Is the Arterial Pulse a Soliton?

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ABSTRACT: The recent discovery of arterial smooth muscle contractions which are synchronized with the activity of the right atrial pacemaker in the rabbit heart gives support to the hypothesis that the arterial pulse wave is a soliton. The hypothesis is amenable to experimental tests.

SOLITONS ARE MATHEMATICAL OBJECTS which appear as solutions of particular nonlinear wave equations (Bullough, 1978; Bullough and Caudrey, 1980). The soliton is a wave which has a stable and permanent waveform (though it may have internal oscillations) and which has peculiar collision properties with other solitary waves (Ablowitz and Segur, 1981). For the experimental scientist it is a nondispersing wave packet with particle-like properties and in the last decade it has found applications in nonlinear optics, condensed-matter physics, and quantum field theories of matter and gravity.

Biological systems are nonlinear and so it is reasonable to look for solitons among the phenomena of life. Davydov (1977, 1979) has suggested that solitons are the energy transmitters in cells and del Giudice, Doglia and Milani (1982) have combined Davydov's ideas with Fröhlich's (Fröhlich, 1980) theory of energy-driven coherent electric vibrations into a dynamics of living matter. This combined theory gives a reasonable explanation of the findings of Webb (1980) who has studied the Raman spectra of metabolically active *E. coli.*

The arterial pulse is a single wave which has some of the properties of the solitons observed in the condensed state of matter (Lamb, 1980). It is constrained as a pseudo-one-dimensional wave by anatomical geometry. Its velocity is much less than the velocity of sound in either the arterial wall or the blood, and the greater its amplitude, the greater its velocity. Moreover it maintains its form as a solitary wave right out to the periphery while its "amplitude" increases. That is, the pulse pressure increases though, of course, the mean arterial pressure gets less. The textbook explanation for the increase in pulse pressure is based on speculations involving reflections of the wave within the arterial tree (Guyton, 1976). Until recently the difficulty with the soliton hypothesis for the arterial pulse was that the energy delivered to the viscoelastic arterial wall must, in part, be dissipated as heat. To be a soliton a wave must be lossless. This is possible in the transmission of the quantum of energy from the hydrolysis of ATP along the α-helix of a protein (Davydov, 1979) but it is clearly absurd in the case of the arterial wall. The function of the smooth muscle in the arterial wall has, however, never been elucidated and it might indeed provide energy to compensate for the viscous loss during stretch and recovery. But the response of smooth muscle to stretch is slow, much slower than what is needed to compensate synchronously for energy loss in the pulse wave.

What has made the soliton concept much more acceptable is the recent finding of a neural connection between the right atrial pacemaker and arterial smooth muscle in the rabbit (Mangel, Fahim and van Breemen, 1982). The arterial contractions remain in synchrony with the pulsatile activity of the heart even when the heart is electrically stimulated at rates of 600 beats per minute. So as each pulse travels down an artery the smooth muscle contracts in well-controlled synchrony.

The hypothesis of a soliton pulse is now amenable to experimental tests. Mangel, et al. (1982) removed the right atrium in their rabbits. The pulse-synchronized arterial contractions ceased, but the authors do not comment one way or the other on any changes in the pulse wave. The smooth muscle could be reversibly paralyzed pharmacologically and the neural pathways, once they have been traced, could be interrupted. These maneuvers could be combined with changing the heart's contractility to vary the amplitude of the pulse. On the soliton hypothesis one would expect alterations to occur in the pulse wave and, in particular, for its peaking, peripherally, to be profoundly affected.

"Real" solitons are, as noted, mathematical abstractions which arise as solutions of certain nonlinear equations. F. S. Khumalo (private communication) has modelled the arterial wall as a system of loosely coupled, nonlinear contractile rings. His wave equation is very similar to the well-known Korteweg-deVries equation (Bullough, 1978, 1980; Lamb, 1980), which has soliton solutions. So there are certainly good grounds for instituting further tests on the present hypothesis.

Life seems to employ any useful mechanism which is available. But thinking teleologically raises the question why the mammalian circulation involves itself in such complications to maintain a large peripheral pulse pressure? It is frequently forgotten (by medical students anyway) that a peripheral artery runs in the same connective tissue sheath as its corresponding peripheral vein. It is the pulse pressure, transmitted by contact to the vein, which together with the valves in the veins drives the blood unidirectionally back to the heart and so provides a substantial part of the "venous return." A nonpulsatile arterial pressure could not effect this essential function.

The author thanks Dr. Frank Khumalo for critical discussions of these ideas.

REFERENCES

Ablowitz, M.; Segur, H. 1981. *Solitons and the Inverse Scattering Transform*, Siam, Philadelphia.

Bullough, R. K. 1978. *Phys. Bull. 29*, 78-82.

Bullough, R. K.; Caudrey, P. J. 1980. *Solitons*, Springer-Verlag, Berlin.

Davydov, A. S. 1977. *Studia Biophys. 62*, 1-8.

Davydov, A. S. 1979. *Phys. Scripta 20*, 387-394.

del Giudice, E.; Doglia, S.; Milani, M. 1982. *Phys. Scripta, 26*, 232-238.

Fröhlich, H. 1980. *Adv. Electron. Electron Phys. 53*, 85-152.

Guyton, A. C. 1976. *Textbook of Medical Physiology* (5th ed.) Saunders, Philadelphia, p. 241.

Lamb, G. L., Jr. 1980. *Elements of Soliton Theory*, Wiley, New York.

Mangel, A.; Fahim, M.; van Breemen, C. 1982. *Science 215*, 1627-1629.

Webb, S. J. 1980. *Phys. Rep. 60*, 201-224.