THE YALE JOURNAL OF BIOLOGY AND MEDICINE 59 (1986), 257-265

The Cardiac Filling Pressure Following Exercise and Thermal Stress

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Received September 15, 1985

Under heat stress, a decrease of the central venous pressure (CVP) was regularly observed, raising the question of whether this reaction is a limiting factor for the circulation.

In animal experiments it could be shown, however, that despite a lowered CVP, which depended on the elevated body temperatures, a high cardiac output (CO), as well as an elevated stroke volume could be maintained. A low CVP went hand in hand with a low total peripheral resistance. It was argued that under these circumstances the low CVP was not limiting because the intrinsic factors of the heart (sympathetic stimulation) were capable of maintaining a high CO.

In human experiments the lowered CVP had to be seen in relation to the degree of dehydration. Regardless of whether the plasma volume remained constant, as in exercise, or declined, as in thermal stress (sauna), the CVP followed the volume depletion of the vascular and extravascular space, and it might well be that under these circumstances CVP is limiting. In this case, however, the altered CVP must be seen first as a monitor for the fluid deficit and not as a factor controlling cardiac function.

INTRODUCTION

Under thermal load, blood flow and blood volume divert from the body core to the skin, favoring heat loss [24,26,27,28,29,32]. The translocation of blood from the intrathoracic compartment toward the periphery is necessarily followed by a lowered cardiac filling pressure, raising the question of whether this lowered filling pressure is a limiting factor in adjusting to heat stress. The question as it stands tacitly implies that cardiac function under these circumstances is strictly dependent on the Frank-Starling mechanism, with cardiac output (CO) going up when central venous pressure (CVP) goes up and vice versa. Conversely, it is known that under heat stress in healthy subjects, a high CO can be maintained despite a lowered CVP [2,12,23,29]. Obviously the question raised cannot be answered simply with "yes" or "no." Before an answer can be given, the term "cardiac filling pressure" must be defined more precisely.

THE NATURE OF THE CARDIAC FILLING PRESSURE

The term "cardiac filling pressure" usually means the pressure measured in the right atrium or close to it. Pressures measured in this location are also referred to as CVP. In this paper, both terms will be used synonymously.

The right atrium is a part of the so-called "low pressure system" (LPS) of the circulation, comprising post-capillary vessels, lung circulation, the right atrium and ventricle, the left atrium, and the left ventricle in diastole. All these parts normally form a functional unit, because pressure changes occurring in one part are transmitted

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to other parts of the system [7]. Gauer, Henry, and associates have worked out this concept of the LPS and validated it in many experiments [4,5,6,7].

The term "cardiac filling pressure" is preferentially used looking downstream toward the heart. Guyton and his school have shown that under their experimental conditions the level of the CVP determines cardiac function, and, conversely, atrial pressure is dependent on cardiac function [8,9]. Within this frame of thinking, Rothe has recently outlined this very useful concept in several review articles [21,22]. The right atrial pressure is a function of the amount of blood returned to the heart and the pumping ability of the heart [8].

If, however, one turns around and looks upstream one becomes aware that the right atrium sits in a key position of the circulation. From this point one can look outside to the extrathoracic parts of the LPS, and get information about the filling state of the circulation, including both the intra- and extravascular parts [7,15,16,17]. Furthermore, the level of the CVP reflects peripheral characteristics of the vascular bed such as vascular tone, which is particularly important under heat stress [3,6,11,25].

The double-faced nature of this parameter becomes obvious if the dimensions are compared. The information about the volume is given in cm^3, while the information about the cardiac function, like the CO, is given in cm^3 per time. Since under heat stress volume changes of both the intra- and extravascular bed as well as changes of the cardiac function can occur [1,2,17,18,30,31,33], we must define the dimensions.

Results will be reported from two kinds of studies. In the first series of experiments, CVP changes were viewed looking downstream toward the central circulation, after raising the body temperature in dogs. In the second series, CVP was observed in healthy young men after changing the hydration level by thermal stress or exercise; in these experiments we looked upstream.

METHODS

The experiments were performed in five adult female dogs weighing between 13 and 20 kg. The animals were splenectomized and a right carotid loop was made. After a wound healing period of at least three weeks the experiments began. The animals were run on a motor-driven horizontal treadmill for 30 minutes. The exercise was continued until the body weight (BW) had decreased by at least 3 percent. The running speed was adjusted to the animal's individual ability, varying between 8 and 15 km/hour. The animals ran for two hours, covering distances between 15 and 40 km. The environmental temperature was 16°C, because it was estimated from the work of Hammel et al. [10] that this was close to the neutral zone of dogs during exercise. The animals had no access to water during exercise and afterward during the measurements.

The control measurements and the after-exercise measurements were repeated at weekly intervals so that two experiments from each animal were obtained.

During the measurements the animals lay conscious, quiet, and relaxed on their left sides. No sedatives were given. The carotid loop was punctured with a cannula through which a catheter for arterial blood pressure (ABP) measurements or a thermistor probe for CO measurements was introduced into the carotid arch. The left jugular vein was punctured and a catheter placed close to the right atrium to measure CVP.

The thermodilution method was used for the measurement of CO. The surface area of the animals was calculated and CO and stroke volume (SV) values were given in liters $\times$ min^{-1} $\times$ m^{-2} (cardiac index) and ml $\times$ m^{-2} (stroke index), respectively. ABP and CVP were sensed with Statham strain gauges (P 23 AA, P 23 BB) and recorded on a Beckman Dynograph (Type RM) [17].

HUMAN EXPERIMENTS

Two series of experiments were done.

A. Eight experienced long-distance runners and ten well-trained cyclists participated in one study. The long-distance runners covered a distance of 5 km within 25 minutes (temperature 23–28°C, ΔBW −1.3 percent). The cyclists performed a race for 2–3 hours, five of them without fluid intake (temperature 24–28°C, ΔBW −4.1 percent), while the other five were given fluid amounting up to 1.5 percent of their BW (ΔBW −2.5 percent). The data of both studies were pooled in Fig. 5, which contains the values taken 90 minutes after exercise when rectal temperatures had returned toward control levels.

Before exercise CVP was measured with the help of the "arm down" method [5] and plasma volume (PV) was measured using I^{131} or I^{125} labeled human serum albumin. After exercise the measurements were taken as early as possible and repeated at 90-minute intervals. PV was determined 90 minutes after exercise [16].

B. Eight volunteers were dehydrated over a period of 24 hours by intermittent sauna (55 to 60°C) bath and fluid restriction, achieving a dehydration level between 3 and 5 percent of BW. One hour after the end of the dehydration period, they were allowed to drink fluid ad libitum.

In the control period and after the dehydration, CVP and PV were measured as reported above, namely 90 minutes after the dehydration experiments, when the rectal temperatures had returned toward control levels. It should be pointed out here that the changes in CVP shown in Fig. 5 were viewed solely with respect to the degree of dehydration, regardless of which model was applied to achieve the water deficit. This approach seemed justified because after 90 minutes following the dehydration procedures there had been adequate time for fluid equilibration between the body compartments, and the specific physiological differences between the dehydration models could be neglected.

RESULTS

Figure 1 contains the results of a typical single experiment in one dog showing parameters from top to bottom: ABP, heart rate (HR), CO, SV, total peripheral resistance (TPR), CVP, and the temperature in the aortic arch (T_{art}). The control values on the left side of Fig. 1 continue to the right side as dashed lines. The experiment started ten minutes after the end of exercise and extended over the next 180 minutes. Most important for our consideration are the elevated HR and CO, and the lowered CVP and TPR in the face of an elevated body temperature. This situation prevailed for about 60 minutes until body temperature returned to the control level. Mean ABP did not show dramatic changes, however. In this case, it is remarkable that the SV remained considerably above normal for a long time despite a low filling pressure. This pattern was consistent in all the animals.

Combining these parameters for all the dogs (Fig. 2) shows that after the end of exercise HR and CO exceeded control values by 40 percent, whereas TPR is 30 percent below the control. At the beginning, the high CO after exercise is entirely due to the high HR; later it is due to an elevated SV.

As can be seen from Fig. 3, a high CO is maintained as long as body temperature is elevated. There is a linear relationship between these variables. CVP remains low as long as body temperature is high. This inverse relationship is strictly linear for each animal.

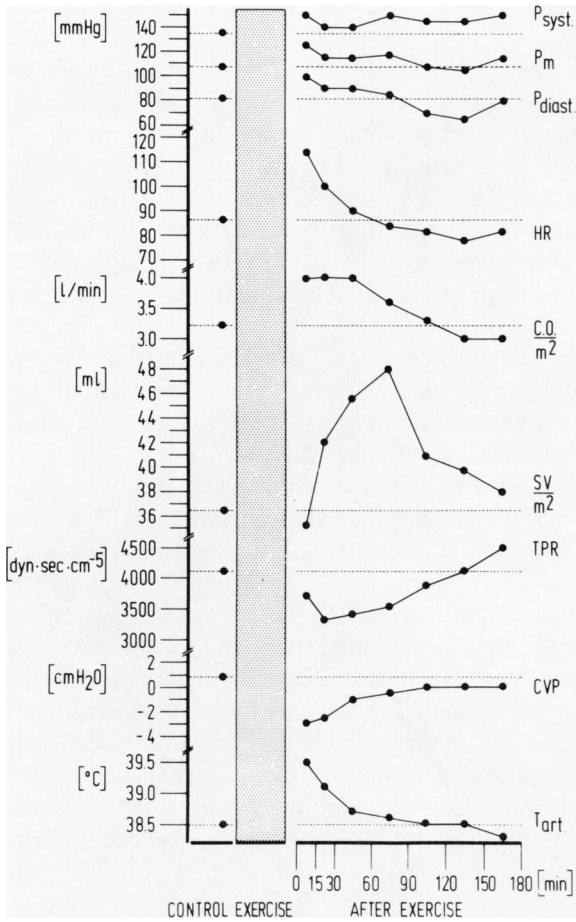

FIG. 1. Typical cardiovascular pattern in one dog in the after-exercise period. Note the elevated stroke volume as compared to the control despite the lowered central venous pressure.

Plotting the CVP against TPR also shows a linear relationship. A low TPR is accompanied by a low CVP and vice versa (Fig. 4). This means that under heat stress a low pre-load is accompanied by a low after-load whereby a high CO is warranted despite low filling pressures. Other authors have shown similar results applying different experimental models over wider temperature ranges [12,26,29,30,31].

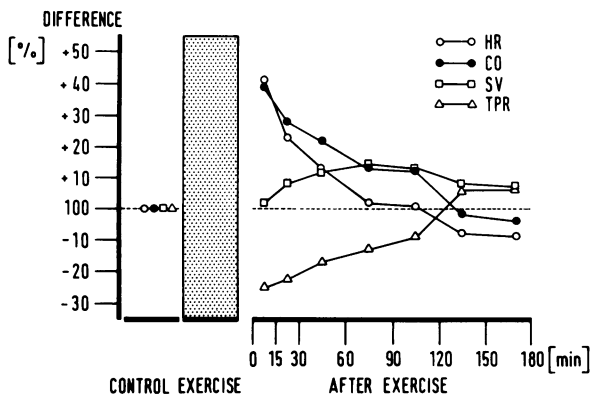

FIG. 2. Changes in cardiovascular pattern during the 180 minutes following exercise. The ordinate depicts the percentage deviations from the controls (100 percent left side). The parameters are derived from mean values from ten experiments in five animals. Initially, high cardiac output is maintained by elevated heart rate, and later by increased stroke volume.

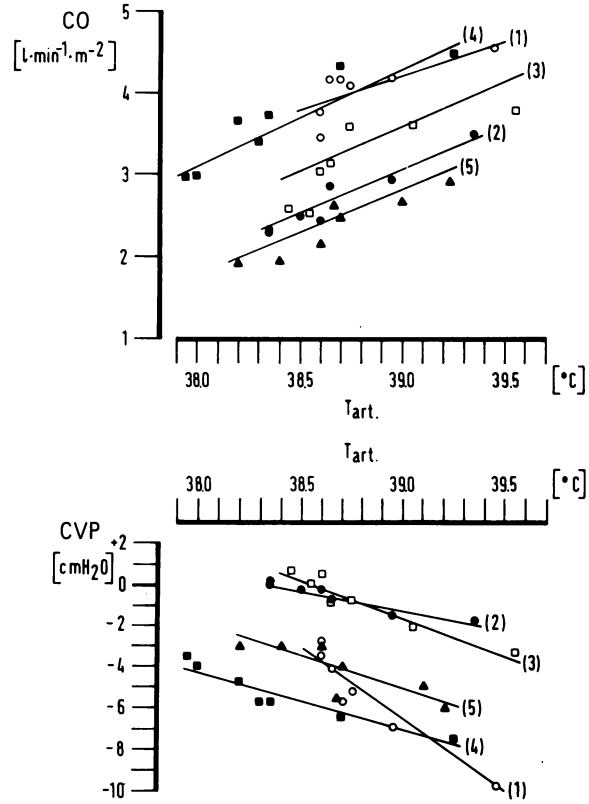

FIG. 3. Cardiac output and central venous pressures in five animals are plotted in relation to the body temperature after exercise. The inter-individual differences between the animals were rather high, but for each animal a linear relationship existed between the variables. The slopes were almost identical. In all cases, a high cardiac output was compatible with a lowered central venous pressure.

In the human experiments, the attempt was always to relate the changes of the fluid volume measured as changes of BW with the concomitant changes of CVP. As can be seen in Fig. 5, changes in BW induced by exercise dehydration in athletes were always accompanied by changes of the CVP. The body apparently monitors the fluid loss whether or not the PV has decreased. It was found, for instance, that the PV remained almost unchanged as long as the dehydration level did not exceed 2.5 percent of BW. In the situation shown here, the extravascular volume was apparently depleted to keep the PV as high as possible.

PV decreased immediately under thermal stress, as can be seen in the next illustration (Fig. 6). Under those conditions CVP is closely correlated with changes of

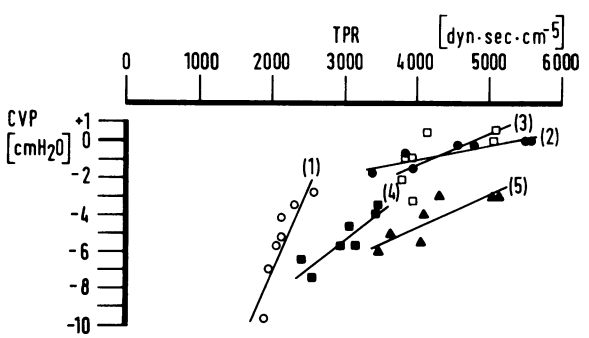

FIG. 4. The central venous pressures in five animals are plotted against the total peripheral resistances. Apparently a vasodilation on the high pressure side accompanies a low filling volume of the intrathoracic compartment. This mechanism is responsible for the fluid translocation toward the periphery. Nevertheless, a high cardiac output was maintained.

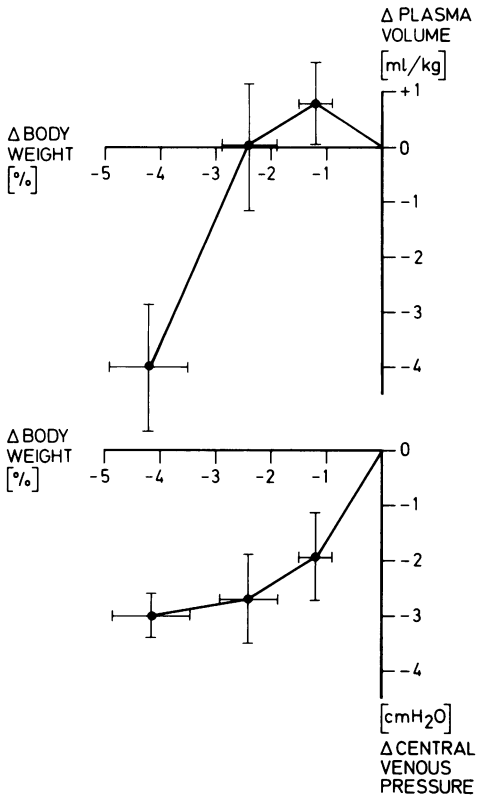

FIG. 5. The relationship between the mean (± SE) decrease in body weight and the simultaneous mean (± SE) changes in plasma volume *(upper ordinate)* and central venous pressure *(lower ordinate)* 90 minutes after exercise and dehydration in trained athletes. The data points in the upper part of the figure, where despite a dehydration no PV changes were seen, stem from the runners and the cyclists who had access to water during exercise (see Methods). In the cyclists dehydrated to 4.1 percent BW, PV decreased by 4 ml/kg. Regardless of the intravascular filling volume, the central venous pressure follows the fluid deficit in this dehydration model.

PV. Several authors have seen, in their experiments applying similar dehydration models, a reduced cardiac function [2,12,30,31]. Under those conditions the reduced cardiac function cannot simply be related to the lowered cardiac filling pressure. Here the low filling state of the extracellular fluid compartment, especially the reduced PV, seems to be a causative mechanism.

CONCLUSION

In the after-exercise period in dogs a lowered filling pressure was very compatible with a high CO and an elevated SV. It was the impression throughout these experiments that, as long as the need for heat dissipation prevailed, the concomitant low CVP was not a critical factor for cardiac function (Figs. 1, 2, and 3). The low pre-load went hand in hand with a low after-load (Fig. 4). Therefore, looking downstream under these conditions, the CVP is not a limiting factor.

At this point we need to be reminded that under these conditions the heart is working in the low range of the Frank-Starling curve, where small increments of the filling pressure can induce large increments of CO. It seems doubtful, however, that these mechanical considerations are of significance as long as the sympathetic outflow is changed in the manner seen under thermal stress [13,14]. The increased sympathetic drive to the heart seems to be more important than the fluctuations of the CVP because intrinsic factors determine the function of the heart rather than mechanical ones such as the filling pressure.

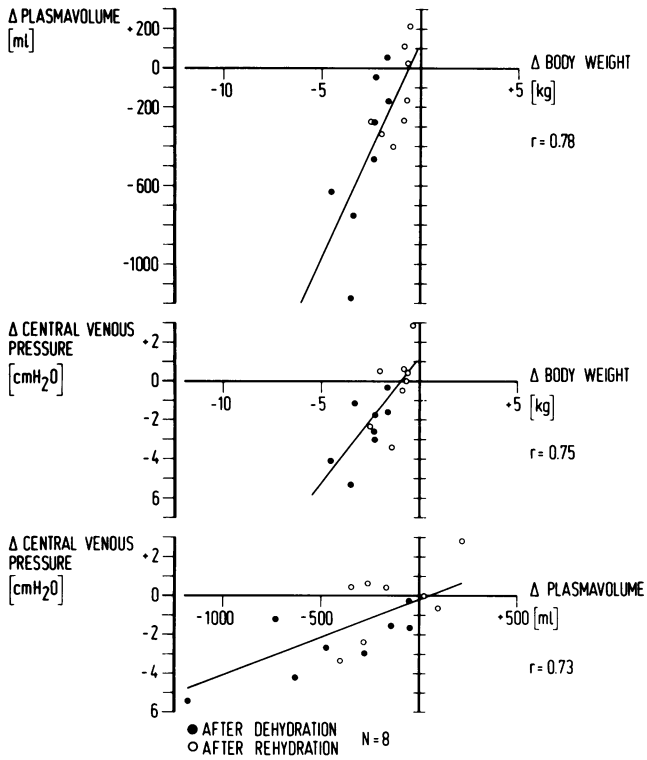

FIG. 6. The interrelationship between the absolute changes in body fluid volumes and central venous pressure (CVP), both immediately after thermal dehydration *(closed circles)* and after rehydration *(open circles)*. Depicted are the individual results of eight subjects, the linear regression lines and the correlation coefficients (**r**) of the following interrelations *(from top to bottom)*: Plasma volume (ΔPV) versus body weight (ΔBW); central venous pressure (ΔCVP) versus ΔBW; DCVP versus ΔPV.

This point of view is more or less in accordance with other authors [26,28,29]. The overall cardiovascular pattern seen under heat stress described above is, among others, brought about by an increased sympathetic outflow from the hypothalamus and spinal cord into the sympathetic nerve fibers supplying the heart, intestines, and kidneys. At the same time, the activity of the cutaneous branch is decreased [13].

This pattern is responsible for the diversion of the blood flow toward the superficial tissues of the body. Furthermore, we must take into consideration the heat-induced increased distensibility of the peripheral veins, which allows the pooling of greater amounts of blood in the skin [24,25,28,29].

We must therefore accept that the body has to live with a lowered CVP under heat stress, which leads to the question of what the lowered CVP means for the circulation.

The pressure gradient from the capillaries to the right ventricle is a critical determinant for the filling of the heart. The lowered CVP widens the gradient, especially from the splanchnic bed toward the right ventricle, and the emptying of the extrathoracic blood stores is improved. As long as enough volume is available in the intravascular bed of the extrathoracic compartment, which can be mobilized by the activation of the muscle pump and venoconstriction, the lowering of the CVP creates no critical situation for the mechanics of the circulation. Under the increased sympathetic drive the heart can generate a higher CO. In the Guytonian view, the heart is shifted toward a curve where even with lowered filling pressures higher outputs can be obtained [9,21,22].

Within this frame of cardiovascular activity, the lowered filling pressure of the heart under heat stress cannot be regarded as a limiting factor, but rather as a necessary

adaptive factor for the recruitment of volume from the extrathoracic parts of the circulation.

The question at this point is whether this view can be transferred to situations where dehydration is superimposed on heat stress. Does the fluid loss change the cardiovascular pattern from the one described above? To answer this question, we have to look upstream.

In general we have to take into consideration a depletion of intra- and extravascular fluid compartments [1,18,20,33]. It seems as if thermal stress particularly affects the intravascular compartment [18] whereas under exercise dehydration the intravascular compartment remains preserved until the dehydration exceeds a certain degree [18,20]. Dehydration invariably leads to a lowering of the CVP as seen by ourselves (Figs. 5, 6) and other authors [2,28,29]. This result was independent of whether exercise or heat stress dehydration was applied. The lowered CVP was seen even when plasma volume was elevated or at control level (Fig. 5).

It seems very likely that, under these circumstances, the sympathetic outflow is modified in such a manner that a relocation of volume into the central parts of the circulation is improved. Generally we saw in these subjects indications of an increased venous vascular tone [16]. Furthermore, the heart rate was elevated from five to ten beats per minute even during rest. Nadel and his group reported similar observations [3,19].

Under heat stress combined with dehydration, CVP apparently monitors the lowered filling volume of the extracellular space, initiating refill mechanisms [7]. Therefore, the idea that CVP is a limiting factor cannot be excluded, especially when the dehydration exceeds 2–3 percent of body weight. However, it is not the pressure per se which is limiting, but the lack of volume. Therefore, we still maintain that the CVP is not a limiting factor.

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