ascorbic acid when large amounts of the vitamin are added to the diet[7].

These findings suggest that ascorbic acid has an active role in brown adipose tissue in rats undergoing cold acclimation. In this process the tissue undergoes morphological and biochemical changes which result in nonshivering thermogenesis[13].

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Enhanced depressor reflex by stimulation of superior cervical ganglion and by propranolol[1]

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Summary. The systemic depressor reflex in cats evoked by inflation of an intrasinusal balloon is enhanced by stimulation of the superior cervical ganglion and by close infusion of 20 µg/ml/min of (d, l)propranolol.

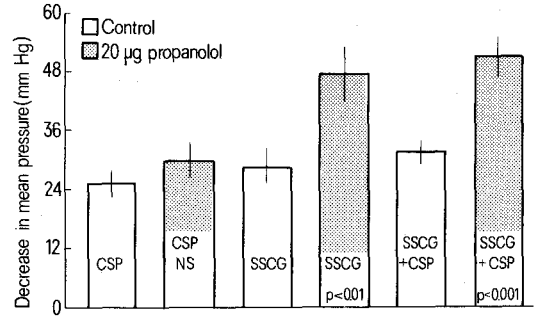
The functional role of the sympathetic nerves supplying the carotid sinus has been disputed[4], but a number of studies claim that stimulation of these nerves evokes a decrease in carotid sinus diameter[5], an increase in sinus nerve activity[6] and inconsistent depressor responses[7]. Recent studies also suggest that propranolol produces an increase in sinus nerve activity in the hydrodynamically restricted sinus, probably by decreasing sinus distensibility[8]. Constriction of smooth muscle by propranolol has been reported both in the guinea-pig trachea[8] and vascular bed of the rabbit ear[9]. Both tissues also receive a postganglionic sympathetic nerve supply from either the superior cervical (SCG) or stellate ganglia. This evidence might suggest that propranolol enhances smooth muscle tone through an interaction with this sympathetic innervation. The present investigation was designed to determine if the depressor reflex, in response to an increase in intrasinusal pressure, was enhanced by stimulation of the superior cervical ganglion (SSCG) and which modality - sinus pressure or stimulation - was most influenced by (d,l)propranolol.

Materials and methods. 7 cats weighing 2–3 kg were selected at random and anesthetized with pentobarbital sodium (50 mg/kg). Tracheal and venous (femoral vein) catheters were inserted. A femoral arterial cannual was connected to a pressure transducer and oscillographic paper recorder. One carotid sinus was isolated by typing the carotid artery above and below the sinus. All collateral circulation was excluded except the lingual artery which was cannulated with a 0.5 mm diameter catheter.

The ascending sympathetic trunk just caudal to the SCG was cut and led through and 'O' ring stimulating electrode. The latter was connected to a Grass stimulator and isolation unit. The ascending and descending vagus nerve was carefully excluded from the region of the electrode by microdissection and extirpation of at least 10 mm of nerve above and below the electrode. The sympathetic nerve was then bathed in warmed mineral oil and stimulated at 8–15 V and 8/sec, threshold was monitored via the nictitating membrane. A Berman No. SC301 angiographic balloon catheter was inserted into the carotid artery and advanced cranially and, by palpation, positioned within the bifurcation of the sinus. The catheter, consisting of the balloon tip and perforated collar for drug administration, was then inflated to a pressure of 180 mm Hg which displaced a volume of approximately 0.10 cm^3.

When a consistent depressor response to balloon inflation (CSP) had been established, the SCG was stimulated alone, and then in combination with balloon inflation. After repeating these 3 events a number of times, propranolol (20 µg/ml/min) was flushed through the sinus via the perforated neck of the catheter and out the sinus via the lingual artery. The tests were then repeated. In some cases, isoproterenol was infused before and after propranolol, but in no case did this catecholamine modify the responses obtained.

Results and discussion. Both CSP and SSCG reduced systemic blood pressure and in combination, they produced an enhanced reduction of this parameter. Propranolol intensified the depressor response evoked by SSCG. When combined, the depressor response was enhanced to an amount more nearly equal to that produced by SSCG alone. This is shown in the bar graph from data in 7 cats evaluated for statistical significance. As shown in the figure, SSCG was the modality most significantly effected by propranolol. This, in turn, contributed to the significant enhancement of



Statistical comparison in 7 cats of the effects of balloon inflation (CSP), sympathetic stimulation (SSCG) and combination (CSP+SSCG) on systemic pressure. Shaded bars and levels of significance (p values) represent change (± SE) to each modality of stimulation after exposure to d,l-propranolol.

the combined response to SSCG and CSP by propranolol. Heart rate (not illustrated) showed no statistically significant reductions, during CSP, SSCG, or after propranolol. These results show that SSCG alone, and especially in conjunction with an elevated intrasinusal pressure, can produce a systemic depressor response. Propranolol, limited only to the sinus region, further enhanced this depressor response, primarily through the effect on SSCG and not through CSP. This would seem to suggest that an intact sinus innervation is necessary if one wishes to demonstrate an effect of propranolol on the sinus depressor and nerve activity[8].

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Changes in the A-band width during contraction in horseshoe crab striated muscle

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Summary. Cinematographic recordings of Ca-activated contraction in glycerinated bundles of myofibrils of the horseshoe crab striated muscle indicated that the A-band width did not change during rapid sarcomere shortening from 11 to 4 μm, while it increased by about 10% in nearly isometric conditions.

Contrary to the general view that contraction in striated muscle results from a relative sliding between the thick and thin filaments[2,3], horseshoe crab striated muscle is known to exhibit marked shortening of the A-band which is accompanied by shortening of the thick filaments under some conditions[4-8], thus suggesting the possibility that the thick filament shortening serves as an additional mechanism producing force and motion. In the above literature, however, the A-band length changes seem not to have been studied under physiological conditions, and it remains to be determined whether or not the thick filament shortening actually takes place in physiological contractions. The present paper deals with changes in the A-band width during the course of Ca-activated contraction in glycerinated horseshoe crab muscle. It will be shown that the A-band shortening may not be involved in physiological contractions.

Material and methods. Adult horseshoe crabs *(Tachypleus tridentatus)* were collected in the vicinity of Fukuoka, Japan, and fed in chilled (18 °C), aerated sea-water in the laboratory until used. Telson depressor muscles[6] were isolated from the animals, and glycerinated by the method of Tanaka et al.[9]. Bundles of myofibrils (20–50 μm in diameter, and about 1 cm in length) were then dissected from the glycerinated muscle fibres in a relaxing solution containing 100 mM KCl, 3 mM EGTA, 5 mM $MgCl_2$, 4 mM ATP and 10 mM histidine (pH 6.8), and placed in a thin layer of the relaxing solution between a slide and a coverslip, both ends being glued to the slide with Aron alpha A adhesive (Sankyo). The preparation was observed under a phase-contrast microscope (Nikon DM40 objective, ×40, n.a. 0.64), and activated with iontophoretically applied Ca ions through a glass pipette (diameter 5–15 μm) filled with 1 M $CaCl_2$[10]. The initial sarcomere length of the preparation mounted in this way was uniform along its length, ranging from 7 to 11 μm. The resulting local contractions were recorded on Kodak 7247 or Fuji Minicopy films with a 16-mm cine-camera (Bolex) at 64 frames/sec (figure 1, a, b and figure 2, a, b). The length changes of a single sarcomere and its A-band at a definite part of the preparation, where the striations were clear, were measured during the course of contraction either directly on the cinefilm with a film motion analyser (Vanguard), or indirectly after microdensitometer (type NLM-D3, Narumi) tracings of the cinefilm with similar results. All experiments were performed at room temperature (18–20 °C).

Results. As shown in figure 1, c, no significant change in the A-band width was observed during the course of sarcomere shortening from 7–11 μm to about 4 μm; the sarcomere shortening was taken up only by the I-band shortening while the A-band width (3–3.5 μm) remained unchanged. When sarcomeres shortened to less than 4 μm, the A-band became invisible because of the contraction band formation[11]. On relaxation, however, the A-band appeared again with sarcomere lengths above 4 μm, and its width remained unchanged until the completion of relaxation.

The above local contractions involved at most 10 sarcomeres while the length of the preparation was about 1 cm, and under such conditions the activated sarcomeres were considered to be shortening against a very small load[12,13]. As a matter of fact, the shortening velocity at the early phase of local contractions (about 1 length/sec at 20 °C) was similar to the maximum shortening velocity determined on intact muscle fibres[14], indicating that the preparation was in a good physiological condition. Thus, the above results indicate that the thick filament shortening mechanism may not be involved in the physiological sarcomere shortening from 11 to 4 μm, since the thick filaments are fairly well aligned within the A-band at sarcomere lengths below 7 μm[6-8] (Suzuki and Sugi, unpublished).

Figure 2 shows the results of experiments in which the distance between the fixed ends of the preparation was made very short (about 100 μm or less), so that all sarcomeres within this short segment were activated fairly