receptors of the aortic areas predominated. The results herein presented indicate that in the rat the inhibitory effect on the sympathetic tonus exerted by the aortic baroreceptors is much more important than that of the carotid baroreceptors[8,9].

Individual values of the direct mean arterial blood pressure and heart rate 5 h after isolated denervation of the carotid or the aortic baroreceptors

	Carotid baroreceptors		Aortic baroreceptors	
	Blood pressure (mm Hg)	Heart rate (beats/min)	Blood pressure (mm Hg)	Heart rate (beats/min)
	112	530	147	430
	115	520	150	460
	118	500	150	480
	129	470	150	410
	131	480	153	450
	132	490	155	430
	135	510	160	490
	137	480	160	510
	142	490	162	460
	143	500	167	510
	154	520	177	400
(Means ± S.E.)	(132 ± 4)	(499 ± 6)	(157 ± 6)	(457 ± 5)

Resumen. Los resultados presentados demuestran que en la rata no existe período latente para el desarrollo de hipertensión neurogénica, ya que inmediatamente después de la sección bilateral de los nervios depresores aórticos y carotídeos la hipertensión arterial alcanza sus valores máximos. Por otra parte, se observó que, aún en esa fase aguda, la destrucción aislada de los presoreceptores aórticos o carotídeos tiene efecto diferente, siendo la denervación aórtica más efectiva em provacar hipertensión. Esos resultados sugieren que en la expecie rata los presoreceptores aórticos son normalmente más importantes para mantener una inhibición tónica sobre el sistema simpático.

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[9] The work was performed with the technical assistance of Mr. E. D. MOREIRA.

Fine Structural Alterations of Presynaptic Endings in the Superior Cervical Ganglion of the Cat after Exhausting Preganglionic Stimulation

For the understanding of the synaptic transmission, it seems necessary to clear up the functional role of the main structural elements of the pre- and post-synaptic terminals. DE ROBERTIS et al.[1,2], HUBBARD and KWANBUNBUMPEN[3] and DYACHKOWA[4] observed morphological changes at interneuronal synapses and neuromuscular junction, respectively, which could be taken in parallel with physiological changes in transmission.

These results support the hypothesis that synaptic vesicles play a certain role in the transmission; however, in most parts of the experiments quoted above no measures were taken to have a direct control over the actual state of the transmission, i.e. no parallel physiological recording was undertaken.

In this respect the superior cervical ganglion of the cat seemed to be more promising because it allows a direct physiological control of the actual level of transmission. Now we publish the first results of such a complex approach trying to correlate the submicroscopic morphology of the synapse with the physiological findings.

Cats of both sexes, weighing 1.5–2 kg, were anaesthetized with Nembutal i.p. (40 mg/kg). The superior cervical ganglia were exposed on both sides with the vessels supplying them. Perfusion experiments were performed using the method of KIBJAKOV[5] as modified by PERRY and PATON[6]. In other experiments the natural blood supply was preserved. Preganglionic stimulation was made with rectangular pulses of the following parameters: frequency 10–20/sec; duration 1 msec; amplitude 6–8 V. The contractions of the nictitating membrane and action potentials were recorded. The duration of stimulation ranged from 15 min to 2 h. After the stimulation period, the ganglia were subjected to routine electron-microscopic examinations.

In the ganglia with natural blood supply, after 90 min of stimulation no sign of fatigue occurred. The action potential shows maximum transmission and the ultrastructure of ganglionic synapses are similar to those in resting state[7], there is no change in number of synaptic vesicles, although more irregular form can be seen (Figure 1).

Perfusion of 60 min with Locke solution did not alter the picture appreciably. The excitability of the ganglion also remained normal. Preganglionic stimulation with the parameters mentioned above, however, led to a rapid exhaustion and after 15 min of stimulation a complete failure of transmission ensued. The electron-microscopic picture of such ganglia (Figure 2) revealed very marked changes: the presynaptic endings proved to be almost completely empty. A few vesicles seem to assemble around the site of the synaptic contact. The mitochondria in the presynaptic endings are heavily damaged: they are swollen and the well-known fine structure is apparently destroyed. The mitochondria of the postsynaptic elements are at the same time quite normal in appearance.

If we added to the perfusion fluid of the ganglion 10 mg/l choline chloride and stimulated them preganglionically with the above parameters, the transmission remained almost intact and we did not observe the above-mentioned

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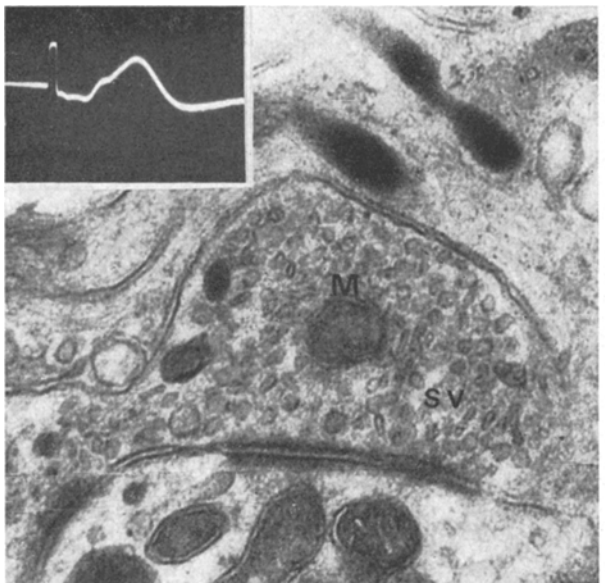

Fig. 1. After 90 min of stimulation, with the exception of some irregularity in the shape and size of the vesicles, no alteration can be seen in the synaptic apparatus in ganglia with natural blood supply. sv, synaptic vesicles; M, mitochondrium. The action potential (inset) shows maximal transmission. × 45,000.

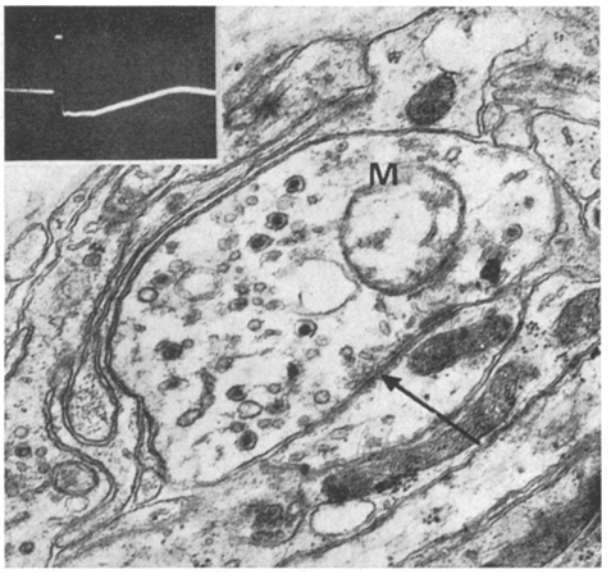

Fig. 2. Presynaptic endings with swollen mitochondrium and synaptic vesicles reduced in number, after 15 min of preganglionic stimulation and perfusion with Locke solution. Arrow indicates the synaptic contact. M, mitochondrium. The action potential shows the total abolition of the transmission. × 30,000.

structural alterations. A more detailed report in this subject will be published later.

Our preliminary experiments indicate that the synaptic vesicles play a decisive role in the storage and release of the transmitter substance. Preserving the normal blood supply, the superial cervical ganglion cannot be exhausted by electrical stimulation because the stores of acetylcholine are refilled by newly synthesized material. Perfusing the ganglion with Locke solution without choline added, the transmitter stores are exhausted rapidly and the transmission fails. At the same time only a small number of vesicles and swollen mitochondria are found in the presynaptic terminals. It seems, therefore, that with exhaustion of the transmitter stores also the vesicular and mitochondrial structure was destroyed. By addition of choline we were able to preserve the transmission and maintain the ultrastructure of the terminals in a satisfactory condition.

Based upon our results, we conclude that exhaustive stimulation releases not only acetylcholine stored in the synaptic vesicles but utilizes also the choline structurally bound in the terminals.

Zusammenfassung. Untersuchungen an sympathischen Ganglien bei intakter Blutversorgung und nach Durchströmung mit Locke-Lösung. An den perfundierten Ganglien wurde nach Reizung eine Abnahme der synaptischen Vesikel sowie eine Schädigung der Mitochondrien gefunden. Cholinzusatz konnte die degenerativen Veränderungen beheben.

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Die Rolle von Glukose im Erholungsprozess des Muskels

Es ist bekannt, dass nach erschöpfender Muskelarbeit die Erholung durch etwas Zucker, gewöhnlich Saccharose, sehr rasch hergestellt wird. Hypoglykaemien anderer Art (zum Beispiel Alters-Hypoglykaemie) werden natürlich auch restituiert. Die Wirkung des Zuckers wird gewöhnlich energetisch erklärt.

Der Mechanismus dieser Wirkung ist jedoch ein anderer. Muskelarbeit beginnt mit der Reaktion ATP → ADP. Die Restitution von ADP → ATP verläuft nach grösseren Arbeitsleistungen insbesondere auf Kosten der Reserven von Kreatinphosphat im Muskel.

Nach Versuchen an Ratten ergab sich, dass erwachsene Tiere rund 50% vom gesamten Kreatin des Muskels als Kreatinphosphat enthalten. Bei alten Tieren ist zwar die gleiche Menge Kreatin vorhanden, aber davon nur rund 25% Kreatinphosphat[1,2].

Füttert man alte Tiere (22–30 Monate alt) mit einer Diät welche 50% Glukose enthält und daneben in jeder Beziehung sonst komplett ist, während 3 oder mehr Tagen, dann steigt das Kreatinphosphat auch bei diesen auf 50%, wie bei jungen Tieren[3].

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