of the transmural pressure is constantly being slightly moved as opposed to the central venous pressure, only the end of the expiratory respiration phase was considered.

The following findings were recorded: 1. There is a close correlation between the central venous pressure and the length of the body (Figure). 2. Contrary to the peripheral venous pressure, the central one shows no age dependence. 3. Small children have a relatively high central venous pressure, explained by the 'physiological centralization'[4]; the relationship = length of the body: the central venous pressure applies only to a length over 140 cm. 4. Measured under constant conditions, the central venous pressure is the same for every experimental person over a number of weeks. 5. No relationship between the central venous pressure and the body weight, sex, haematocrit and total protein in the serum is seen.

Our findings made a more exact physiological correlation between the central venous pressure and the corresponding length of the body possible. Similar relationships are known for the cardiac output, plasma volume, total blood amount and other values[2]. The practical consequence of the results for the clinic is the possibility of a more exact volume substitution which necessitates the registration of the venous pulse. Loss of volume and changes of the venous tonus (cold, pain, respiration, etc.) are always expressed in a distortion of the venous pulse before distinct pressure changes become measurable.

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The Change of Vagal Activity Evoked by Spinal Cord Thermal Stimulation in Anesthetized Rabbits

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Summary. Vagal activity decreased significantly during spinal cord warming and increased significantly during spinal cord cooling in anesthetized, immobilized rabbits. The results provide the first direct proof of changes in parasympathetic activity during spinal thermal stimulation.

Thermal stimulation of the spinal cord elicits changes in various autonomic functions, for instance, circulation, respiration, shivering and non-shivering thermogenesis and sweating (reviewed by Simon[1]). Changes in regional sympathetic activity provide one of the important underlying mechanisms involved in these responses[2,3]. Despite the fact that autonomic functions are well kown to be regulated normally not only by the sympathetic system, but also by the parasympathetic system, participation of the parasympathetic system in these autonomic changes has not been reported. In this experimental series, the change in activity of vagal efferents during spinal thermal stimulation were directly investigated to clarify this problem.

Methods. The experiments were performed with 10 rabbits of either sex weighing 1.8–2.6 kg. The animals were anesthetized with sodium pentobarbital (40 mg/kg as initial dose and subsequent continuous infusion of 14 mg/animal/h) and immobilized with succinylcholine (40 mg/animal as initial dose and subsequent infusion of

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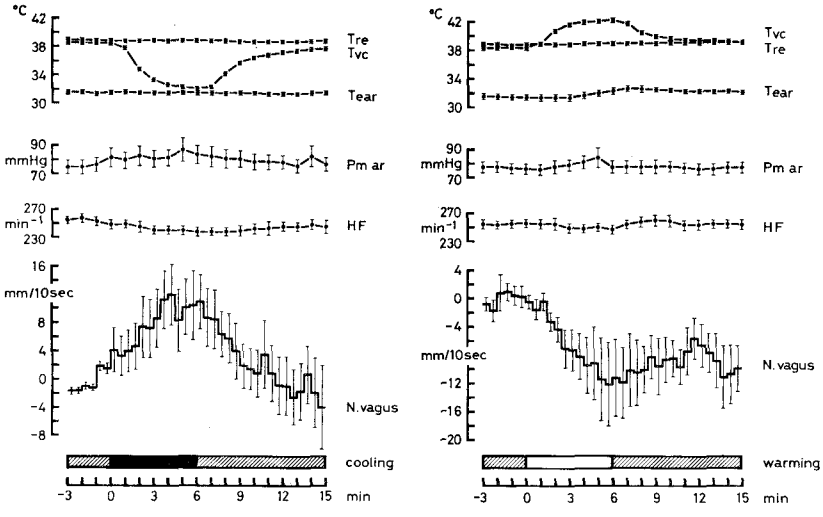

Integrated vagal activity (N. vagus), rectal, vertebral canal and ear temperatures (T_{re}, T_{vc}, T_{ear}), arterial mean pressure (Pm ar) and heart rate (HF) as influenced by spinal cord cooling (left figure) and warming (right figure) in anesthetized, immobilized rabbit. Mean values from 10 experimental animals with standard errors. The courses of vagal activity are visualized by graphing the changes in amplitude of integrator signals in mm/10 sec, reference level: average integrator signal amplitude during the prestimulation period.

300 μg/animal/min). The animals were artificially ventilated by means of Starling Pump, and were placed on a heating pad perfused with water at 40 °C, to avoid a fall in rectal temperature during experiments. Ambient air temperature was kept constant at 25 °C.

Thermal stimulation of the spinal cord was performed by perfusing cold or hot water for a period of 6 min through polyethylene tubing, which was introduced into the peridural space of the vertebral canal extending from the sacral bone to the lower cervical vertebrae. One of the twigs of the vagus to the gastrointestinal tract just below the diaphragm was separated, and the mass electrical discharges were recorded with bipolar stainless steel electrodes. The mass discharge was recorded directly and was additionally fed into operational amplifiers by which the activity was integrated during 5 sec intervals. This integrated activity was used to evaluate the change of vagal activity in a semiquantitative manner, by comparing the changes in the integrator deflection amplitude throughout the experimental period with the prestimulation levels. In addition to vagal activity, temperatures of the rectum, spinal cord and ear skin, arterial blood pressure and heart rate were all recorded simultaneously on a UV-direct writing oscillograph. Mean arterial pressure was calculated as $P_{diast} + 0.3\ (P_{syst} - P_{diast})$. The statistical significance of differences between paired observations were tested using the Wilcoxon matched-pairs signed-ranks test.

Results and discussions. The time course of the change in vagal activity and of other parameters evoked by spinal cord thermal stimulation are demonstrated in the Figure. Each point indicates the mean value with its standard error for 10 experiments. During spinal cord warming (Figure A), vagal activity decreased significantly, ear skin temperature rose significantly, but mean arterial blood pressure and heart rate showed no significant changes. After the end of spinal cord warming, all changed parameters returned gradually to the prestimulation levels. During spinal cord cooling (Figure B), vagal activity increased significantly, but there were no significant changes in any of the other functions. No significant decrease in ear skin temperature during spinal cord cooling in this experimental series could be attributed to the condition of ear blood vessels in the prestimulation period.

The Table indicates the change in vagal activity during and after spinal thermal stimulation, compared with the prestimulation period. Mean values during the last 3 min of each period were used for statistical analyses. As shown in this Table, activity of the vagal efferents was increased significantly during spinal cord cooling and decreased significantly during spinal cord warming.

The peripheral end of nerve twigs used for recording, was crushed with a pincette to avoid mixing of afferent elements. The influence of bilateral vagotomy at the cervical level was measured to ascertain the connection between the recorded nerve twig and vagal nerve. Vagotomy resulted in a marked decrease of the discharges. Further, because sympathetics could be mixed in the recorded nerve twigs, the change of activity during spinal cord thermal stimulation after bilateral vagotomy was ascertained; no significant change was observed. Therefore, the recorded change in nervous activity during spinal thermal stimulation was based on a change in vagal activity, and not on the influence of other factors.

TSUCHIYA et al.[4] reported that gastrointestinal motility is changed during spinal cord thermal stimulation and that sympathetic elements participate in this response, because the change was still observed after bilateral vagotomy at the cervical level. This result is supported by the fact that splanchnic efferent activity decreased during spinal cord cooling and increased during spinal cord warming[2]. However, because the parasympathetic system is also important in the regulation of gastrointestinal motility, the contribution of parasympathetics to this response has to be considered. In fact, as shown in this paper, this response can be also explained by the change in vagal activity, i.e., increase during cooling and decrease during warming, because an increase in vagal activity stimulates gastrointestinal motility. That is, spinal cord thermal stimulation causes changes in both sympathetic and parasympathetic activity, and the change in gastrointestinal motility evoked by spinal thermal stimulation is based on both these functions.

These results also provide the first direct proof of changes in parasympathetic activity during spinal thermal stimulation, thereby indicating that further investigations of the mechanisms of changes in autonomic functions evoked by central or peripheral thermal stimulation are warrented.

Change of vagal activity during spinal cord thermal stimulation of 6 min duration

		Vagal activity (mm/10 sec)		
		Before	During	After
Time after start of stimulation (min)		−3–0	3–6	12–15
Spinal cord warming	$\bar{x}$	0	−9.8[a]	−8.6
	$S\bar{x}$		±4.3	±3.6
Spinal cord cooling	$\bar{x}$	0	+9.9[a]	−1.8[b]
	$S\bar{x}$		±4.0	±4.6

10 rabbits. The data of the table were calculated as average values for 3 min intervals, reference value: discharge level during 3 min before start of stimulation. $\bar{x}$, mean values; $S\bar{x}$, standard errors.
[a] Data significantly different from the values before stimulation.
[b] Data significantly different from the values at the end of stimulation ($p<0.05$, Wilcoxon matched-pairs signed-ranks test).

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