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Venous Physiology: The Overlooked Key to Syncope and The Regulation of Cardiac Output

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Abstract

As the years go by, the physiology of the human body becomes ever better understood. But because venous tone cannot easily be measured it is no wonder the physiology of veins has been consistently overlooked. The author describes his experiences suggesting the key role it appears to play in vasovagal syncope, the regulation of cardiac output, high altitude pulmonary edema, beri-beri, and in those with cardiac arrest.

Venous Physiology

As the various physiologic mechanisms of the body were discovered one by one, measurements were made which disclosed their function in health and disease. Veins have a smooth muscle coating responsive to autonomic and circulating pressor control. But this "control" cannot be easily followed. Veins are conduits open at both ends so venous pressure measures mean right atrial pressure not venous tone. To my knowledge only one person has ever actually measured venous tone. In the 1950's an investigator at the NIH inserted a double-balloon catheter in a subject's arm vein. By recording the pressure in this isolated segment he was able to follow venous tone for the first time. It increased when the subject stood and fell when he was supine. It rose when a pint of blood was drawn from the other arm. It is understandable why the physiology of veins has been largely overlooked, the rest of us have never made the proper measurements.

When I was a medical student in the class of '56 at the University of Washington I had just measured a patient's venous pressure at 12 cm. of saline, when suddenly the saline in the manometer fell to zero. I looked down thinking it had become disconnected, but everything was intact. I asked him how he was, and he said, "Fine." The over the next few seconds he became progressively pale and fainted. Here was the actual cause of the loss of consciousness in vasovagal syncope. Four of our five liters of blood are in our veins. With the sudden loss of venous tone, blood flows back into the chest all but stops. With the heart rate slowed to 60 or so, each stroke volume falls progressively until the liter per minute blood flow the brain requires for consciousness is no longer available.

A week or so later I had just measured a patient's spinal fluid pressure at 10 cm. when the same thing happened—it fell to zero. He claimed he felt fine but I knew what was coming. I stopped the procedure, put him on his back and raised his legs. His pulse slowed and his pressure fell but he didn't lose consciousness.

Later when I was a Cardiology Fellow with Robert Bruce I had the opportunity to review the many indicator dilution curves in his files. These were done by injecting Evan's Blue dye in an arm vein and recording from an earlobe oximeter. Curves such as these reveal the cardiac output, the central blood volume (the blood in the chest), the cardiac stroke volume, and the presence of any intracardiac shunts. I found the Korner and Shillingford literature on dye curves to be invaluable in teaching me the fine points of dye curves.

It was quickly apparent that the cardiac stroke volume was always directly correlated with the central blood volume. The heart pumps whatever is handed to it. The stroke volume hinges on the blood in the chest which in turn hinges on the status of venous tone. The autonomic nervous system controls venous tone, and this is what regulates the cardiac output. What are some examples where this can be seen?

This mechanism gives a clearer understanding of acute pulmonary edema. When a diseased heart can no longer deliver the expected cardiac output, venous tone increases to raise it. But a diseased heart can't handle the added load; the left ventricular end-diastolic pressure rises until pulmonary congestion results. Emergency therapy is usually centered on getting blood out of the chest with rotating tourniquets. Knowing the role of venous tone in this condition, it suggests giving something to relax veins such as nitroglycerine or two. This is how NTG relieves angina pectoris, so it should be no mystery.

What is it that calls for more cardiac output and where in the body might we find it? Clues come forth when high cardiac output states are examined to see what they might have in common. Anything that interferes with the Krebs cycle is interpreted by the body as a need for more blood supply—such as thiamine deficiency, sub-lethal Potassium Cyanide poisoning, or carbon monoxide exposure. Whatever results in insufficient conversion of ADP to ATP by the electron transfer system of the Kreb's cycle, is a call for more cardiac output even when this is not the basic problem. If the venous tone regulation is the efferent limb of this reflex, where is the afferent arm?

Every organ in the body can extract more oxygen from the blood passing through it if the flow should fall for any reason, except for the heart itself. The coronary sinus blood is normally almost black. This is a peculiar way to design a heart unless there is a hidden purpose. Should the cardiac output fall for any reason the heart would be the first to know. If this were so, one would expect those with denervated hearts (those with heart transplants) to react differently from normal to a challenge such as breathing an atmosphere low in oxygen.

Mountain climbers who go above 20,000 feet know about the risk of high altitude pulmonary edema. The partial pressure of oxygen is half normal compared to sea level, so I am sure the venous tone mechanism

kicks in on all of them to raise their cardiac outputs. Why do only a few get in trouble? Most climbers like this are in unusually good condition, but if some degree of ventricular hypertrophy is present as part of their "fitness" volume loads will not be handled well. Although there are many who feel the problem is primarily pulmonary, I would advise these climbers to carry a few NTG just in case. It may help to buy a little time while they get to a lower altitude.

Another area where venous tone (or the lack of it) comes into play is in the patient with a cardiac arrest. CPR is known the world over and has been a life-saver, but the results are still disappointing. Unless a defibrillating shock can be delivered in a timely manner, some degree of permanent brain damage is common in survivors.

About forty years ago I happened to be present when a monitored patient in Seattle's Providence Hospital went into ventricular fibrillation. While others looked for the defibrillator I started CPR. In less than a minute he opened his eyes, looked at me and said, "What the hell do you think you're doing?" Thinking he must have converted on his own, I stopped. In seconds he was unconscious again. Only in retrospect have I realized how rare this was. Doing CPR does not wake people up. I must have been generating more cardiac output than usual. What was I doing that was different? Because he was on a high flat examining table I had climbed up and was sitting on him. This might do several beneficial things. It is the jump in the intra-thoracic pressure from one's chest thump that squeezes the heart. Two things interfere—air is pushed out of the lungs, which is good, and the diaphragm is pushed down, which is not good. Sitting on him tended to stabilize the diaphragm which allowed a higher intra-thoracic pressure.

Sitting on him also compressed his flaccid aorta against his spine so cardiac output was not wasted down his legs. And it pushed blood in the belly into the chest. The arrested patient is very likely like the patient who faints—no venous tone. Anything to increase the blood in the chest would be helpful. Epinephrine is commonly given to these patients. I suspect the primary beneficial effect here is the restoration of venous tone.

After a career as a Seattle Cardiologist I retired many years ago to this farm in North Idaho. It is my hope that inventive minds in academia will consider these ideas, test them out and confirm them if possible. In the meantime I hope ER physicians will think of NTG in pulmonary edema that climbers to high places will carry some NTG, and those called on to do CPR will sit on them and maybe even have someone else lift their legs. Obviously waking people up are not the goal here. It's preserving brain function. If patients everywhere start waking up during CPR however, it could be the "shot heard around the world."

Biography of Author

Kenneth Eyer graduated AOA in the class of '56 at the University Of Washington School Of Medicine. After an internship and a residency in Internal Medicine he was a Cardiac Fellow under Robert Bruce. He served as a Cardiologist at Seattle's Providence Heart Center, and was a Full Professor of Clinical Medicine at the Uof W until he retired in 1985. He is perhaps best known for his discovery of an EKG sign of heart failure and for his article in the Jan-Feb 2015 issue of the Journal of Electrocardiology.