sory nerve in acute preparations, as well as of the vagal trunk and cardiac branches in animals with chronic vagal radicotomy, causes EKGraphic changes which parallel those obtained from the vagal root, or from the vagal trunk after chronic radicotomy of the bulbar root of the accessory nerve, and which depend upon the side of stimulation. On the right side, sinus bradycardia develops and proceeds, as the current strenght is increased, to cardiac arrest, without significant alteration in the A–V conduction rate. On the left side, the slowing down of the A–V conduction, leading, as the current strength is increased, to partial (2:1, 3:1) and complete A–V blocks, predominates over the effects upon the sinus rythm, which become prominent only after having the A–V block fully developed; but, while the sinus rythm appears to be only moderately affected even by maximal stimulation of the ipsilateral vagal trunk in animals with chronic vagal radicotomy, a sinus arrest can still be produced in animals with chronic section of the bulbar accessory root, as in normal ones.

In conclusion, our observations indicate that the cardio-inhibitory fibers leave the medulla through both the vagal and the bulbar accessory roots. Both components enter the cardiac branches as unmyelinated fibers and show the same pattern of distribution to the intrinsic structures of the heart, the right side components involving predominantly, but not exclusively, the sino-atrial node, the left side components the atrio-ventricular node. Quantitatively, however, the vagal component is largely predominant.

Riassunto. Le fibre cardio-inibitrici della radice bulbare del nervo accessorio spinale presentano un piano di distribuzione periferica (rami cardiaci del vago-strutture intrinseche del cuore) non distinguibile da quello delle fibre della radice del vago, che rappresentano la componente numericamente predominante.

L. Sperti, M. Midrio and E. Xamin

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Effects of Selective Intracranial Section and Stimulation of Vago-Accessory Roots. IV. Reflex Activity of 'Accessory' Cardio-Inhibitory Neurons

In order to analyse the reflex activity and related properties of the cardioinhibitory neurons sending their axons into the bulbar root of the accessory nerve[1,2], 24 dogs with unilateral, chronic or acute, intracranial section of the vagal root have been submitted to several experimental procedures. Vagal radicotomy has been limited to one side, in view of possible accidental lesions of the IX th nerve root during the intracranial procedure[1]. In both chronic and acute preparations vagal de-efferentation was carried out by sectioning the contralateral vagal trunk at the neck, and the dependence of the heart rate responses upon the bulbar accessory fibers was then checked by ipsilateral vagotomy.

(a) Section (or cooling) of the vagal trunk of the same side of the chronic or acute vagal radicotomy does not significantly affect, either when it is performed before or after section (or cooling) of the contralateral vagal trunk, the pre-existing heart rate in otherwise normal animals; on the other hand, a definite increase in the heart rate has been shown, in both cases, by decerebrate or morphine-treated animals, displaying low basal heart rate and marked sinus arrhythmia. (b) Electrical stimulation of the central end of the contralateral vagal trunk causes a remarkable fall in the heart rate, almost completely prevented by the division of the ipsilateral vagal trunk. (c) Section of the contralateral vagal trunk strongly increases the corresponding threshold but does not prevent the slowing of the heart rate or even the cardiac (or ventricular) arrest in response to mechanical (including Moissejeff's pouch) or electrical stimulation of either carotid sinus pressoceptors; such a response is no longer present after section of the ipsilateral vagal trunk. (d) Intravenous administration of epinephrine or nor-epinephrine (0.5–2 μg/kg body weight) induces an arterial hypertensive phase upon which a bradycardiac response, although less marked and shorter than before, still develops after section of the contralateral vagal trunk. Ipsilateral vagotomy abolishes, almost completely, such a bradycardiac response. (e) Electrical stimulation of points in the antero-lateral hypothalamus or in the pre-optic area, through bipolar, insulated except at the tip, stereotaxically placed electrodes, evokes bradycardiac responses (mostly post-stimulatory) which are not significantly affected by the division of the contralateral vagal trunk, but completely abolished by the ipsilateral vagotomy.

From the above results it is concluded that the cardio-inhibitory neurons sending their axons into the bulbar root of the accessory nerve can be activated through the same pre-synaptic channels as the vagal ones. The similarity, therefore, between vagal and 'accessory' cardio-inhibitory neurons, as far as functional properties and pattern of peripheral distribution[2] are concerned, strongly suggests that the 'accessory' neurons have to be considered as a fraction of the cardio-inhibitory center. Such an 'accessory' pool of cardio-inhibitory neurons, which is large enough to cause cardiac (or ventricular) arrest when extensively activated[1,2], does not seem to play a significant rôle in the control of the normal 'resting' heart rate, but can display relevant levels of activity in conditions of increased excitability of the cardio-inhibitory neurons (decerebrate or morphine-treated animals), and (epinephrine, nor-epinephrine), or in response to increased pressoceptors discharge.

Riassunto. I neuroni cardio-inibitori le cui fibre escono con la radice del nervo accessorio spinale possono essere attivati attraverso le stesse vie presinaptiche di quelli vagali e devono quindi essere considerati come una frazione del centro cardio-inibitore.

L. Sperti, M. Midrio and E. Xamin

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[1] L. Sperti and E. Xamin, Exper. *16*, 556 (1960).
[2] L. Sperti, M. Midrio, and E. Xamin, Exper. *18*, 96 (1962).