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*Effects of Dehydration, Physical Conditioning  
and Heat Acclimatization on the  
Response to Passive Tilting*

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ABSTRACT

BEETHAM, W. P., JR. AND E. R. BUSKIRK. *Effects of dehydration, physical conditioning and heat acclimatization on the response to passive tilting.* J. Appl. Physiol. 13(3): 465-468. 1958.—The effects of dehydration on the response to passive tilting have been studied in 15 young men after physical conditioning, with and without heat acclimatization. Dehydration was associated with the following modifications of the 'normal' orthostatic response: an essentially unchanged systolic pressure, a consistent rise in diastolic pressure and a moderate decrease in pulse pressure. The pulse rate increased more rapidly with time in the upright position after dehydration than when hydrated. Physical conditioning, either with or without heat acclimatization, produced no apparent improvement in the pulse rate or blood pressure response to passive tilting after dehydration.

HEMORRHAGE, venous occlusion of the extremities and the assumption of the erect posture (passive tilting) are known to reduce effective circulating blood volume. The cardiovascular response to these three situations has been well studied. Another common cause of reduction in blood volume is dehydration. However, we have been unable to find any studies of the effects of dehydration on the cardiovascular response to orthostasis. This paper reports the effects of dehydration on cardiovascular responses to passive tilting in healthy, young men. Since both physical conditioning and heat acclimatization are manifested by improved cardiovascular responses to work (1-3), it was also of interest to determine if these treatments would improve the cardiovascular response to passive tilting after dehydration. Related data on work performance after dehydration were presented in an earlier issue of this journal (4, 5).

PROCEDURES AND METHODS

Fifteen healthy, young men (1 subject in the sedentary group was unable to complete

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the study) were dehydrated overnight in a hot, dry atmosphere (115°F, 20% R.H.) on two occasions (D<sub>1</sub> and D<sub>2</sub>). When they had lost 5% of their initial body weight, the subjects were moved to a comfortable chamber (78°F, 50% R.H.) where they were allowed to sleep or rest for 2-4 hours. In every case, enough time was spent in the comfortable environment to dissipate the heat accumulated during dehydration (as evidenced by return of rectal temperature to normal levels). The subjects were divided into three equal groups: one group, S, lived under a relatively sedentary schedule, another group, C, underwent a rigorous program of physical conditioning and the third group, AC, was acclimatized to heat in addition to physical conditioning. Group AC spent 4 hours a day doing intermittent work (alternately walking on a treadmill 30 minutes and resting 30 minutes) in an ambient temperature of 120°F. The details of the procedures of physical conditioning and heat acclimatization are reported elsewhere (4). There was a 3-week period between the first and second dehydration. Measurements of pulse rate and blood pressure responses to passive tilting were made in the morning before breakfast.

Blood pressure was measured by auscultation using a mercury manometer. The diastolic reading was taken at the disappearance of sound. Pulse rate was determined by palpation. Position was always changed passively by means of a tilt table and postural sway was reduced by body straps. The 'erect' position was  $70^\circ$  (horizontal =  $0^\circ$ ) with the weight partially supported by the legs (feet placed on a footrest). Position was changed from erect ( $70^\circ$ ) to horizontal ( $0^\circ$ ); after a 4-minute interval, the subject was returned to the  $70^\circ$  erect position and measurements were continued for another 4 minutes. The heart rate and blood pressure were measured simultaneously for each position at 30 seconds, 1, 2, 3 and 4 minutes. The results obtained on  $D_1$  and  $D_2$  were compared with control values obtained prior to  $D_1$  using analysis of variance. Analysis of specific differences was carried out with a sign test and/or a multiple comparisons test (6), where appropriate.

#### RESULTS

Figure 1 shows the mean blood pressures and pulse rates before and after acute dehydration in 14 subjects who were subjected to passive tilting. The dehydration values represent the mean for both  $D_1$  and  $D_2$ . The values for  $D_1$  and  $D_2$  were pooled, since no major differences in pulse or blood pressure response to tilting were detected between  $D_1$  and  $D_2$ , using a multiple comparisons test with a 95% protection level (6). In addition, there were no significant differences between groups  $S$ ,  $C$  and  $AC$  at  $D_2$ , so the results of the three groups were also pooled. Table 1 gives the means and standard deviations for all cardiovascular measurements before and after dehydration.

A significant difference between the mean pulse rates with conditions, i.e. predehydration, dehydration, was found for the initial erect position ( $70^\circ$  from horizontal). A significant difference between conditions was also found, by 1 minute, after assuming the supine position from the erect position. This difference persisted for as long as the subject remained supine and was due to a larger decrease in pulse rate after dehydration than in the control condition (predehydration). However, there was no significant difference

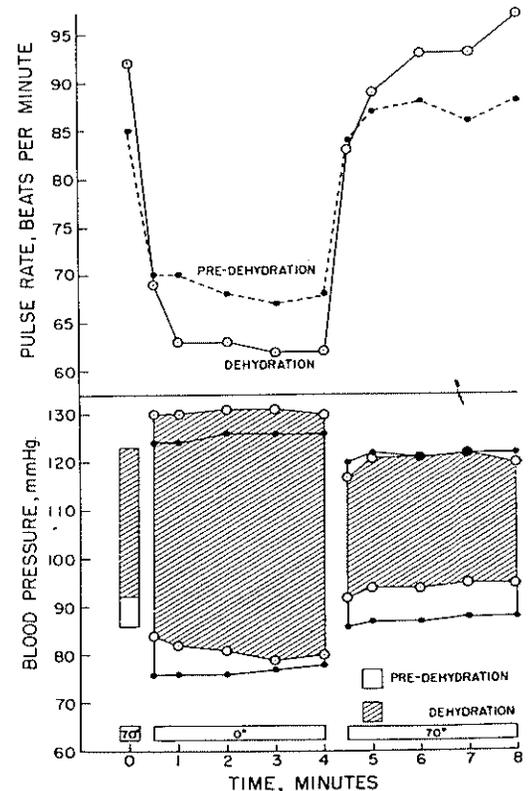


FIG. 1. Cardiovascular responses to passive tilting before and after dehydration. Blood pressures and pulse rates represent mean values for all subjects. Erect position is  $70^\circ$  from horizontal and supine position equals  $0^\circ$  horizontal.

between the mean pulse rates for conditions (predehydration, dehydration) with orthostasis ( $70^\circ$  from horizontal) at 30 seconds, 1, 2, 3 or 4 minutes, although a near significant difference was found for the 4-minute values. An elevation in pulse between 30 seconds and 4 minutes in the erect position occurred in all 14 subjects at  $D_1$  and in 13 of 14 at  $D_2$ ; whereas only 10 of 14 (1, no change) displayed the same trend under control conditions. The mean pulse rate increased 4 beats per minute after 4 minutes of standing before dehydration, whereas the same measurement after dehydration showed an increase of 14 beats per minute.

Dehydration had little effect on the systolic pressure in any position after 1 minute of adjustment to position. There was no sig-

TABLE I. CARDIOVASCULAR RESPONSES TO TILTING BEFORE AND AFTER DEHYDRATION\*

	Initial 70° Tilt	4' Tilt 0°	30" Tilt 70°	4' Tilt 70°
<i>Systolic Pressure, mm Hg</i>				
C	122.8 ± 10.4	125.6 ± 11.1	119.9 ± 7.3	121.9 ± 9.6
D	123.2 ± 8.4	130.2 ± 8.7	116.8 ± 8.6	120.8 ± 10.3
<i>Diastolic Pressure, mm Hg</i>				
C	85.5 ± 6.7	77.9 ± 7.4	86.3 ± 6.4	87.8 ± 5.7
D	92.3 ± 6.1	79.8 ± 10.3	92.6 ± 4.9	95.7 ± 6.8
<i>Pulse, Beats per Min.</i>				
C	84.8 ± 9.3	68.2 ± 11.1	83.6 ± 10.1	88.1 ± 10.8
D	92.5 ± 11.0	62.5 ± 9.2	83.0 ± 11.3	97.2 ± 20.8
<i>Pulse Pressure, mm Hg</i>				
C	37.3 ± 6.3	47.8 ± 7.1	33.6 ± 4.4	34.1 ± 6.3
D	30.0 ± 7.2	50.5 ± 10.7	25.9 ± 7.5	25.8 ± 7.1

\* See text for explanation of headings of columns.

C = Control. D = Dehydration.

nificant difference in systolic pressure between conditions (predehydration, dehydration).

The usual elevation in diastolic pressure occurred with orthostasis, which resulted in a decrease in pulse pressure since the systolic pressure remained relatively constant. An additional rise in diastolic pressure occurred with orthostasis when the subjects were dehydrated. This conclusion was drawn from the fact that analysis of variance indicated a significant difference between conditions ( $P = <.01$ ) with the change in diastolic pressure as the variable. Subsequent analysis, using the multiple comparisons test, indicated that the control values were significantly different from D<sub>1</sub> and D<sub>2</sub>, while D<sub>1</sub> and D<sub>2</sub> did not differ from each other. Similar analysis of the data on pulse pressure revealed the usual decrease in pulse pressure with orthostasis. Dehydration augmented this effect.

Fainting and related symptoms did not occur in the erect position when control measurements (predehydration) were made. However, two of fourteen subjects in each dehydration period became apprehensive, pale and faint. These symptoms were accompanied by tachycardia and a slight fall in the mean blood pressure. The condition of these subjects resembled the peripheral type of circulatory failure seen in mild heat exhaustion.

#### DISCUSSION

Previous work (7-9) on cardiovascular responses to dehydration and orthostasis in-

dicates that acute dehydration is associated with a primary decrement in the circulating blood volume to which the vascular bed adjusts and that orthostasis results in a primary change in the vascular bed which alters the distribution of blood throughout the body. Therefore, both dehydration and orthostasis result in a reduction of the effective circulating blood volume, although by different mechanisms.

Although blood volume immediately after dehydration was not measured, the reduction in effective circulating blood volume in the present study may be roughly estimated. A loss of 5% of the body weight by dehydration may be equivalent to a 10% decrement in effective blood volume. This estimate is based on the work of Adolph, who demonstrated that in acutely dehydrated men in the heat, the plasma volume diminished disproportionately relative to other fluid compartments (2.5-fold) (9). Furthermore, it has been shown that orthostasis similar to the 70° passive tilt may result in the pooling of as much as 600-700 cc of blood in the legs alone, thus reducing the effective blood volume approximately 10% (10, 11). Although the combined effect of orthostasis and dehydration may not reduce blood volume in an additive fashion, it is reasonable to expect that their combined effect reduces blood volume more than either one alone.

Although a loss in effective blood volume of somewhat greater than 10% cannot be considered large enough to induce large consistent changes in heart rate or systemic arterial pressure (8, 12), certain small but significant changes in these measurements were observed when the effects of dehydration were superimposed on the normal orthostatic responses. The higher pulse rate and the increased tendency to faint, associated with orthostasis after dehydration, indicate that venous return is inadequate. It is possible that reflex changes in venous tone could play a major role in the adjustment of the vascular bed to a moderate decrease in blood volume (13-15). However, for the maintenance of circulation to vital areas during the short period of time (4 minutes) in the erect position, the key reflex is no doubt constriction on the arterial side, particularly in the legs. Any

changes in venous tone at 4 minutes in the erect position probably would have made little difference, since after dehydration there might have been too little circulating fluid in the venous side for venoconstriction to have an effect.

In this study we have reduced the blood volume by dehydration and have found that the cardiovascular response to orthostasis produces a consistent pattern which is not affected by physical conditioning, with or without heat acclimatization. The passive tilting procedure used could well be classified as a relatively 'low grade stress.' If repeated tilting or prolonged orthostasis had been used, it is possible that the above treatments might have altered the pulse rate and blood pressure response.

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