Effects of Sodium and Temperature on Tension in Isolated Canine Coronary Artery

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The effects of sodium and temperature on tension of isolated canine coronary arterial strips were studied.

In 20 mEq · l^{-1} K solution, the strength of tension was inversely related to the Na concentration. At 37°C, the tension was significantly increased at 70 mEq · l^{-1} Na and below. The tension was gradually suppressed by lowering of the temperature from 37°C to 10°C. At 10°C, tension did not developed significantly at Na concentrations between 127 mEq · l^{-1} and 12 mEq · l^{-1}.

It was concluded that the decrease in Na concentrations increased the tension of the canine coronary artery and the lowering of temperature supressed the tension inducted by the decrease in Na concentrations. (Key words: coronary artery, sodium, hypothermia, tension)

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The electrolyte composition and temperature are the important factors affect tension of smooth muscle. For example, high-K solution produces the tension of the smooth muscle, and the strength of which is dependent on Ca concentration in the solution[1]. And the reduction in extracellular Na concentration increases the tension of the myometrium of the rat[2]. On the other hand, it is known that hypothermia increases the tension of the rabbit ear vessels[3].

We studied the effects of the Na concentration and temperature of the extracellular fluid on the tension of isolated canine coronary artery.

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Methods

The experiments were performed on the anterior descending coronary arteries taken from dogs (10~14 kg, either sex) anesthetized with thiamylal natrium (25 mg·kg^{-1}, i.v.). The coronary artery was dissected and its surrounding tissue was removed under a microscope. Helical strips of about 3 mm in width and 15 mm in length were excised from the segments of coronary artery of 0.8 to 1.2 mm in diameter. Each strip was attached to an isometric force transducer (Shinko UL-10GR) and suspended in a small volume organ bath (37°C) through which the normal solution (see below) was flowed at a constant rate of 2 ml·min^{-1}. After 30 min equilibration, the strip was stretched to give a resting tension of approximately 0.5g and the normal

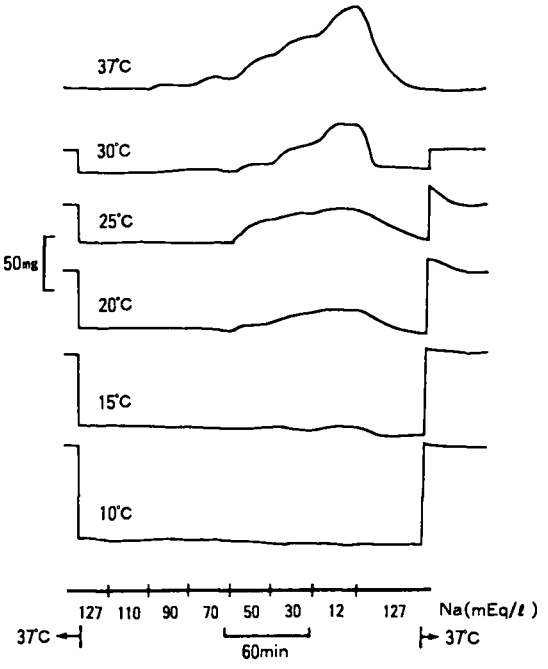

Fig. 1. A typical record of effects of temperature and Na in the high-K, Ca-free solution.

The increases in tension occured in proportion to the decreases in Na concentration, and lowering of the temperature suppressed the increases in tension.

solution was changed to high-K (see below), Ca-free solution. When the tension was stabilized (basal tension, 0 ± 0 mg), temperature of the bath solution was lowered to six different levels, and at the temperature of each level, Na concentration was lowered to seven different levels. At each temperature and Na concentration, the strength of tension was measured.

The normal solution had the following electrolytes composition ($mEq \cdot l^{-1}$): Na 127, K 5.9, Ca 4.7, Mg 2.4; and glucose 11.8 mM. The high-K, Ca-free solution was made by removing Ca from the normal solution, adding 0.5 mM EGTA (ethylene glycolether tetraacetic acid), and adjusting K to 20 $mEq \cdot l^{-1}$. When the Na concentration was decreased, Na was replaced with saccharose on an equiosmolar basis (337 $mOsm \cdot l^{-1}$).

Statistical evaluation was performed by analysis of Wilcoxon rank-sum test or Wilcoxon signed-rank test. A p less than 1% was regarded as statistically significant.

Results

A typical record is shown in figure 1. In the high-K, Ca-free solution, the tension fell gradually with cooling. This decrease in tension caused by cooling was about 5.5 $mg \cdot °C^{-1}$. As shown in table, the tension was −40 ± 8 mg at 30°C (mean ± SD, n = 10), and this is statistically significant ($P < 0.01$) compared with the tension at 37°C (0 ± 0 mg). The tension at 20°C was −98 ± 12 mg, and −133 ± 29 mg at 10°C compared with basal tension. These negative values means relaxation of the strips.

As shown in figure 1 and table, the increase in tension occured in proportion to the decreases in Na in high-K, Ca-free solution at the temperatures of six different levels. At 37°C, the tension increased from 0 ± 0 mg (in 127$mEq \cdot l^{-1}$ Na solution) to 17 ± 7 mg (in 70 $mEq \cdot l^{-1}$ Na solution) ($P < 0.01$). The tension increased from −40 ± 8 mg (127 $mEq \cdot l^{-1}$) to −26 ± 8 mg (70 $mEq \cdot l^{-1}$) at 30°C, −77 ± 10 mg to −61 ± 10 mg (50 $mEq \cdot l^{-1}$) at 25°C, −98 ± 12 mg to −86 ± 8 mg (50 $mEq \cdot l^{-1}$) at 20°C, and −125 ± 16 mg to −177 ± 14 mg (50 $mEq \cdot l^{-1}$) at 15°C. These were statistically significant ($P < 0.01$). But at 10°C, the reduction of Na did not increase the tension (−133 ± 29 mg at any Na concentration).

Cooling gradually suppressed the tension, and completely at 10°C. At 25°C, 12 $mEq \cdot l^{-1}$ Na increased the tension from −77 ± 10 mg to −22 ± 5 mg (table 1). As shown in figure 1 and 2, this increased tension (−22 ± 5 mg) did not reach basal tension (in 127 $mEq \cdot l^{-1}$ Na solution at 37°C, 0 ± 0 mg) ($P < 0.01$).

Discussion

It is reported that denervated blood vessels respond to hypothermia with an increase in tension[4]. On the other hand, prolonged cooling below 12°C causes vasodilatation of the human skin[5]. Our experiments showed that the canine coronary arterial strips were relaxed by lowering of the temperature.

At 25°C, the tension caused by the reduc-

Table 1. The tension at each temperature and Na concentration

		Na (mEq/l)						
		127	110	90	70	50	30	12
Temperature (°C)	37	0±0	0±0	2±3	17±7*	37±9*	65±23*	97±23*
	30	−40±8	−40±8	−38±8	−26±8*	−15±12*	−4±11*	14±5*
	25	−77±10	−77±10	−76±9	−72±9	−61±10*	−50±7*	−22±5*
	20	−98±12	−98±12	−97±13	−94±10	−86±8*	−78±8*	−73±7*
	15	−125±16	−125±16	−125±16	−121±13	−117±14*	−114±14*	−111±13*
	10	−133±29	−133±29	−133±29	−133±29	−133±29	−133±29	−133±29

The values are mean ± SD (mg), and n = 10.

*$P < 0.01$ vs control.

The tension at each Na concentration was compared with the one in 127 mEq · l^{-1} Na at each temperature. Positive values and negative values in tension indicate contraction and relaxation, respectively.

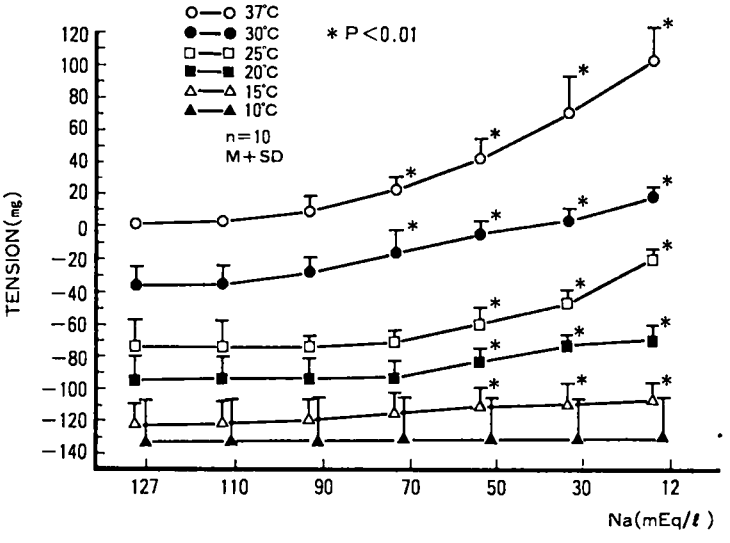

Fig. 2. Na concentration-tension curves in the high-K, Ca-free solution at six levels of temperature.

The strength of tension was dependent on the Na concentration and temperature.

The decreases in Na concentrations below 70 mEq · l^{-1} were required for the development of tension between 15°C and 37°C.

tion in Na concentration to 12 mEq·l^{-1} was smaller than basal resting tension (37°C, 127 mEq·l^{-1}). At 10°C, 12 mEq·l^{-1} Na did not develop a tension. This mean that hypothermia suppresses the increase in tension caused by a decrease in Na concentration significantly below 25°C and inhibits completely below 10°C. The mechanism of this relaxation caused by lowering of the temperature is unclear, but it was demonstrated that the rate of shortening of the actomyosin was markedly slowed by cooling[6] and the temperature had the direct effect on the rate of detachment of crossbridges[7]. Other report showed the cold vasodilatation might not be caused by local release of vasodilator substances such as acethylcholin and histamine[8]. The experiments on isolated blood vessels shows that mammalian arteries did loss their ability to respond to noradrenaline when they are cooled, so the dilatation may be caused by direct cold-induced paralysis of the peripheral blood vessels[9]. On the other hand, the phase transition of the myocardial cell membrane is caused by hypothermia, and there is a possibility that this phase transition changes the permeability of ions, various pumps for ions and/or Na-Ca exchange system[6].

The increase in tension of the myocardium

produced by Ca-free, Na-deficient solution is mediated by the Na-sensitive Ca transport (Na-Ca exchange)[10]. Similarly, reduction of the external Na concentration is known to produce an increase in tension of the pregnant myometrium of the mouse[11], and involvement of a Na-Ca exchange mechanism has also been suggested[12]. The tension induced by Ca-free solution is strongly dependent on extracellular Na concentration, and is substantially suppressed by EGTA which reduces the amount of contaminating Ca[10]. The tension induced by the reduction in Na is probably due to limited Ca influx[13] and Ca transport across the sarcolemma by Na-Ca exchange, may play a critical role in the maintenance of intracellular Ca and resting vascular smooth muscle tone[14]. But our findings showed that Ca-free, low-Na solution containing EGTA produced the increase in tension of the coronary artery. And this increase in tension did not occur above 90 $mEq \cdot l^{-1}$ Na, irrelevantly with temperature. It has been reported in the myometrium that the removal of the external Na in Ca-free solution containing EGTA produced a small sustained tension[13]. It may be that these tension produced by vasoconstrictive transmitter and/or translocation of Ca within the cell and at the inner surface of the smooth muscle cell membrane[15].

In conclusion, the increas in tension of canine coronary artery occured in proportion to the decrease in Na in high-K, Ca-free solution, but this increase in tension did not occur above 90 $mEq \cdot l^{-1}$ Na. Decreases in temperature markedly supressed this increase in tension below 25°C, and completely inhibited at 10°C.

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