Reflex Peripheral Venoconstriction Induced by Carotid Occlusion

By Edwin W. Salzman, M.D.

The present investigation was designed to obtain additional evidence for the effect of carotid sinus reflexes on venous tone. It was found that occlusion of one or both common carotid arteries increases tension in a miniature balloon inserted into a peripheral vein. Deafferentation of the aortic arch by cervical vagotomy enhanced the venous response. The time course and magnitude of the venous reactions paralleled the increase in arterial pressure. It appears that the homeostatic increase in peripheral resistance as a result of reflex arteriolar constriction is complemented by a decrease in size of the peripheral venous reservoir.

Constriction in small peripheral veins has been described following the intravenous administration of sympathomimetic drugs and during electrical stimulation of the lumbar sympathetic chain. Reflex peripheral venoconstriction has also been noted during acceleration on the centrifuge and following tilting to the vertical.

The carotid sinus mechanism appears to be involved in the reflex control of venous tone. Charlier observed that common carotid artery occlusion was accompanied by a rise in right atrial pressure, despite the associated increase in cardiac output. Stimulation of the sinus nerve resulted in a decreased cardiac output which Holt attributed to peripheral pooling of blood consequent to venodilation. Heymans noted a decrease in caliber of veins in the mesentery accompanying induced hypotension in the carotid sinus and suggested the sinus as a principal site of origin of reflex venomotion in the splanchnic bed. This hypothesis was supported by Alexander, who interpreted variations in mesenteric venous distensibility associated with alterations in intracarotid pressure as evidence of reflex venomotion in the portal network.

It seemed likely that the peripheral veins might resemble the veins of the mesentery in their responsiveness to the carotid sinus. A previously reported miniature balloon technic appeared suitable for a demonstration of this interrelationship.

From the Aero Medical Laboratory, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio.

Received for publication October 22, 1956.

Methods

Fourteen adult mongrel dogs were studied, using chloralose anesthesia to avoid brainstem depression. A fluid filled miniature latex balloon in the lesser saphenous vein responded to changes in tone of the vein wall and to hydrostatic pressure about the balloon, which was measured directly through a catheter in a nearby collateral vein. Pressures were monitored with Statham transducers and recorded photographically. Venoconstriction was evidenced by a rise in pressure in the balloon accompanied by a lesser rise, or no rise, in pressure in the venous catheter. In 4 dogs, the pressure in an adjacent subcutaneous balloon was simultaneously recorded to detect the effect of possible changes in tissue tension on the intravenous balloon. Femoral arterial pressure was recorded in the opposite thigh.

The carotid arteries were exposed in the neck and hypotension in the carotid sinus was produced by common carotid occlusion. The sinus was de-nervated by stripping the adventitia from the sinus area and adjacent arteries. The criterion for complete denervation was failure to elicit a tachycardia and arterial hypertension by carotid occlusion on that side. Bilateral cervical vagotomy was performed in 4 dogs.

Results

Unilateral or bilateral carotid occlusion in the normal animal was followed within a few seconds by tachycardia, arterial hypertension and a rise in pressure in the intravenous balloon. Peripheral venous pressure remained constant or declined 1 to 3 mm. Hg. The pressure rise in the intravenous balloon varied between 1 and 30 mm. Hg in different experiments. Mean arterial pressure increased between 10 and 90 mm. Hg. The pressure in the subcutaneous balloon remained constant or decreased a few millimeters. With release of
Bilateral common carotid occlusion. Calibration in millimeters mercury. The spike near the middle of the intravenous balloon and venous pressure records is a movement artefact. Signals on baseline indicate the beginning and end of carotid occlusion.

Following bilateral carotid occlusion, all parameters returned gradually to the baseline level. Following bilateral carotid occlusion, the rise in arterial pressure and in pressure in the intravenous balloon was greater than after unilateral occlusion. Bilateral carotid sinus denervation eliminated the response to carotid occlusion. After unilateral denervation, the response to unilateral occlusion on that side was lost, but the effect of unilateral occlusion of the opposite side was enhanced, the net result being in the same range of magnitude as with bilateral carotid occlusion before sinus denervation. In a few cases, venoconstriction was observed following sinus denervation itself.

The record shown (fig. 1) demonstrates the effect of bilateral carotid occlusion in an otherwise intact animal. The intravenous balloon pressure rise was 29 mm. Hg; arterial pressure increased 47 mm. Hg; peripheral venous pressure was unchanged.

In most cases, venoconstriction began 2 to 5 sec. after the onset of carotid occlusion. The arterial pressure rise began a second or two earlier. This discrepancy in time lag may not reflect a true difference between arteriolar and venous constriction, since the observed change in heart rate might be expected to be more prompt than the contraction of arteriolar smooth muscle. Kelly and Visscher² have recently reported small vein end pressures and small artery pressures in the hind foot of a dog during lumbar sympathetic stimulation, presumably the same efferent pathway whereby the carotid sinus mechanism influences peripheral venoconstriction. Their published records show no appreciable difference between arteries and veins in time of onset of constriction.

In our experiments, the time lag on the venous side roughly paralleled the lag in arterial pressure rise—the longer the delay in onset of arterial hypertension, the longer the lag in venoconstriction.

Maximum pressure in the intravenous balloon was reached, in most cases, by 16 sec. after carotid occlusion. An occasional constriction reached its peak as early as 7 sec.; a few cases required as long as 65 sec. The peak arterial pressure was usually attained a few seconds earlier. Following the release of carotid occlusion, the return to baseline values was also slower on the venous side.

The venous response to unilateral carotid occlusion was much less than that with bilateral occlusion. The depressor effect of the intact opposite carotid sinus continues to be manifest during unilateral occlusion. In addition, the greater response to bilateral carotid occlusion may be, in part, a function of the pressor effect of hypoxia and hypercapnea on the carotid bodies.

Following bilateral cervical vagotomy, the venoconstrictor effect of carotid occlusion was considerably enhanced. The arterial pressure rise was also more marked than before, despite the higher baseline value. The effect of vagotomy was, in all likelihood, a reflection of the elimination of the homeostatic function of the deafferented aortic arch. Transient venoconstriction was occasionally observed following section of the vagus on each side.

**DISCUSSION**

Although no direct measurements of arteriolar tone were made in these experiments, it seems safe to assume that the demonstrated increases in arterial pressure were at least in part a result of arteriolar constriction as well as tachycardia. Brind and associates⁸ have...
recently reported a rise in total peripheral resistance after carotid occlusion.

The data appear to justify the conclusion that hypotension in the carotid sinus may initiate peripheral venoconstriction. Arteriolar constriction alone would be expected to lower the pressure in an intravenous balloon by reducing venous inflow but an elevation of balloon pressure was observed. Neither hydrostatic venous pressure nor tissue tension was elevated by carotid occlusion, so an increase in intravenous balloon pressure could not be attributed to these factors. Therefore, it was considered valid to interpret it as an expression of a reflex increase in tone of the vein wall.

These experiments are consistent with Gollwitzer-Meier's suggestion of parallel responsiveness of peripheral arteries and veins under the control of the brainstem vasomotor center. Although our data stress the importance of the carotid sinus in the regulation of peripheral venous tone, other sites of reflex influence are, of course, not excluded. The results of vagotomy suggest that the aortic arch mechanism is also significant. Intrathoracic receptors on the low pressure side of the circulation may also be important. It is likely that the total play of pressor and depressor influences on the vasomotor center is also affected by various chemoreceptors. These may contribute to the observed difference between the effects of unilateral and bilateral carotid occlusion.

Parallel activity of peripheral arterioles and veins is suggested by the association in time course and magnitude of venoconstriction and arterial pressure rise following carotid occlusion. The greatest rise in venous tone was seen in dogs which also exhibited a dramatic arterial pressure response.

The role of peripheral venous tone in influencing the circulating systemic blood volume by altering the size of the venous reservoir has been emphasized by Celander and Sjöstrand. It appears that the reflex control of this important homeostatic function may lie in the classical mechanisms previously elucidated in studies of the high pressure side of the circulation.

**Summary**

By a miniature balloon technic, reflex constriction of superficial peripheral veins has been demonstrated following carotid occlusion. Carotid sinus denervation eliminated the venous response. Venoconstriction was intensified by deafferentation of the aortic arch by cervical vagotomy. An association in time course and magnitude was observed between venous and arteriolar activity. Previous suggestions of parallel responsiveness and function of peripheral veins and arterioles were confirmed.

**Acknowledgments**

I am grateful to Miss Lois Drawdy of Johns Hopkins Medical School for her assistance in the experiments reported and to Mr. John Olt of the Aero Medical Laboratory for the manufacture of the miniature balloons.

**SUMMARY IN INTERLINGUA**

Per medio de un technica a ballones in miniatura, reflexe constrictiones del venas peripheric superficial ha essite demonstrate post occlusion carotida. Le disnervation del sino carotic eliminava le responsa del venas. Le venoconstriction esseva intensificate per disafferentation del arco aortic effectuate per vagotomy cervical. Un association inter le activitates venose e arteriolar esseva notate in magnitude e curso temporal. Previe suggestiones que il existe un parallelitate de responsa e de function inter periferic venas e arterioles esseva confirmate.

**REFERENCES**

The Circulation as of 1785

Harvey's *De Motu Cordis et Sanguinis* appeared in 1628. The meager progress made from morphological inferences and presumptive reasoning during the following century and a half can be inferred from a physiology text available to Edinburgh medical students after 1785.

A few extracts indicate the limitations of knowledge preceding the era of experimentation:

CLVII.—The contraction and relaxation of the heart, or, as these are called, its systole and diastole, are necessarily alternate by the general law affecting all muscles, and by the stimulus from the influx of venous blood being alternately applied and removed.

CLVIII.—If we may be allowed to estimate the vigour of muscles by the number of their fibres, we must suppose the force of the heart to be very considerable; but it is very difficult to obtain any exact estimate of its absolute force.

CLXIV.—The motion of the blood in the arteries of any particular part is promoted by the action of adjoining muscles.

CLXV.—The blood in the vena cava, and its branches, is moved by the action of the heart and of the arteries. These powers are assisted by the action of muscles, which, in their contraction, press the veins lying between their several fibres, etc.

CLXIX.—The velocity of the blood passing out of the left ventricle of the heart into the aorta, may be estimated by knowing the quantity of blood passing out at each systole, the area of orifice of the aorta, and the time occupied by the systole; but none of these circumstances are exactly ascertained.

CLXXI.—The velocity and the causes of retardation being given, the velocity of the blood in the arteries will be as the frequency of the systole of the heart.

CLXXIX.—The flexibility and contractility of the blood-vessels render the effects of all increase or diminution of resistance in any particular part most considerable in the nearest, and very little so in the more remote, vessels of the system.

Reflex Peripheral Venoconstriction Induced by Carotid Occlusion
EDWIN W. SALZMAN

Circ Res. 1957;5:149-152
doi: 10.1161/01.RES.5.2.149

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1957 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/5/2/149

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation Research can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation Research is online at:
http://circres.ahajournals.org/subscriptions/