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Time Course of Changes in $(dP/dt)/P$ of Carotid Artery Pulses during Cold Pressor Test

Hirotoshi IFUKU, Kohachi TANIGUCHI, Yasuharu OISHI,
Hisahiro MATSUMOTO, and Koji OSHIGE*

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Abstract

We measured $(dP/dt)/P$ of carotid artery pulse (CAP) beat by beat during a cold pressor test and compared it with the results of a previous study performed using muscle sympathetic nerve activity. The time course of changes in CAP $(dP/dt)/P$ could be classified into two patterns : in pattern A, CAP $(dP/dt)/P$ was increased by cold water immersion, whereas in pattern B it was decreased. In pattern A, heart rate increased significantly at 30 s and 60 s of the immersion ($P<0.001$), and CAP $(dP/dt)/P$ increased significantly at 30 s of the immersion ($P<0.01$) and at 30 s after the offset of immersion ($P<0.05$). In pattern B, heart rate also increased significantly at 30 s and 60 s of the immersion ($P<0.001$) and at 30 s after the offset of immersion ($P<0.05$), but CAP $(dP/dt)/P$ decreased significantly at 60 s of the immersion ($P<0.01$) and maintained the decrease until 90 s after the offset of immersion. These changes in CAP $(dP/dt)/P$ were different from those of muscle sympathetic nerve activity. In conclusion, the present study indicated that CAP $(dP/dt)/P$ did not reflect the level of muscle sympathetic nerve activity during the cold pressor test.

Key words : carotid artery pulse, $(dP/dt)/P$, cardiac contractility, cold pressor test

Introduction

In earlier studies, we showed an improved method of fixing a transducer to measure carotid artery pulse (CAP), one which enables continuous and stable recording of both the pressure (P) of CAP and its first derivative (CAP dP/dt) during moderate exercise (IFUKU et al. 1993). Using this method, we subsequently reported that the ratio of the CAP dP/dt to P, CAP $(dP/dt)/P$, is an easy, noninvasive index of cardiac contractility even in moderate exercise, and reflects the state of sympathetic nerve activity (IFUKU et al. 1994). However, that paper focussed on relationships between heart rate and CAP $(dP/dt)/P$, and did not consider the time course of changes in CAP $(dP/dt)/P$.

The cold pressor test, reported by HINES and BROWN (1936), has been used to assess the function of the neural control of the cardiovascular system by observation of the pressor response during the immersion of one hand in cold water. This pressor response has been shown to be induced by enhanced sympathetic nerve activity (HINES and BROWN 1936, CUMMINGS et al. 1983, YAMAMOTO et al. 1992).

As a basal study to clarify time-dependent change in CAP $(dP/dt)/P$ during exercise, we measured CAP $(dP/dt)/P$ beat by beat during the cold pressor test. We then observed the time course of changes in CAP $(dP/dt)/P$ during the cold pressor test and compared CAP $(dP/dt)/P$ with the results of a previous study performed using muscle sympathetic nerve activity

* Graduate School of Engineering, Kagoshima University, Kagoshima 890, Japan

(YAMAMOTO et al. 1992).

Methods

Subjects. Four healthy male subjects, aged 21-23 years, volunteered for this study. They had no medical history of circulatory disease, and their CAP contour, electrocardiogram (ECG), and blood pressure were normal. Informed consent was obtained.

CAP and ECG. To record CAP, a pulse transducer (45259, NEC SAN-EI) fixed on a special apparatus (IFUKU et al. 1993) was held over the right carotid artery. The subjects were allowed to swallow to relieve any discomfort from wearing the apparatus. The ECG was recorded using bipolar chest leads. The CAP and ECG were simultaneously recorded on a data recorder (R-61, TEAC) throughout all the experiments.

Experimental procedures. After resting 10-20 min in a supine position, the subjects underwent the cold pressor test according to Hines and Brown's method (HINES and BROWN 1936), which required them to immerse the right hand in water at 4°C for 1 min. Experimental protocol was resting for 1-2 min, immersion for 1 min, and recovery for 3 min.

Data processing. The CAP and ECG recorded on the data recorder were reproduced and led to a laboratory-oriented microcomputer (PC9801FA, NEC) via an A/D converter. The sampling rate of the A/D converter was 200 Hz (5 ms). After some artifacts of CAP and ECG were eliminated by a moving average method (5 data points), CAP P and dP/dt were measured in millimeters of mercury and millimeters of mercury per second, respectively (IFUKU et al. 1993), and $(dP/dt)/P$ (per second) was calculated. The R-R intervals of ECG were also measured, and the instantaneous heart rate was calculated by multiplying the inverse of the R-R interval (per second) by 60 s. The obtained data were compared with control data using the Student's t-test. A P value of less than 0.05 was considered significant. Further details may be found in our previous paper (IFUKU et al. 1994).

Results

Beat by beat measurement of CAP $(dP/dt)/P$ during cold pressor test

Figure 1 shows an example of beat by beat measurement of CAP $(dP/dt)/P$ in the cold pressor test. During cold water immersion, CAP $(dP/dt)/P$ increased transiently and restored to pre-immersion levels. After the offset of immersion, it maintained these levels. Thus, CAP$(dP/dt)/P$ could be measured beat by beat during the cold pressor test.

Also during test, the time course of changes in CAP $(dP/dt)/P$ could be classified into two patterns : in pattern A CAP $(dP/dt)/P$ was increased by cold water immersion (Fig. 1), while in pattern B it was decreased. Of the four subjects, two showed pattern A and the other two pattern B.

Responses of heart rate and CAP $(dP/dt)/P$ to cold pressor test

Changes in heart rate and CAP $(dP/dt)/P$ during the cold pressor test are shown in Table 1. All values are expressed as an average of ten successive values immediately before each time.

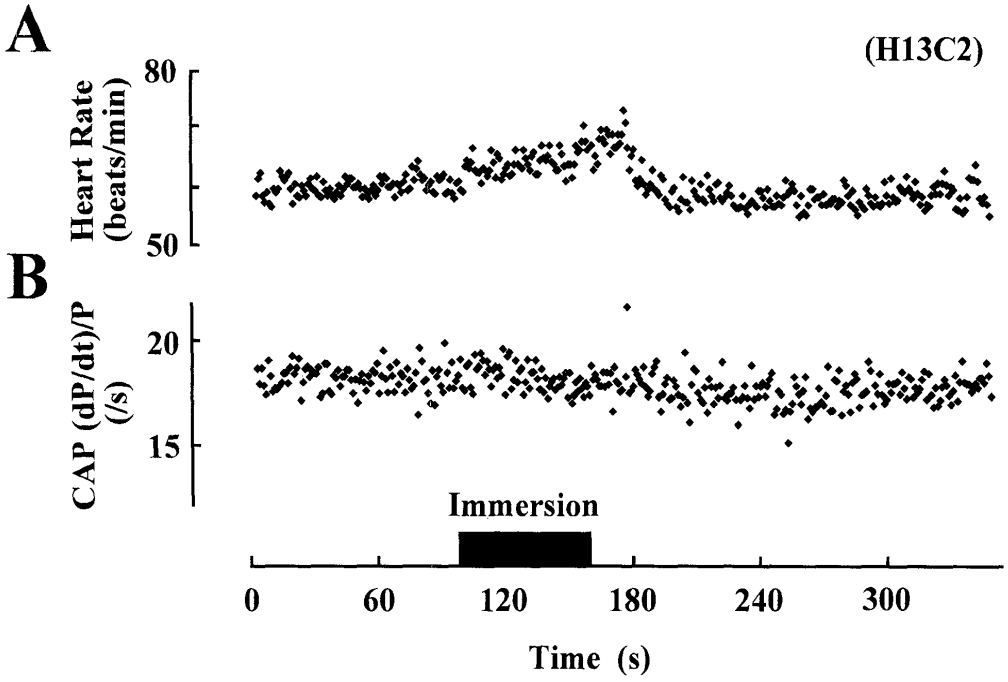

Fig. 1. An example of beat by beat measurement of heart rate (**A**) and CAP $(dP/dt)/P$ (**B**) during the cold pressor test. By our improved method of fixing a pulse transducer, CAP $(dP/dt)/P$ could be measured beat by beat during the cold pressor test.

During immersion in cold water, the heart rate increased significantly ($P<0.001$) in both patterns, from 61.9 beats•min^{-1} to 69.6 beats•min^{-1} at 30 s and 69.3 beats•min^{-1} at 60 s in pattern A (Fig. 2A), and from 78.1 beats•min^{-1} to 91.3 beats•min^{-1} at 30 s and 60 s in pattern B (Fig. 3A). After the offset of immersion, although the heart rate decreased to pre-immersion levels within 30 s in pattern A, it maintained the significant increase ($P<0.05$) at 30 s in pattern B.

In pattern A, CAP $(dP/dt)/P$ increased significantly from 17.1•s^{-1} to 18.0•s^{-1} ($P<0.01$) at 30 s of the immersion, and to 17.7•s^{-1} ($P<0.05$) at 30 s after the offset of immersion (Fig. 2B). In pattern B, by contrast, it decreased significantly from 17.2•s^{-1} to 15.9•s^{-1} ($P<0.01$) at 60 s of the immersion, and to 14.8•s^{-1} ($P<0.001$), 15.2•s^{-1} ($P<0.001$), and 15.8•s^{-1} ($P<0.01$) at 30 s, 60 s, and 90 s after the offset of immersion, respectively (Fig. 3B).

Discussion

CAP $(dP/dt)/P$, an index of sympathetic nerve activity, can be used as a noninvasive approach to the assessment of cardiac contractility even in moderate exercise (Ifuku et al. 1994). In particular, it increases steeply when the heart rate is greater than approximately 100 beats•min^{-1}, when significant leakage of noradrenaline from sympathetic nerve endings into the plasma begins (Christensen and Brandsborg 1973, Rowell 1984, Escourrou et al. 1984). In the present study, we compared the time course of changes in the CAP $(dP/dt)/P$ during the cold pressor test with a previous study which used muscle sympathetic nerve activity (Yamamoto et al. 1992).

Table 1. Responses of CAP $(dP/dt)/P$ and heart rate to the cold pressor test

		Cold water immersion		Recovery					
	Control	30 s	60 s	30 s	60 s	90 s	120 s	150 s	180 s
Pattern A									
Heart rate (beats/min)	61.9± 5.8	69.6± 6.7***	69.3± 6.7***	62.0± 7.1	61.5± 5.1	63.6±6.5	64.0±6.4	63.4±6.4	64.6±6.8*
CAP$(dP/dt)/P$ (/s)	17.1± 1.3	18.0± 0.8**	17.6± 1.0	17.7± 0.8*	17.2± 0.7	16.9±0.9	17.1±0.8	17.2±0.9	17.3±1.0
Pattern B									
Heart rate (beats/min)	78.1±11.4	91.3±15.3***	91.3±14.6***	84.7±12.6*	82.5±10.9	80.3±7.3	78.1±8.9	75.7±5.5	74.8±5.4
CAP$(dP/dt)/P$ (/s)	17.2± 2.3	16.7± 2.3	15.9± 1.8**	14.8± 1.4***	15.2± 1.3***	15.8±1.6**	16.3±2.1	16.3±1.8	16.4±1.8

Data are shown as mean ±SD. ***, **, * Significantly different from control values at rest: $P<0.001$, $P<0.01$, $P<0.05$, respectively.

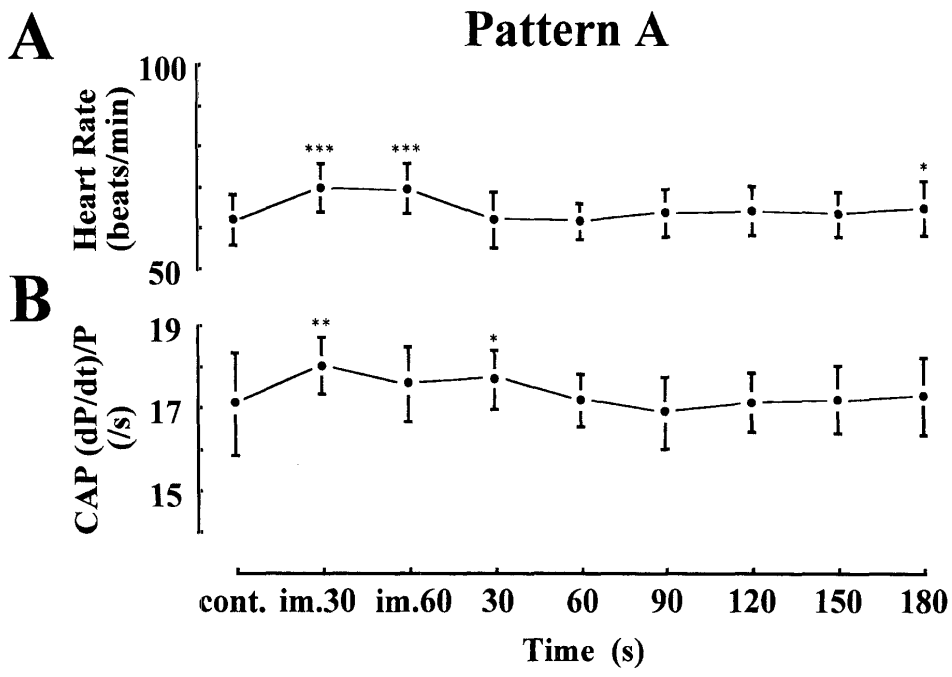

Fig. 2. Changes in heart rate (**A**) and CAP (dP/dt)/P (**B**) during the cold pressor test in pattern A. Heart rate and CAP (dP/dt)/P were increased by cold water immersion. *** $P<0.001$; ** $P<0.01$; * $P<0.05$.

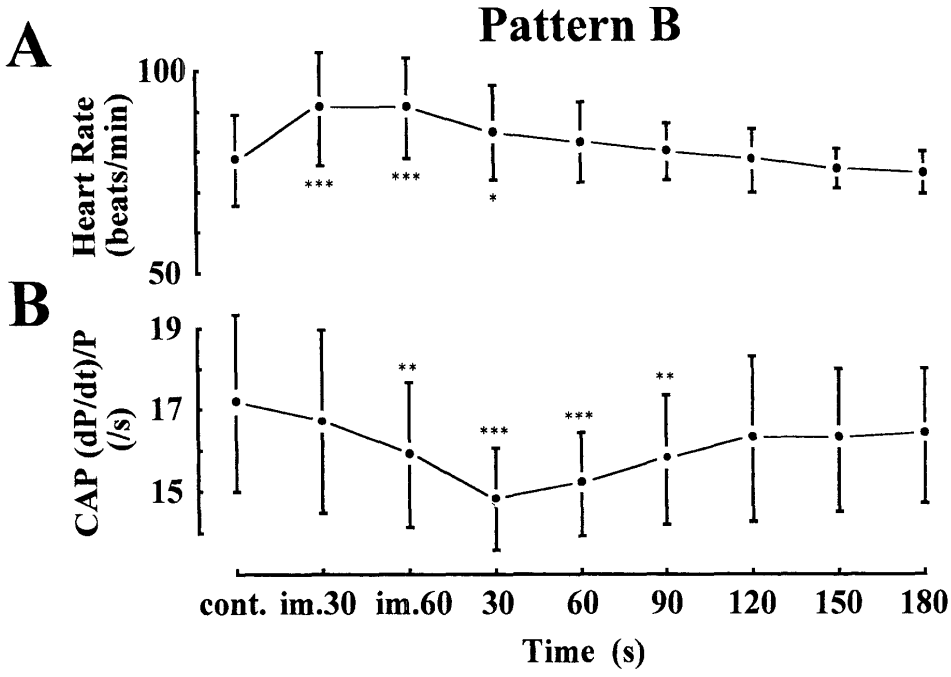

Fig. 3. Changes in heart rate (**A**) and CAP (dP/dt)/P (**B**) during the cold pressor test in pattern B. By cold water immersion, heart rate was increased, whereas CAP (dP/dt)/P was decreased. *** $P<0.001$; ** $P<0.01$; * $P<0.05$.

Muscle sympathetic nerve activity is activated by a decrease in blood pressure and is suppressed by a rise in blood pressure (WALLIN and NERHED 1982). During the cold pressor test, however, muscle sympathetic nerve activity is highly activated in spite of the increase in blood pressure. YAMAMOTO et al. (1992) reported that muscle sympathetic nerve activity remained unchanged during the initial period of 0-30 s of the cold pressor test and increased remarkably from 30-90 s. In the present study, changes in CAP $(dP/dt)/P$ showed a different time couse from that of muscle sympathetic nerve activity, in spite of a similar time couse for changes in heart rate. Firstly, they could be classified into two patterns : pattern A in which CAP $(dP/dt)/P$ was increased by cold water immersion, and pattern B in which it was decreased. Secondly, even in pattern A, the time course of change in CAP $(dP/dt)/P$ differed from that of muscle sympathetic nerve activity. CAP $(dP/dt)/P$ increased significantly at 30 s of cold water immersion and at 30 s after the offset of immersion, but it did not increase significantly at 60 s of the immersion. These findings suggest that CAP $(dP/dt)/P$ does not reflect the level of muscle sympathetic nerve activity during the cold pressor test. Since the activation of muscle sympathetic nerve activity plays an essential role in the increase in total peripheral resistance during the cold pressor test (YAMAMOTO et al. 1992), it might not contribute to the control of cardiac contractility.

Of the four subjects who participated in this study, the two of pattern A were athletes and the two of pattern B were non-athletes. It is known that parasympathetic nerve activity at rest in athletes is more dominant than that in non-athletes, training vagotony (ÅSTRAND and RYHMING 1954), which might cause a difference in the activation mode of CAP $(dP/dt)/P$ during the cold pressor test. In this study, in fact, the average value of heart rate at rest in the athletes was lower by 16.2 beats$\cdot$min^{-1}. This point remains to be studied.

In conclusion, the present study indicated that CAP $(dP/dt)/P$ did not reflect the level of muscle sympathetic nerve activity during the cold pressor test.

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