The following is the abstract of the article discussed in the subsequent letter:

Krediet, C. T. Paul, Ivar G. J. M. de Bruin, Karin S. Ganzeboom, Mark Linzer, Johannes J. van Lieshout, and Wouter Wieling. Leg crossing, muscle tensing, squatting, and the crash position are effective against vasovagal reactions solely through increases in cardiac output. J Appl Physiol 99: 1697–1703, 2005; doi:10.1152/japplphysiol.01250.2004 —Tensing of lower body muscles without or with leg crossing (LBMT, LCMT), whole body tensing (WBT), squatting, and sitting with the head bent between the knees (“crash position,” HBK) are believed to abort vasovagal reactions. The underlying mechanisms are unknown. To study these interventions in patients with a clinical history of vasovagal syncpe and a vasovagal reaction during routine tilt table testing, we measured blood pressure (BP) continuously with Finapres and derived heart rate, stroke volume, cardiac output (CO), and total peripheral resistance using Modelflow. In series A (n = 12) we compared LBMT to LCMT. In series B (n = 9), WBT was compared with LCMT. In series C (n = 14) and D (n = 9), we tested squatting and HBK. All maneuvers caused an increase in BP, varying from a systolic rise from 77 ± 8 to 104 ± 18 mmHg (P < 0.05) in series A during LBMT to a rise from 70 ± 10 to 123 ± 9 mmHg (P < 0.05) in series B during LCMT. In each maneuver, the BP increase started within 3–5 s from start of the maneuver. In all maneuvers, there was an increase in CO varying from 54 ± 12% of baseline to 94 ± 21% in WBT to a rise from 65 ± 17% to 110 ± 22% in LCMT in series A. No maneuver caused significant change in total peripheral resistance. We conclude that the mechanism underlying the effects of these maneuvers is exclusively an increase in CO.

Improved orthostatic tolerance by leg crossing and muscle tensing: indisputable evidence for the arteriovenous pump existence

To the Editor: Dr. Krediet and coworkers (6) analyzed the beneficial effects of leg crossing, combined with muscle tensing against vasovagal reactions. They concluded that the effects of these maneuvers are exclusively due to the increase in cardiac output (CO). Their conclusions are based on the very important finding that “there are remarkable resemblances between the effects of the physical countermaneuvers in the present study on CO and those when inflating an antigravity suit at the onset of an impending vasovagal faint.” This finding resulted from the establishment that “none of the physical countermaneuvers in the present study had any significant effect on total peripheral resistance,” a view that coincides with cited publications (26) reporting experiments with antigravity suits. The authors present convincing evidence that, excluding initial central command, the effect on blood pressure is a mechanical and not a reflex one. It is interesting that the latter should provoke the conclusion that the term “vasovagal” is not suitable to characterize the nature of all types of syncope. Unfortunately, the enormous confusion in the venous return theory evidently hinders the right explanation of orthostatic intolerance and the measures for its counteraction. To explain the increased CO resulted from all antisyncope maneuvers, Dr. Krediet et al. (6, 7), Dr. Cui et al. (4), and Dr. Levine in his response to a Letter to the Editor (9), use Guyton’s classic view that central venous pressure is an active element in the venous return, a concept recently criticized (3).

However, in disputing the blood circulation theory, our basic question is: Which is the energy source for the venous return against gravity and venous occlusions? Any answers to that question with some static displacements of blood volumes from one part of the body to another simply due to changes in the body position or continuous compression are against the logic of basic physical laws. Such displacements themselves cannot provide energy in a steady state. In the past 3 yr, we have made enormous efforts to promote our new circulation theory (8, 9), in which we assert that the primary role for the venous return is assigned to the transmural transmission of energy from arteries to veins, i.e., the arteriovenous pump (AVP). According to this theory, the major AVPs are in the extremities, where the anatomic structure of the arteriovenous (AV) complexes strongly depends on the specific forces acting on the venous blood and on the presence, or development, of the surrounding muscles as supportive tissue. In the terrestrial extremities, which work periodically contacting the ground (the inertial force from the rotation alternating between a centrifugal and centripetal one), the AV complexes are expressed usually as a triad. Where the muscles are not sufficiently developed to support the pulse transmission, as in the “lazy” animals, the complex takes the form of a rete (10). In the absence of muscles, it is also a rete [the ophthalmic rete (1, 2), the effectiveness of which varies depending on the inertial forces acting on the venous blood], the effectiveness of which in speedy birds is greater (1). In birds’ wings (2) or whales’ fins (11), where great centrifugal forces exist and the muscles are either absent or weak, there is a well-developed venae comitantes system completely enveloping the accompanying artery. This configuration turns again into a triad (2), where the inertial force weakens, or changes direction, as in the distal part of the wing, which is bent by intense flight (our observations). All these deviations from the standard triad are interpreted as heat exchangers (1, 2, 10, 11). We believe, however, that these are primarily AVPs, because the venous return has priority over heat exchange and that their configuration follows more closely the mechanical change (2) than the thermal one.

We hope that the above arguments will help to convince the reader that the leg crossing maneuver, combined with muscle tensing, acts in increasing the muscles’ supporting action for the work of the AVP. The implementation of lower body positive pressure has a similar effect, i.e., compressing the muscles to the AV triad. All measures for increasing vascular tone act in the same direction to enhance the transmural transmission of energy from arteries to veins. Indicative in this regard are the fluid infusions, sympathetic activity, gravity, venous occlusion, and skin cooling (4). The effect of all these is the increase of the vascular tone in the deep vessels, i.e., in the AVPs, and not simply the increased central venous blood volume, as explained by Cui et al. (4). An opposite action is produced by all measures that lead to a decreased vascular tone in the deep vessels: reduced blood volume, skin heating (5), vasodilators, or reduced compression on the AV complexes by skeletal muscles, such as the lower body negative pressure or head-up tilt. The lower grade of orthostatic intolerance that is reached on the tilt table, in comparison the active standing, we believe is explained by the loose muscles in the lower extremities.

REFERENCES

Letters to the Editor


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REPLY

To the Editor: Dr. Panchev and coworkers continue to present their arteriovenous pump (AVP) theory (7, 8). This theory holds that the kinetic energy from the arterial pulse wave is in part “transmurally transferred” to the venous system, thus facilitating venous return to the heart. However, so far there is no experimental evidence supporting this theory. Where Dr. Panchev et al. refer to observations in the whale indirectly alleging evidence for their theory, it may be considered that whole body immersion does not apply directly to the land-dwelling human circulation.

As a start for future investigations, one could measure interstitial fluid pressures in the tissues where presence of the proposed AVP may be substantiated by revealing any relevant pressure-transducing mechanism. The evidence available seems as yet inadequate to give convincing reason for the title of Dr. Panchev et al.’s letter when referring to our results as “indisputable evidence” for their theory.

The main finding of our study was that isometric leg muscle contractions at the onset of a vasovagal faint instantly raise cardiac output (3). We did not measure thoracic blood volume but compared the effects of leg muscle contraction with those in response to rapid inflation of an antigavity suit (12). We assumed thoracic blood volume to increase due to an immediate emptying of the large veins through muscular compression in the lower body (5, 12). The assumption that venous pooling capacity in the lower body remains minimal as long as muscle tensing is continued, with a more beneficial volume distribution, is supported by a recent study where we documented in healthy subjects that leg crossing without leg muscle tensing during 20 min of orthostatic stress reduces thoracic impedance (4).

We agree that right atrial pressure in a dynamic system is a result of venous return and not a determinant (2). Given the linear relationship between stroke volume and thoracic fluid content vs. a nonlinear one for right atrial pressure, we prefer impedance as a measure of cardiac preload (9, 10).

The question posed by Panchev et al. “which is the energy source for the venous return against gravity and venous occlusions?” is to our current belief satisfactorily answered by the experimental evidence for mechanical factors, including the muscle (1, 5, 11) and respiratory (6) pump.

REFERENCES


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