An Inquiry into the Role of Cardiac Filling Pressure in Acclimatization to Heat

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During the first exposure of exercising subjects to hot environments (30–50°C), cardiac output, heart rate, and body temperature increase over that seen in cool environments, while stroke volume decreases. If daily heat exposures occur, during the second heat exposure, heart rates and rectal temperatures are decreased from day 1 while cardiac output is maintained. This decrease in physiological strain occurs with little or no increase in evaporative heat loss. The alleviating agent appears to be an expansion of plasma volume. Several brief studies have indicated decreases in cardiac filling pressure during exercise in heat, and though inferential, it appears that the progressive increase in plasma volume during the first five to six days of heat exposure assists in maintaining cardiac filling pressure. Later, with increased evaporative heat loss due to increased sweat secretion, the mechanism of supplying increased volume to maintain cardiac filling is changed; fluid is transferred from extravascular to intravascular compartment, thus protecting venous return and cardiac filling pressure. These statements are based on limited data, and there is need of experiments designed to confirm or deny certain conclusions as to the role of cardiac filling pressure in acclimatization to heat.

Forty-five years ago Taylor, Henschel, and Keys [1] concluded that improved heat tolerance is gained by increased cardiovascular efficiency early in heat exposure. How this comes about is still a subject of debate. Rowell et al. [2] tested subjects before and after an 11–12-day period of heat acclimatization and concluded that the increased efficiency was mainly due to a decrease in body temperature, allowing heart rate to decrease and stroke volume to increase. A study by Wyndham’s group in 1968 [3] showed that stroke volume decreased on day 1 of heat exposure but returned to control values by day 3. Similar results were also published from the same laboratory some eight years later [4]. These latter results could not be assigned to a decrease in body temperature brought about by increased evaporative heat loss. Certain of these results should be examined more closely since the importance of guarding cardiac filling pressure (CFP) early in heat exposure will be seen not to depend on increased sweat rates and must be due to some other mechanism.

Figure 1 is taken from Rowell et al. [2]. Note that the heart rate on day 2 was 10 percent less than on day 1, and rectal temperature averages 0.7°C less on day 2 than on day 1. These decreases occurred with minimal increases in sweat rates. It stretches the imagination to believe that the decreases in heart rate and body temperatures are owed to increased evaporative efficiency.

Wyndham et al. [3] also published a similar figure wherein day 2 heart rate was 8 percent less than on day 1, and rectal temperature showed a decrease of 0.5°F, all of this with little or no change in sweat rate. Stroke volume also increased 14 percent
(hour 1) to 22 percent (hour 3) on day 2. Wyndham et al. published similar findings in 1976, except that in this study stroke volume did not begin to increase until day 4 of heat exposure [4]. Figure 2 presents a summary of their data concerned with heart rates and rectal and skin temperatures. The mean data for the four subjects in this study show little or no change in rectal temperature on day 2 of heat exposure, but the heart rates decreased and this decrease was 50 percent of that attained by day 10. These mean figures, however, hide two subjects who showed rather different temperature and heart rate responses to heat exposure. In Fig. 2, subject C’s heart rate changed but little over the acclimatization period, but his rectal and skin temperatures did decrease. For subject D, rectal and skin temperatures as well as heart rate remained elevated until day 4 of heat exposure—at which time heart rate dropped precipitously.

With these facts in mind, the next measurements to consider in these same subjects are changes in stroke volume (Fig. 3). First, when compared with the control experiments (25°C db, 18°C wb), stroke volume was decreased for the entire observation period. Second, during acclimatization, mean stroke volume began to increase by day 4,

![FIG. 1. Averages and standard deviations of heart rate (A), sweat loss (B), oxygen uptake (C), rectal temperatures (top of shaded area), and mean skin temperature (base of shaded area) (D) during ten days of acclimatization. Heart rate, Tr, and Ts are means of final exercise values. Modified from [2], with permission.](image)

![FIG. 2. Mean and individual fourth hour heart rates, skin temperatures, and rectal temperatures during ten-day acclimatization regimen. From [4], with permission.](image)
plateaued on days 5, 6, and 7, and showed a tendency to decrease on days 8, 9, and 10. Subject A also followed this scenario, while B managed to bring his stroke volume back to control levels. As we shall see later, both A and B had large expansions of their plasma volumes. The stroke volume of subject C appeared to oscillate and had little definitive change during heat exposure. Subject D is our subject of greatest interest. As had been noted for heart rate and for skin and rectal temperatures, the stroke volume of subject D initially decreased and, between days 4 and 5, his stroke volume practically returned to control values.

The results of heart rate and stroke volume change are seen in Fig. 4, where cardiac output is plotted for each heat exposure day. In all subjects, cardiac output equalled or exceeded control values for most of the heat exposure. Thus, these results do not agree with those of Rowell et al. [2] who recorded a decrease in cardiac output with exercise in the heat.

Now we come to the question: what is the relationship of these adjustments in rectal temperature, skin temperature, heart rate, stroke volume, and cardiac output to sweat rate? As noted by Mitchell et al. [5], the decreases in heart rate, rectal and skin temperature, the changes in stroke volume, and cardiac output prior to day 4 or 5 appear to be totally separate from any change in evaporative heat loss. It would appear that expansion of plasma volume parallels the changes noted above [6]. Figure 5 summarizes these data and Table 1 contains the regression equations relating changes in plasma volume with those in stroke volume, heart rate, and cardiac output [6]. Clearly, the changes in plasma volume appeared to influence the cardiac parameters. In addition to these regression equations, I also calculated the regressions for the group over the first four and final six days of the experiment. The correlation coefficients were considerably higher for the group in the first four than during the final six days of exposure.

When considered in toto, it would appear that the early cardiovascular adjustments
to heat exposure are related to an increase in plasma volume (Fig. 5). By inference, this increase in plasma volume should adjust venous return and return cardiac filling pressure to values which allow for an upward adjustment in stroke volume. The increase in stroke volume appears to level off at the same time that evaporative heat loss increases. With the increase in evaporative cooling, there was a decrease in both

FIG. 4. Daily changes (percentage) in cardiac output from control values. Legend as in Fig. 3. From [4], with permission.

FIG. 5. Individual and group mean changes in plasma volume prior to each exposure to heat and work. Daily plasma volumes were compared to control values and differences were expressed as percentages of control plasma volumes. From [6], with permission.
stroke volume and cardiac output, although with little change in heart rate. Clearly, all these results suggest that the diminution in cardiovascular strain occurs during the first few days of heat exposure and owes little to changes in evaporative heat loss.

Does other evidence support this view? And are there measurements of central venous or cardiac filling pressures which we can use to support this conclusion? We have no measurements of cardiac filling pressures before and after acclimatization; what is offered here must be regarded only as inferential evidence in support of the hypothesis that filling pressure is fundamental to the process of heat acclimatization.

Rowell and co-workers have presented several studies designed to ferret out the influence of elevated skin (and body) temperatures on a number of cardiovascular events [7,8,9]. These reports have been integrated by Rowell into two reviews of human physiologic responses to heat and/or exercise [10,11]. For our purposes, a most fundamental study was presented by Rowell et al. in 1966 [8]. During progressive exercise, Rowell et al. noted that when the subjects performed graded exercise in the heat, stroke volume decreased about 20 ml from that seen in a cool environment. Heart rates were increased in the heat, but the resulting cardiac output was slightly reduced in the heat, particularly at the higher levels of exercise (VO₂ 2.2–2.8 l/minute). These studies are widely quoted but have never been confirmed. The reduction in stroke volume accompanied a reduction in central blood volume. The implication of these studies is clear: due to a decrease in total peripheral resistance, peripheral blood pooling reduced venous return and probably reduced cardiac filling pressure, thus decreasing stroke volume [8]. As noted by Convertino et al. [12], both the exercise and the heat stress stimulate expansion of plasma volume. With 20 years’ hindsight, I wonder what would have been the results if Rowell et al. [8] had recorded responses to exercise in heat on two consecutive days?

Two other experiments by Rowell and co-workers add more detail to the 1966 report [7,9]. In both studies, subjects had their skin temperatures elevated to 38–41°C. One group of subjects was supine and at rest [7], while in the second study, subjects were upright and engaged in cycle exercise [9]. The resting group (Fig. 6) increased cardiac output some six liters through an increase in heart rate and stroke volume, while the exercising group only increased cardiac output by about three liters when heated (Fig. 7). Here, heart rate increased 40 bpm, but stroke volume decreased 10 ml. Since the final levels of cardiac output in liters/minute were similar in both studies, as were total peripheral resistance and mean aortic blood pressures, the reduction in stroke volume can be explained as an inadequate amount of venous return per unit of time. Clearly, the addition of the heat stress to mild exercise led to a condition where cardiac output was increased though stroke volume was decreased. Is this result only a physiological consequence of an increase in heart rate which was mainly due to mental stress? Considering that cardiac output, mean arterial blood pressure, and total peripheral

| TABLE 1 |
| Regression Coefficients for the Equation \( Y = mX + b \), Relating Day-to-Day Changes in Various Cardiovascular Parameters (\( Y \)) with Day-to-Day-Changes in Resting Plasma Volume (\( X \)) Modified from [14] |

| \( Y \) | m | b | r | P |
|---|---|---|---|---|
| % Cardiac output | 0.818 | -10.84 | 0.699 | <0.001 |
| % Stroke volume | 1.185 | -10.60 | 0.717 | <0.001 |
| % Heart rate | -0.612 | -2.214 | -0.675 | <0.001 |
resistance were similar in both studies, as were right atrial mean pressure, one is hard pressed to explain rationally the reduction in stroke volume as being due to heat stress.

Two studies reported by Nadel's group emphasize the importance of alterations in blood volume as a basic cause of changes in stroke volume. In the first of these reports, Fortney et al. [13] studied the influence of blood loss and infusion upon stroke volume, heart rate, and cardiac output during a standard exercise test (60 percent VO\(_2\) maximum) in a room maintained at 35\(^\circ\)C and 30 percent relative humidity (Fig. 8). The experiments dealing with blood volume reduction were not as dramatic as those dealing with blood volume expansion because of the rapid readjustment of plasma volume after blood loss. However, the results were clear-cut; the slight decreases in blood volume appeared to be compensated for by a slight reduction in skin blood flow, thus maintaining cardiac filling pressure, stroke volume, heart rate, and cardiac output. The results following infusion were more dramatic—heart rate was significantly decreased, while stroke volume and cardiac output were significantly increased. These experiments imply that increased blood volume, which leads to increases in stroke volume, probably acts via an increased cardiac filling pressure during exercise. We do know that central venous pressure is increased in upright resting subjects following infusion of 1 to 1.2 liters of blood. Indeed, Robinson et al. [14] recorded a
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1.47-liter increase in resting cardiac output after infusion. Unfortunately, these investigators were interested only in maximal exercise and did not record any central venous pressure except that at maximal exercise, which reached 8.6 mm Hg, an increase of 6.7 mm Hg over resting values.

In a second study, Fortney et al. [15] reduced blood volume via diuretics and increased blood volume by infusion of human albumin in lactated Ringer's solution. In all three conditions, i.e., control, reduced blood volume, and augmented blood volume, the subjects exercised at 60 percent VO₂ maximum for 30 minutes in a room set at 30°C. Hypovolemia reduced stroke volume an average of 17 ml/beat, while hypervolemia increased stroke volume an average of 13 ml/beat. Cardiac output was significantly reduced in hypovolemic subjects, whereas the cardiac output only tended to be greater in hypervolemic subjects. This latter finding seems to indicate that if the stroke volume and heart rate combine to maintain an adequate cardiac output, increasing cardiac filling pressure may not augment cardiac output though stroke volume is raised. Fortney et al. [15] also found that forearm venous volume—which they took as an indicator of venous tone—varied directly with blood volume (Fig. 9). Regardless of initial blood volume, however, the forearm venous volume decreased during the 30-minute exercise bout. Fortney et al. [15] as well as Rowell [11] pointed

FIG. 7. Average circulatory and temperature data from six subjects during mild exercise (0 percent grade, 3.5 mph). CO, cardiac output; Tb, blood temperature in the right atrium; CBV, central blood volume; Ao MP, aortic mean pressure; RA MP, right atrial mean pressure; TPR, total peripheral resistance. From [12], with permission.
out that such a reduction in forearm venous volume may be, in part, due to an increased amount of angiotensin II in the circulation.

I have presented a potpourri of studies—some designed to investigate the influence of heat and exercise on central circulatory responses and some designed to manipulate the central circulatory events. A working hypothesis will now be presented, and its central assumption will be that adequate cardiac filling pressure is central to successful acclimatization to heat and for long-term work in hot environments.

Exposure of an exercising (or working) individual to heat, whether or not endurance-trained, causes cardiac output to increase mainly by an increase in heart rate, for stroke volume generally decreases. Apparently, the subject's blood volume is inadequate to supply both muscle and heat removal. Fortney et al. [15] pointed out that hypovolemic subjects stored more heat (i.e., suffered a greater rise in $T_r$) than control or hypervolemic subjects over 30 minutes of exercise.

The initial heat exposure, if adequate in duration, appears to result in an expansion of the plasma volume after heat exposure and exercise have ended. There is little doubt about the expansion, but the mechanism behind the expansion is still being debated. If the subject returns again to exercise in the heat—usually the next day—and if his or
her plasma volume has increased, venous return and cardiac filling pressure would probably be increased. This increase in cardiac filling pressure over the preceding exposure day is best seen in a decrease in heart rate and a decrease in body temperature. The continued reduction in body temperature and heart rate during the first three to four days of exposure is most probably due to the continued expansion of the plasma volume with concomitant increases in stroke volume and convective heat transfer. With the increase in sweat rate, and the evaporative heat loss which occurs after this initial plasma volume expansion, skin temperature is lowered. To remove a certain quantity of heat, less cutaneous blood flow would be needed, and thus a subject can maintain a particular core temperature though cutaneous blood flow may be reduced. We now have a subject who has an expanded plasma volume, whose convective heat transfer within the body is certainly adequate, and whose sweat rate is still increasing. Where initially we had adequate venous return and cardiac filling pressure, we now have a situation where peripheral vascular tone is increased, cutaneous blood flow is probably reduced, and venous return and cardiac filling pressure—particularly the latter—are increased. These are or may be the findings if one follows subjects for 10–12 days, but what happens over the long term? Bass et al. [16] noted that men who had acclimatized for three weeks had blood volumes similar to those measured before heat exposure, I have also followed the course of plasma volume beyond the usual 10–12-day exposure, and my findings agree with those of Bass et al. [16]. After three or more weeks of exercise in heat, resting plasma volume returns to that seen before acclimatization began. Then how is cardiac filling pressure guarded now? Cardiac output still must be directed both to active muscle and into heat dissipation channels. Does increased evaporation account for continued reduction in heart rate? For many of these questions we have no direct answers, but partial answers are known. From experiments that I performed with R. Kok [17], we showed that subjects who had been acclimatized to heat for longer than three weeks enlarged their plasma volumes during the heat exposure and lost this volume between exposures. It would appear then that, in long-term acclimatization, cardiac filling pressure is guarded by the same mechanism that operated in the first ten days of heat exposure, i.e., an increase in circulating blood volume. The difference is the manner in which the plasma volume is expanded and contracted between exposures. We know little or nothing of the mechanisms involved.
In summary, if humans are to acclimatize to heat and exercise, their first necessity is an adequate circulating blood volume. If this is assured, then cardiac filling pressure becomes sufficient to augment stroke volume and cardiac output. Once these events are put in motion, certain variations appear which are probably due to increased evaporative heat loss, and to as yet unexplained change in the dynamics of fluid exchange between the extravascular and vascular compartments.

REFERENCES

1. Taylor HL, Henschel AF, Keys A: Cardiovascular adjustments of man in rest and work during exposure to dry heat. Am J Physiol 139:583–591, 1943
2. Rowell LB, Kraning KK II, Kennedy JW, Evans TO: Central circulatory responses to work in dry heat before and after acclimatization. J Appl Physiol 22:509–518, 1967
3. Wyndham CH, Benade AJ, Williams CG, Strydom NB, Goldein A, Heyns AJA: Changes in central circulation and body fluid spaces during acclimatization to heat. J Appl Physiol 25:586–593, 1968
4. Wyndham CH, Rogers GG, Senay LC, Mitchell D: Acclimatization in a hot human environment: cardiovascular adjustments. J Appl Physiol 40:779–785, 1976
5. Mitchell D, Senay LC, Wyndham CH, van Rensburg AJ, Rogers GG, Strydom NB: Acclimatization in a hot, humid environment: energy exchange, body temperature, and sweating. J Appl Physiol 40:768–778, 1976
6. Senay LC, Mitchell D, Wyndham CH: Acclimatization in a hot, humid environment: body fluid adjustments. J Appl Physiol 40:786–796, 1976
7. Rowell LB, Brengelmann GL, Murray JA: Cardiovascular responses to sustained high skin temperature in resting man. J Appl Physiol 27:673–680, 1969
8. Rowell LB, Marx HJ, Bruce RA, Conn RD, Kusumi F: Reductions in cardiac output, central blood volume, and stroke volume with thermal stress in normal men during exercise. J Clin Invest 45:1801–1816, 1966
9. Rowell LB, Murray JA, Brengelmann G, Kraning KK II: Human cardiovascular adjustments to rapid changes in skin temperature in exercising man. Circ Res 24:711–724, 1969
10. Rowell LB: Human cardiovascular adjustments to exercise and thermal stress. Physiol Rev 54:75–159, 1974
11. Rowell LB: Cardiovascular adjustments to thermal stress. In Handbook of Physiology, The Cardiovascular System III. Edited by JT Shepherd, FM Abboud. Bethesda, MD, American Physiological Society, 1983, Ch 27, pp 967–1023
12. Convertino VA, Greenleaf JE, Bernauer EM: Role of thermal and exercise factors in the mechanism of hypervolemia. J Appl Physiol 48:657–664, 1980
13. Fortney SM, Nadel ER, Wenger CB, Bove JR: Effect of acute alterations of blood volume in circulatory performance in humans. J Appl Physiol 50:292–298, 1981
14. Robinson BF, Epstein SE, Kahler RL, Braunwald E: Circulatory effects of acute expansion of blood volume: studies during maximal exercise and rest. Circ Res 19:26–32, 1966
15. Fortney SM, Wenger CB, Bove JR, Nadel ER: Effect of blood volume on forearm venous and cardiac stroke volume during exercise. J Appl Physiol 55:884–890, 1983
16. Bass DE, Buskirk ER, Iampietro PF, Mager M: Comparison of blood volume during physical conditioning, heat acclimatization and sedentary living. J Appl Physiol 12:186–188, 1958
17. Senay LC, Kok R: Body fluid responses of heat-tolerant and intolerant men to work in a hot wet environment. J Appl Physiol 40:55–59, 1976