Respiratory Variations in Blood Pressure

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A study of respiratory blood pressure variations in normal subjects suggests that variations in both cardiac output and peripheral resistance are involved, the latter mechanism being essentially similar to that producing the Traube waves in apnea. The interactions of the two mechanisms are compared to the known behavior of coupled electronic oscillators.

The fluctuation of the arterial pressure with respiration was first noted by Stephen Hales in 1733. The subject has since attracted the attention of many workers and has a large literature. In spite of this there are, as we hope to show, aspects of this apparently simple and easily observed phenomenon which remain mysterious.

In this article we shall not attempt to review the literature. We shall summarize our experience as a series of propositions supported by illustrative records, merely indicating the relation of our views to received opinion. We shall then discuss the underlying mechanisms and describe some relevant experiments.

Material and Methods

Some 50 records of blood pressure and respiration, many with additional information, from normal volunteers and from patients with normal circulations, form the basic material of this report. All pressures were measured with capacitance manometers. Chest movements were recorded by a Manning spirograph operating either a capacitance or photoelectric pickup.

Results

1. Abdominal and thoracic breathing have similar effects on the blood pressure (fig. 1). The contrary view has often been expressed. The disparity probably arises from a failure to standardize respiratory rate. Unless care is taken most subjects will breathe more deeply and more slowly with abdominal than with thoracic respiration.

2. The amplitude of the blood pressure swings increases with decreasing respiratory rate (fig. 2). This is generally agreed. We have found it to hold down to rates of about 6 per minute.

3. The relation between the blood pressure variation and the respiratory phase is a function of respiratory rate. At moderate rates the pressure is falling during most of inspiration. At slower rates inspiration is associated throughout with rising pressure. This rate dependence, which we have found very regularly, does not appear to have been widely recognized and may account for some conflicting statements made in the past. (fig. 3).

4. Blood pressure swings are enhanced by the upright posture (fig. 4). The enhancement is associated with an increase in the variation of pulse pressure. The left hand side of the figure shows that considerable pressure swings can occur with little change in pulse pressure.

5. Sinus arrhythmia does not contribute to the blood pressure swings. This statement is based on the frequent occurrence of an event illustrated in figure 4. Gross arrhythmia suddenly appears where there has been little change of heart rate in previous respiratory cycles: the pressure waves continue their course, the slowing being compensated for by increased stroke volume. When present, slowing always appears in the descending limb of the pressure wave, irrespective of the respiratory phase.

6. During apnea regular pressure waves may occur (fig. 5). These, which are conveniently called Traube waves, are not associated with material pulse pressure changes unless, as sometimes happens, there is accompanying rate change, when the slowing on the descending limb is balanced by an increased pulse pressure. The waves are often fugitive, but when they occur their rate is consistently about 6 per minute.

7. The blood pressure may exhibit fluctuations at a submultiple of the respiratory rate (fig. 6). This phenomenon is quite common, but is not...
easy to investigate as it often disappears when a subject changes from unconsidered to deliberate breathing. Sinus arrhythmia may follow the slow wave rather than, or as well as, the respiratory waves.

**Fig. 4.** Two sections of a continuous tracing; the left hand one with the subject supine, the right hand one, erect. In the erect posture the fluctuations in pulse pressure are exaggerated. Sinus arrhythmia occurs from time to time and has little effect on the blood pressure waves.

**Fig. 5.** Traube waves during apnea.

**Fig. 6.** In the above record there is a fluctuation at one third of the respiratory rate. The subject varies his respiratory rate from 15 per minute to 20 per minute and back. The slower waves maintain their relationship with the superimposed respiratory waves.

The slower waves are thus occurring at near the Traube (apnea) rate of 6 per minute but their frequency is entrained by variation in respiratory rate.

**Interpretation of Pressure Records**

If the heart rate does not change, variations in peripheral resistance should produce corresponding variations in mean blood pressure with little change in pulse pressure, whereas
variations in stroke volume should produce variation in pulse pressure and mean pressure in about equal proportion. Conversely one may provisionally interpret pressure changes of the first type (equal systolic and diastolic swings) as indicating changes in peripheral resistance and changes of the second type (proportional systolic and diastolic swings) as indicating changes in stroke volume. Intermediate patterns may be attributed to the action of both factors, whose relative contributions may be to some degree assessed by noting the relation of pulse pressure to mean pressure modulation. For such an analysis, cycles with minimal rate variation must be selected. As will be seen later, the circumstances in which the pure patterns occur support these interpretations.

**Mechanism of Stroke Volume Modulation**

It is generally agreed that respiratory modulation of stroke volume is produced as follows. Inspiration lowers intrathoracic pressure and enhances filling of the right heart from the extrathoracic veins. Right ventricular stroke volume thus increases, and hence the effective (distending) pressure of the lesser circulation rises. The rise in effective pressure in the pulmonary veins leads to increased filling of the left heart and so to increased left ventricular stroke volume.

The dynamic implications of this mechanism have not, so far as we are aware, been recognized. It can be seen that the resistance and the hydraulic capacitances of the lesser circulation interpose a "lag" mechanism between inspiration and right ventricular output increase on the one hand and the rise of effective pulmonary venous pressure and left ventricular filling on the other. It follows that, for a given depth of respiration, stroke volume modulation will decrease with increasing respiratory rate. Moreover, while there is a primary phase relationship between inspiration and increased stroke volume, with increasing rates, the phase of the stroke volume change will lag increasingly behind the corresponding respiratory phase. It thus comes about that at moderately rapid rates stroke volume (and blood pressure) is falling throughout most of inspiration, the fall being due not to the accompanying inspiration but to the preceding expiration. The true causal connections may be displayed by interrupting breathing for a few seconds at different points in the cycle (fig. 7).

Stoke volume modulation is seen in its purest form when autonomic activity has been blocked by, for example, tetraethylammonium (fig. 8). The relationship between stroke volume change and chest expansion is inverted by passive lung inflation, and the dependence of the stroke volume on thoracic pressure rather than thoracic posture is thus confirmed (fig. 9). The enhancement of stroke volume modulation by the assumption of the upright posture, which is known to lower right auricular pressure, suggests that the relationship between right ventricular output and effective filling pressure is not a linear one but is steeper when the filling pressure is low.
Changes of intrathoracic pressure, acting as they do equally on pulmonary veins, left auricle and left ventricle, cannot immediately modify the existent intravascular pressure gradients between these regions, and no immediate effect on left-ventricular filling is to be expected. Nevertheless, the first one or two beats accompanying inspiration after an expiratory pause often show a small but definite reduction of pulse pressure before the expected increase occurs. This is because ventricular events are not wholly independent: since there is some sharing of the ventricular muscle the increased filling of the right ventricle at the start of inspiration tends to be at the expense of the filling of the left. This effect is always trivial in the normal but becomes important in cardiac tamponade. It is discussed in more detail elsewhere.  

**Mechanism of Peripheral Resistance Modulation**

There is little doubt that the sympathetic nerves constitute the efferent pathway, and our experience is that it is blocked by tetraethylammonium but not by atropine. The purest form of this type of pressure variation is the Traube wave occurring in apnea (fig. 5). However, large respiratory variation in a supine subject often appears to depend more on resistance than on output modulation (figs. 1, 3, and 4a), and analysis of the records suggest that resistance changes usually play some part in the more conspicuous swings. The two influences are in general timed to reinforce each other (fig. 4). The question immediately arises how this synchronization is brought about. There is no shortage of possible synchronizing influences: for example, impulses might radiate from the respiratory to the vasomotor center, or might arise in lung stretch or other receptors sensitive to respiratory movements. In the first case, passive ventilation by lung inflation should abolish the synchronization, while in the second, it should invert the relationship between stroke volume and resistance changes. We have performed a number of experiments with passive lung inflation and while we have consistently produced the expected reversal of stroke volume modulation, the effects on the resistance changes have been less clear-cut as the blood pressure patterns have tended to be unstable (fig. 9). On the whole the results do not support the idea that lung stretch or similar receptors supply the sole "keying-in" stimulus.

Synchronizing impulses might also arise from receptors anywhere in the vascular system between the venae cavae and the carotid sinuses. Those near the right heart would tend to maintain the relationship between resistance change and respiratory phase; those near the left heart that between resistance change and stroke volume change. Since the two changes tend to reinforce each other the latter arrangement would imply that the baroreceptors were acting at certain frequencies not as a stabilizing negative feedback but as a positive feedback mechanism. This is made unlikely by the following observation. Sudden digital compression
of both femoral arteries causes a rise in brachial pressure of 5 to 10 mm. Hg. Rhythmic compression and release of the arteries over a wide range of repetition rates failed to evoke any regenerative effects in the three subjects tested.

Figure 10 shows the effect of short isolated inspirations and expirations (square-wave breathing). Since whether expiration is followed by constriction or dilatation depends on the repetition rate, it appears that the connection between resistance change and respiratory act cannot be a simple one.

One must conclude that the nature of the interaction between respiration and peripheral resistance remains obscure. However, it is worth attempting to describe succinctly the over-all action of the system.

**Formulation of the Over-all Action**

The following is an attempt to frame a general viewpoint conformable with the facts.

There exists a mechanism tending to produce rhythmic waxing and waning of vascular tone at a rate of around six a minute. This mechanism we may call the "Traube oscillator." For our present purposes it is immaterial whether this is, as suggested by Guyton and Harris, a feedback oscillator or whether the rhythm is dependent on some quasi-autonomous pacemaker in the nervous system. We postulate some form of loose coupling between respiratory events and this oscillator so that the oscillator frequency is entrained by the respiratory rate. When the latter is near the "free running" oscillator rate of 6 per minute the magnitude of the oscillation is greatest: as the rate moves away the magnitude decreases along some sort of resonance curve. At fairly remote rates the subharmonic nearest to the free running rate may appear. This in its turn may be entrained over short ranges (fig. 6). The type of behavior here described is well recognized in coupled electronic oscillators and although the analogy may seem farfetched it is difficult to see how any simpler one will explain, for example, the happenings in figure 6.

Thus at rates of, say, 20 a minute, respiratory variation is small and what there is is largely due to stroke volume modulation. As the rate slows the stroke volume modulation increases (and the phase lag decreases) as explained above. Simultaneously the contribution of peripheral resistance modulation increases as the Traube frequency is approached. A normal subject achieves maximal blood pressure swings by breathing at 6 per minute in the upright posture. He thus produces a large stroke volume modulation and reinforces this by resonating the Traube oscillator.

![Figure 10](http://circ.ahajournals.org/)

**Fig. 10.** The effect of "square-wave" breathing at different frequencies. Small transient spikes are seen at the moment of expiration: they are due to direct transmission of the intrathoracic and abdominal pressure impulse to the arterial system.

Familiarity with the normal behavior is obviously necessary for the early recognition of abnormal patterns of respiratory blood pressure variation, for example, pulsus paradoxus, and it was with this aim that this study was undertaken. We suggest that the results have an additional interest as indicating the complexity of circulatory interactions and the scope for further work in apparently simple phenomena.
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Summary

The normal behavior of the respiratory blood pressure variations with alterations of respiratory rate and pattern, and of posture is described.

An analysis of the results suggests that variation in stroke volume and in peripheral resistance both contribute to the larger swings of pressure.

The mechanisms are discussed in the light of some simple experiments and a formulation of the general pattern of their interaction is attempted.

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