Cardiorespiratory Responses at the Onset of Chair Rotation in the Open- and Closed-eye Conditions

椅子回転直後における閉眼と開眼の呼吸循環応答の比較

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キーワード:第I相、視覚刺激、換気量、心拍数、血圧

Abstract

In the present study, we attempted to confirm whether or not cardiorespiratory response immediately after passive chair rotation is the same in the open- and closed-eye conditions in man. Inspiratory minute volume (VI), tidal volume (VT), respiratory frequency (f), heart rate (HR) and mean blood pressure (MBP) were determined by breath-by-breath and beat-by-beat techniques before, during and after rotation for a total of 45 sec. It was found that there are no significant differences in increment rate and peak attained time of VI, HR and MBP at the onset of chair rotation between open-eye and closed-eye conditions in both right-turn and left-turn. These results suggest that impulses from the visual system may be not contribute to ventilatory and cardiovascular responses at the onset of physical exercise with rotational movement for short time as applied here in healthy subjects.

要約

本研究の目的は、ヒトを対象に受動的椅子回転直後における呼吸循環応答が閉眼と開眼で同じであるか否かを確かめることである。椅子回転前、回転中および回転後計 45 秒間の毎分換気量、一回換気量、毎分呼吸数、毎分心拍数および平均血圧を一呼吸および一拍動毎に連続的に測定した。本実験では、右回転、左回転共に椅子回転開始直後の毎分換気量、毎分心拍数および平均血圧における増加率と最高値達成時間には、閉眼と開眼との間に有意差は認められなかった。これらの結果は、視覚系からの入力は本実験で行なったような短時間の回転動作を伴う身体運動開始時の換気・循環応答には影響を及ばさないことを示唆するものである。

Introduction

It is well known that pulmonary ventilation (VE) and heart rate (HR) increase immediately after physical exercise. This abrupt increase in VE and HR usually occurs from the first breath (Eldridge and Waldrop, 1991). The ventilatory profile in transition from rest to light or moderate intensity exercise is characterized by an abrupt step-like increment in ventilation without accompanying changes in alveolar partial pressures of O2 and CO2, and gas exchange ratio, and is termed phase I, as first defined by Whipp (1977). This phase I response, from the first breath and lasting for about 15s, is observed not only during voluntary exercise, but also passive movement following electrically induced muscle contractions or flexion-extensions of the lower legs with ropes (Whipp et al. 1982; Adams et al. 1987; Miyamura et al. 1992).

Nevertheless many investigators have pursued mechanisms that are responsible for the phase I response, and their opinions as to its nature are still a matter of dispute. Since the changes in ventilation are so rapid during the transition from rest to exercise, the phase I response cannot be explained by humoral agents because it appears to take at least about 10s for the metabolic substance from the exercising muscles to reach the arterial chemoreceptors. At present, it is considered that two neural mechanisms, central command and peripheral reflex, mainly trigger the increase in ventilation that appears at the onset of exercise (Mitchell 1990; Miyamura 1994; Mateika and Duffin 1995). Central command arises from the activation of the cerebral cortex and hypothalamus (Goodwin et al. 1972; Williamson et al. 2003) and peripheral reflex originates in the stimulation of group III and IV muscle afferents (McCloskey and Mitchell 1972; Kaufman et al. 1983), respectively. Recently, stimulation of the vestibular system has been reported to elicit cardiovascular and respiratory changes (Biaggioni et al. 1998; Yates and Miller 1998). In support this concept, Jauregui-Renaud et al. (2001) observed that stimulation of vertical semicircular canal increased breathing frequency in normal subjects but not vestibular-deficient patients. Monahan et al. (2002) also reported significant changes in respiratory frequency and minute ventilation from baseline during dynamic chair rotation for one minute. In our previous study, it was suggested that the activation of horizontal semicircular canals is one causal factor of ventilatory response at the onset of exercise with rotational movement in healthy subjects, but heart rate response is not (Miyamura et al. 2004). On the other hand, visual-vestibular interactions and vestibular-autonomic interactions have been the focus of much research, and a visual contribution has been shown in an animal model. Deficits in transitory tilt table responses after bilateral vestibular lesions are particularly large when animals are deprived for visual cues indicating position in space (Jian et al. 1999; Yates and Miller 1998). From these results, it is possible to hypothesize that abrupt increase in minute ventilation and heart rate immediately after exercise may be related to not only central command, afferent impulse from working muscle and vestibular sensory inputs, but also visual inputs which would be stimulated in various situations of sports and/or physical movements in daily life. To our knowledge, however, there are no data available concerning this hypothesis in healthy subjects.

The purpose of this study, therefore, was to clarify whether or not the visual afferents mediate the ventilatory and circulatory responses at the onset of passive chair rotation in man.

1 Methods

Eight healthy men volunteered to participate in the present study as subjects. No subjects had a history of cardiorespiratory diseases, took medications that seriously affected cardiorespiratory responses and smoked. Mean and standard errors of age, height and weight of the subjects were 27.0 (1.92) years, 171.6 (1.20) cm, and 63.1 (2.40) kg, respectively. The subjects were informed of the experimental protocol and possible risks involved in this study before giving written consent. The present study was approved by the Human Research Committee of the Research Center of Health, Physical Fitness and Sports of Nagoya University.

Subjects were asked to refrain from performing vigorous exercise for 24 h prior to actual experiments. The vestibular stimulus tests were carried out on each subject using a rotation chair in both open- and closed-eye conditions, i.e., during experiment, subjects sat with their backs against an experimental chair. The subject's torso and head were securely fastened in a swivel chair with belt. The subjects were rested comfortably on the chair in sitting position for 20 min, and then asked to relax during experimental periods. The rotation chair can be swiveled through 360 degrees about a central axis by the experimenters. The rotation chair was turned manually by experimenter for 180° in mean $1.5 (1.4 \sim 1.6)$ sec from right to left or the reverse (Fig. 1).

A rotation will be denoted as "right-turn" and "left-turn". The rotation tests was carried out totally 12 times for each subject, i.e., the right-turn and the left-turn were



Ventilatory response (breath-by-breath)

VI, PETO₂, PETCO₂

Circulatory response (beat-by-beat)

HR, SBP, DBP, MBP

Figure 1. Schematic diagram of experimental set-up.

repeated randomly three times at approximately a few minutes intervals in both "openeye" and "closed-eye" conditions, respectively. These rotating tests were initiated just before the start of the inspiratory phase; the experimenter checked this on an oscilloscope which connected to the hot-wire flow meter (RF-H, Minato Ikagaku, Japan). The goniometer was attached to the axis of the rotation chair in order to detect the start of turning. Inspiratory minute volume (VI), tidal volume (VT), inspiratory periods, expiratory periods, and partial pressures of end-tidal carbon dioxide and oxygen (PETCO2 and PETO2) were determined by the breath-by-breath technique before, during and after turning for a total of 45 sec, i.e., VI and VT were measured continuously for 30 sec before, 1.5 sec during, and 13.5 sec after chair rotation, respectively. The subject breathed through a respiratory face mask attached hot-wire flow meter. It was calibrated prior to each experiment using a 2-liter calibration pump at different flow rates. The dead space of the respiratory face mask was about 100 ml. Respiratory gases were sampled using a thin vinyl tube (inner diameter 1 mm) inserted into the face mask, with tip being positioned as close to each subject's mouth as possible. Respiratory frequency (f) was calculated from the total respiratory time, and VI was obtained as the product of VT and f. PETCO2 and PETO2 were calculated from end-tidal CO2 % and O2 %, which were obtained by analyzing gas samples being drawn continuously through the vinyl tube with the use of a gas analyzer (Minato Ikagaku, MG-360, Japan).

As for circulatory responses, on the other hand, heart rate (HR) and blood pressure (BP) were monitored beat-to-beat before, during and after rotation for a total of 45s. HR was calculated beat to beat from R spike using an electrocardiogram through a bioamplifier (model AB-621G, Nihon Kohden). BP was also measured noninvasively beat

to beat by using photoplethysmographic method (Finapres). The sensor of Finapres was attached to the middle finger of non-dominant hand in order to record blood pressure, and the hand being kept at heart level. The instaneous pressure output was transferred on-line to the Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were determined by the Finpres's signal, and mean blood pressure (MBP) was calculated from SBP and DBP using equation (MBP = 1/3 (SBP-DBP) + DBP).

Measured values were expressed with means and standard deviations (\pm SD) or standard errors (\pm SE). The relative changes of the parameters in the open-eye and closed-eye were analyzed of variance with repeated measurements. If a significant F ratio (p<0.05) was obtained, Dunnett's test was used to determine when the differences during the turn occurred. In comparing open-eye with closed-eye, the Wilcoxon test was used. The level of significance was set at 0.05.

2 Results

Since the values at rest of VI, and HR and BP were different in the two turns, we compared the differences in ventilatory and circulatory responses at the onset in the open-eye and closed-eye by the Δ value estimated from a mean value of rest for 30 sec of 100% as described previously. Fig. 2 shows the results of ventilatory and circulatory responses during and after chair rotation of left-turn and right-turn in the open-eye

and closed-eye conditions with respect to $\triangle VI$, $\triangle HR$ and $\triangle MBP$. $\triangle VI$ increased significantly (p<0.05) immediately after rotation in both right-turn and left-turn. Mean

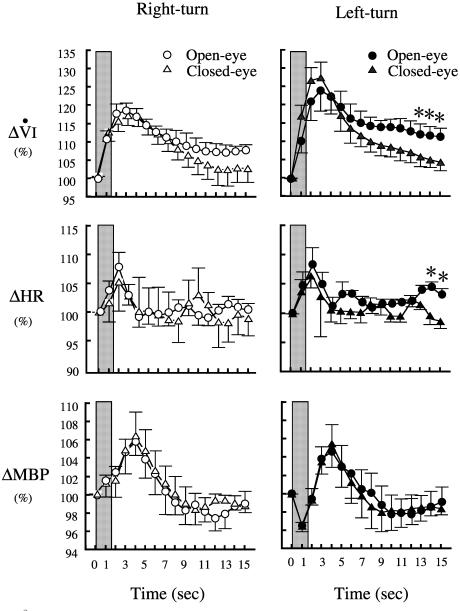


Figure 2.

The percentage changes in inspiratory minute volume (\triangle VI), heart rate (\triangle HR) and mean blood pressure (\triangle MBP) during and after chair rotation of right-turn (left panel) and left-turn (right panel) in the open-eye (circles) and closed-eye (triangles) conditions when the average of the values at rest was 100%. The turn period is shown with shade area. Time 0 indicates the onset of rotation. Values are expressed as mean (\pm SE). *significant difference (p<0.05) between open-eye and closed-eye.

values (\pm SD) of peak value of \triangle VI were 121.7 \pm 8.0% for the open-eye and 120.3 \pm 8.2% for the closed-eye in the right-turn, and 127.4 \pm 15.7% for the open-eye and 130.4 \pm 13.3 % for the closed-eye in the left-turn, respectively. There are no significant differences in the \triangle VI between open-eye and closed-eye, except at 13~15 sec. Mean values (\pm SD) of peak value of \triangle HR were 108.0 \pm 6.7% for the open-eye and 107.5 \pm 5.6% for the closed-eye in the right-turn, and 109.5 \pm 8.4% for the open-eye and 107.8 \pm 5.8% for the closed-eye in the left-turn, respectively. There are also no significant differences in the \triangle HR between open-eye and closed-eye, except at 14 and 15 sec. Furthermore, mean values (\pm SD) of peak value of \triangle MBP were 106.1 \pm 4.7% for the open-eye and 106.4 \pm 7.8% for the closed-eye in the right-turn, and 105.2 \pm 5.9% for the open-eye and 105.6 \pm 6.0% for the closed-eye in the left-turn, respectively. No significant difference was found in the peak values of \triangle MBP between open-eye and closed-eye condition, while \triangle MBP increased at 1 sec after rotation of right-turn and \triangle MBP decreased at 1 sec after rotation of left-turn as shown in Fig. 2.

On the other hand, peak attained times of $\mathring{V}I$, HR and MBP were indicated in Fig. 3. Mean values of the peak attained time of $\mathring{V}I$, HR and MBP in the open-eye were 3.4 (2.5), 2.1 (0.9) and 4.0 (0.6) sec for the right-turn and 4.6 (3.5), 2.4 (0.8) and 4.1 (1.1) sec for the left-turn, respectively. Mean values of the peak attained time of $\mathring{V}I$, HR and MBP in the closed-eye were 4.3 (4.0), 2.6 (1.0) and 4.0 (0.0) sec for the right-turn and 2.7

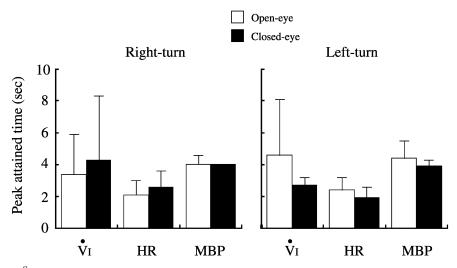


Figure 3. Open-eye (white bar) — closed-eye (black bar) comparison of peak attained time as an index of the kinetics of \dot{V}_I , HR and MBP in the right-turn (left panel) and left-turn (right panel). Values are expressed as mean (\pm SE).

(0.5), 1.9 (0.7) and 3.9 (0.4) sec for the left-turn, respectively. There are no significant differences in peak attained time of $\overset{\bullet}{V}$ I, HR and MBP between open-eye and closed-eye conditions as shown in Fig. 3.

3 Discussion

In the present study, we attempted to confirm whether or not ventilattory and circulatory responses at the onset of passive chair rotation are the same in both openeye and closed-eye conditions in healthy subjects. It was found that there are no significant difference in the peak value of $\triangle VI$, $\triangle HR$ and $\triangle MBP$ between open-eye and closed-eye conditions. Also no significant difference in the peak attained times of VI, VI, VI and VI and VI between open-eye and closed-eye was found as shown in VI Figs. 2 and 3. We believe that this is the first study showing a effect of the visual stimuli to ventilatory and circulatory responses at the onset of passive rotation in humans.

When physical exercise starts, various cardiorespiratory adjustments take place for accommodating the greatly increased metabolic requirements. In particular, the transition from rest to light or moderate intensity exercise is typically accompanied by an abrupt in ventilation and heart rate at the first breath. The initial rapid increase in ventilation appearing at the onset of exercise has been referred to as phase I. (Whipp 1977). This phase I is observed during not only voluntary exercise and passive movement, but also during electrically induced muscle contraction. Why is the increasing pulmonary ventilation elicited so quickly just at the onset of exercise? Nevertheless many investigators have pursued the mechanisms of this phase I response and their opinions as to its nature are still a matter of dispute. Since the changes in pulmonary ventilation are so rapid immediately after exercise, phase I response cannot be explained by humoral agents because of the delay in transport. It has hitherto been reported that the causal factor of phase I are classified largely into three, i.e., central and peripheral neurogenic stimuli, or both (Miyamura 1994; Williamson et al. 2003).

Recently, Monahan et al. (2002) have determined cardiorespiratory parameters such as inspiratory time, expiratory time, ventilation, heart rate, and mean blood pressure during various seven (dynamic upright pitch, dynamic lateral pitch, dynamic head roll, dynamic yaw, dynamic chair rotation, static head down rotation and static head rotation in lateral decubitus position) conditions in order to confirm the hypothesis that activation of the semicircular canals would increase respiration in humans. Since

significant changes in inspiratory time, expiratory time and minute ventilation from baseline during dynamic chair rotation with 15 cycle/min were observed, they suggested that semicircular canal, but not otolith organs or neck muscle afferents, mediate increased ventilation in humans and support the concept that vestibular activation alters respiration in humans. However, because respiration was measured continuously for one minute in their experiments but not for 15 sec as applied here, it is unclear whether this horizontal vestibular activation produced functional alterations in phase I, i.e., phase I response is considered to last variously for 10 to 20 sec among different investigators (Miyamura 1994). We have chosen the ventilatory response to be defined as rapid change in ventilation occurring within 15 sec, in which chemical substances may have not reached the peripheral chemoreceptors. In other words, it is important to emphasize that ventilatory and heart rate responses should be determined continuously with breath-bybreath and beat-by-beat techniques during within 15 sec rather than totally one minute in order to clarify whether horizontal semicircular canal stimulus is related to ventilatory response (phase I) at the onset of exercise. In our previous study, we attempted to confirm whether pulmonary ventilation and heart rate increase at the onset of passive chair rotation, which stimulates horizontal semicircular canals, in healthy subjects. As results, VI increased significantly immediately after passive chair rotation, while HR unchanged at the onset of chair rotation in both left- and rightturns. These results suggest that activation of horizontal semicircular canal is one of the causal factor of ventilatory response at the onset of exercise with rotational movement, but not heart rate response, in human subjects (Miyamura et al. 2004).

On the other hand, visual-vestibular interaction and vestibular-autonomic interactions have been the focus of much research, and a visual contribution has been shown in an animal model (Yates and Miller 1998). It has hitherto been reported that visual cues may be important in regulating blood pressure during movement in the cat, whereas a previous study suggested that visual information does not trigger changes in sympathetic out flow in humans (Shortt and Ray 1997). Wood et al. (2000) have determined whether visually induced changes in perceived orientation trigger transitory autonomic reflexes in humans. They observed that several subjects showed significant transient decreases in mean blood pressure resembling their initial response to passive head-up tilt using a mirror-bed device or a dome projector, while significant changes in cardio-respiratory parameters to illusory tilts could not be demonstrated for the entire

group. Aoki et al. (2000) also reported that rapid roll tilt provoked pressor response that one might expect as an arousal-readiness response to significant spatial reorientation, whereas with vection, some subjects BP increased (type I) and others' BP decreased (type II). To our knowledge, however, the role of visual stimulus in cardiorespiratory response at the onset of exercise has not been demonstrated in human subjects. In the present study, therefore, we attempted to confirm whether ventilatory and circulatory responses at the onset of passive chair rotation are the same in both open-eye and closed-eye conditions in healthy subjects as described previously. It was found that there are no significant difference in the peak value of $\triangle VI$, $\triangle HR$ and $\triangle MBP$ between open-eye and closed-eye conditions. Furthermore, no significant differences in the peak attained time of VI, HR and MBP between open-eye and closed-eye was found (Figs. 2 and 3). These results suggest that visual stimuli is not sufficient to significantly alter cardiorespiratory responses at the onset of passive movement accompany with rotation for short time as applied here. This suggestion is partly supported by report of Peters et al. (2000) who observed that horizontal optokinetic stimulation by means of drum does not significantly affect SBP, DBP or pulse in human. The lack of consistent changes between open-eye and closed-eye conditions may be due to the nature of the stimuli and individual variability in visual cardiorespiratory responses.

Another interesting result of the present study was that mean blood pressure (MBP) increased at 1 sec immediately after rotation of right-turn in both open-eye and closed-eye conditions, whereas it was decreased in the left-turn as shown in Fig. 2. At present, we cannot explain based on the physiological ground why changes of mean blood pressure at the 1 sec after rotation in the left-turn was in a strikingly contrast to right-turn. It is possible to assume that venous return to the heart is related to the direction of body rotation, i.e., blood pressure may be decrease temporarily by the decrement of venous return in the left-turn because heart is locating in left side. However, it will be necessary to conduct further investigation about this assumption.

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