemic blood pressure is lowered, provokes an immediate very marked rise of systemic arterial pressure (IV, fig. 2).

**DISCUSSION**

These experiments show that noradrenaline, which is known to induce a contraction of the arterial wall and thus an increased tension and resistance to stretch of the arterial wall, applied to the carotid sinus areas, causes a stimulation of the receptors of the carotid sinus nerves which moderate reflexly the systemic arterial pressure. This stimulation of the carotid sinus pressorceptors, inducing a reflex fall of systemic arterial pressure and a decrease or suppression of the hypertensive reflexes normally provoked by decrease of pressure in the carotid sinus, also occurs after local carotid sinus application of noradrenaline while the intrasinusal pressure is maintained at constant physiologic or nonphysiologic levels.

These experiments thus demonstrate that the same influences of locally applied noradrenaline to the wall of the arteries of the carotid sinus occur at different physiologic or nonphysiologic ranges of intrasinusal pressure levels.

The observations of Landgren, on the isolated carotid sinus preparation in vitro of cats, that adrenaline causes a decreased distensibility of the carotid sinus wall in the pressure region below 100 mm. Hg, but an increased distensibility between 100 and 200 mm. Hg in the carotid sinus, thus do not interfere with the circulatory reactions induced by locally applied noradrenaline to the arterial wall of the carotid sinus in vivo of the dog. These circulatory reactions in dogs are identical at different intrasinusal pressure regions. In the intrasinusal pressure region below as above 100 mm. Hg, local application of noradrenaline on the arterial wall of the carotid sinus of the dog, indeed provokes identical reactions of reflex fall of systemic arterial pressure by stimulation of the carotid sinus pressorceptor mechanisms.

Why, according to Landgren, there is no impulse activity of large pressorceptor spikes during the lower intrasinusal pressures after local adrenaline application on the carotid sinus while the small pressorceptor spikes increase in frequency after local application of adrenaline at all intrasinusal pressure ranges, cannot be answered. It may be suggested however that only the pressorceptor fibers with small spikes are of importance for the reflex regulation of blood pressure, while the pressorceptor fibers with large spikes are of importance for the reflex influences on the respiratory center.

Our experiments showed, indeed, that local carotid sinus application of adrenaline or noradrenaline induces, at all intrasinusal pressure ranges, a stimulation (hyperactivity of small spikes) of the pressorceptors which reflexly moderates arterial pressure, while low intrasinusal pressure still induces a reflex hyperpnea due to a decrease of the reflex inhibition of the respiratory center by the low intrasinusal pressure (decrease of impulses of large pressorceptor spikes).

**SUMMARY**

Experiments performed on dogs show that:

1. Noradrenaline applied to the arterial wall of the carotid sinus areas induces a stimulation of the pressorceptors and thus a reflex fall of the systemic arterial pressure, and a decrease or suppression of the hypertensive reflexes normally provoked by a decrease of pressure inside the carotid sinus.

2. The same circulatory reactions induced by local carotid sinus application of noradrenaline also occur at different constant physio-
logic or nonphysiologic intrasinusal pressure ranges.

3. The tension and resistance to stretch of the arterial wall where the carotid sinus pres-
soceptors are located, are the fundamental factors affecting these receptors which regulate and moderate reflexly the systemic arterial pressure.

The significance of these experimental observations is discussed.

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