3B.5 TO THE MECHANISM OF PHYSIOLOGICAL EFFECTS OF ATMOSPHERIC PRESSURE FLUCTUATIONS

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1. INTRODUCTION

Natural fluctuations of air pressure (FAP) of very low frequencies are considered as a potential causal agent of health effects of the weather on humans (Richner, 1983; Delyukov and Didyk, 1999). FAP originate in the natural atmosphere mostly from turbulence and convection and get stronger on days with rough weather (Gossard and Hooke, 1975). Some part of FAP is generated by solar wind particles and short-wave solar radiation thus depending on solar activity. The other sources for FAP are atmospheric turbulence and orographic influences on airflows. FAP amplitudes at ground level reach tens, sometimes hundreds Pascals with a main contribution from extra-low frequencies below 1 Hz. The mechanism of physiological effects of FAP was not clarified so far. This study examines the effects of artificially generated oscillations of air pressure (OAP), simulating FAP, on the regulation of the cardiovascular system in healthy volunteers.

2. METHODS

OAP was created in a chamber by an electronically controlled ventilation unit. Periodic OAP close to sinusoid was used with a period of 30 s or 45 s and an amplitude of 50 Pa. Control tests were carried out with the same subjects and the same setting but without OAP exposure. OAP effects on endpoints characterizing central haemodynamics were studied in 10 healthy women (20-21 years of age) in a supine position. Systolic and diastolic blood pressure was measured by Korotkov's method.

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Cardiac output was determined by a tetrapolar chest rheography technique. The test lasted about 30 min; the subject was exposed to OAP (30 s, 50 Pa) for 20 minutes. OAP effects on heart rate time structure were studied in 10 healthy subjects (4 men and 6 women aged 21 to 64) in a supine position. Each test lasted 75 min, and the subject was exposed to OAP (45 s, 50 Pa) for 45 minutes. During the entire test consecutive inter-beat cardiac intervals (*R-R* intervals of electrocardiogram) were recorded. Components synchronous with OAP (fundamental tone and its harmonics) were extracted from the sequence of *R-R* intervals by cosinor (least squares) technique, superposing epochs and spectral filtering.

3. RESULTS

On average, exposure to OAP (30 s, 50 Pa) caused a reduction in systolic blood pressure of 7% ($P < 0.05$) compared to control. At the same time, cardiac output increased by 10 % ($P < 0.05$). These changes were attributed to a reduction (≈ 11%) in the peripheral vascular resistance.

Heart rate in all the subjects showed a coherent response to periodic OAP. Oscillation of *R-R*, repeating OAP, was found with an amplitude of 3-15 ms ($P < 0.001$). This fundamental component had a phase roughly opposite to that of OAP. Besides, sequences of *R-R* intervals in 5 subjects regularly showed a steady fine structure synchronized with OAP and significant at level $P < 0.001$ or $P < 0.0001$. That structure, in addition to the fundamental component, also included oscillation with a period of about 10 s representing a so-called baroreceptor oscillation of *R-R*, generated spontaneously by the blood pressure control system. No similar steady structure arose in the absence of OAP. Apparently, OAP synchronized the baroreceptor oscillation in those 5 subjects.

4. DISCUSSION

Synchronizing effects of OAP on heart rate as well as its effect on the smooth muscle of peripheral vessels indicate that OAP is perceived by baroreceptors of the baroreflex loop. The baroreceptors are located in the walls of aortal arch and carotid sinus. They are stretch detectors sensitive to a difference in pressure inside and outside the vessel. The baroreceptors together with their afferent pathways are part of a feedback contour comprising also the cardiovascular center in *medulla oblongata* and smooth muscle of peripheral arteries. This contour known also as a baroreflex loop stabilizes arterial pressure at a prescribed level. Besides, nervous centers of the *medulla oblongata* use baroreceptor impulses to form parasympathetic signals slowing down heart rate as the baroreceptor signals increase.

Perturbations of arterial pressure, caused e.g. by changes in pose, modify the character of baroreceptor impulsation. As a result, a signal is generated in the *medulla oblongata*, causing vascular smooth muscle to change arteriolar cross-section in order to return arterial pressure back to normal level. Like any system with a feedback, stabilizing given parameter, the baroreflex loop shows spontaneous oscillation of arterial pressure with a period of about 10 s, called baroreceptor oscillation. Heart rate, responding to periodic changes in the baroreceptor signal, reveals an analogous oscillation.

Our basic idea is that the baroreceptors of the blood pressure control system are not indifferent to changes in external air pressure. The ambient air presses immediately two carotid sinuses located symmetrically just underneath the skin of the neck. Also, impact of air pressure in the lungs on the walls of the aortic arch through intrapleural pressure cannot be excluded (Sayers, 1973), although this way seems to be less efficient. Anyway, variations in the external air pressure sway the reference point of the baroreceptors, thereby feeding a false signal into the baroreflex loop.

If the external pressure oscillates, e.g. OAP is applied, an additional alternating signal appears in the baroreflex loop that modifies its spontaneous oscillation and causes *R-R* interval and arterial pressure to oscillate. A phase shift as against to OAP may be expected to be about zero for arterial pressure and about π for *R-R* interval. Typical values of baroreflex sensitivity are 2 to 13 ms/mm Hg (Kollai et al., 1994), therefore OAP with the amplitude of 50 Pa should vary the *R-R* interval within 10 ms. Such a value of constrained oscillation is comparable with the amplitude of spontaneous fluctuations in the baroreflex loop and must be essential for its functioning. From this viewpoint, changes in peripheral resistance and corresponding changes in arterial pressure and cardiac output under exposure to OAP may result from gradual changes in the functional state of nervous centers controlling the set point of arterial pressure.

It must be noted that the baroreceptor impulses do not only reach the cardiovascular center in *medulla oblongata*. They proceed further up to the hypothalamus where they cause changes in the activity of many neurons and influence homeostasis (Thomas and Calaresu, 1972). Baroreceptor signals, reaching nervous centers in hypothalamus and, probably, brain-cortex, participate in the formation of emotional state and working capacity (Bard, 1928). This also can explain the activating effect of OAP on the mental process, reported earlier (Delyukov and Didyk, 1999). The mechanism considered may provide a link between symptoms in weather sensitive persons (the so called Atmosphere Related Syndrome) and changes in weather, which are usually accompanied by changes in the FAP pattern.

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