Standing-Wave Components in Arterial Pulses of Hypothermic Dogs

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Anesthesitized dogs were artificially cooled to temperatures at which a marked prolongation of systole was observed. Under these conditions, it was possible to identify undulations in pressure during the prolonged systole without confusion with incisural pressure changes. These pressure undulations in the aortic arch were observed to occur simultaneously with but in opposite phase relations to the primary peak and dicrotic oscillations recorded from the femoral artery. This evidence offers additional support to the standing-wave theory.

Early observations of arterial pulse forms utilizing sphygmonographie technics yielded little valid information because of the distortions introduced by inadequate recording methods. It was not until the work of Otto Frank that a rigorous analysis of the instrumental requirements was presented and a reliable recording system developed. His observations led Frank to suggest that peripheral arterial pulses appeared to represent a transmitted pressure wave modified by a low frequency resonance of the arterial system, comparable to that observed in attempting to record pressure pulses with a low frequency manometer.

An intensive analysis of accurately recorded pulse forms became possible when Carl J. Wiggers, a student of Frank, combined the theoretical specifications established by his teacher with the technical ingenuity of a gifted experimenter in the design of manometers of high fidelity which could be readily adapted to a wide variety of experimental situations. On the basis of his experience with the recording of peripheral pulses from many sites through a wide spectrum of different experimental conditions, Wiggers described the transformation of the central pulse into the peripheral pulse forms in terms which in general agreed with the concepts of Frank. Wiggers, however, tended to emphasize the great variety of local reflection phenomena which gave pulses from different sites their unique characteristics, and did not develop further the suggestion that there existed a major resonance phenomenon in the aortic system as a whole.

In 1939 Hamilton and Dow extended the idea that a resonating system existed when they described the dicrotic oscillations observed in the femoral arterial pulse of the dog as representing a standing-wave oscillation in the femoral-aortic system. Rigorous proof of their concept would require the demonstration of 3 characteristics of this wave system: 1. A node must exist where the energy flux corresponding to the standing wave is manifest by surges in flow without any corresponding pressure undulations. 2. Below this node, the pressure oscillations must occur simultaneously in time and increase progressively in magnitude as the distance from the node increases. 3. Above the node, there must be a similar pattern of pressure oscillation occurring simultaneously but with opposite phase relationships to the pressure oscillations below the node.

Definite indications of there being such a node in the aorta in the region of the diaphragm have been presented. Pressure recordings reveal no detectable pressure undu-

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ARTERIAL PULSES OF HYPOTHERMIC DOGS

lations and yet flow studies reveal the predicted surges in flow in this region. The second characteristic of simultaneous pressure peaks below this node was documented by Hamilton and Dow in their original publication. Minor discrepancies in the exact timing of these peaks have been attributed to local distortions of the basic pulse form of the type described by Wiggers. The third characteristic has been much more difficult to establish, due to the fact that the standing wave should appear in the thoracic aorta above the node at about the time of the incisura. Attempts to interpret this region of aortic pulses can be justifiably criticized because the exact nature of changes occurring in the region of the incisural waves, and therefore, one cannot be sure of the precise nature of the physical system producing these waves.

It has been noted, however, that observations of central aortic pulses during hypothermia may avoid this confusion of the incisural waves with the standing waves because of the marked prolongation of the duration of systole. The present study was designed to extend these observations and establish with greater certainty the nature of the aortic pressure oscillations observed in hypothermia.

METHODS

Dogs were anesthetized with sodium barbital following morphine sedation, and prepared for recording arterial pulses by means of optical membrane manometers. The pressure in the aortic arch was obtained through a sound passed down the left carotid artery. In some experiments lateral pressures were recorded from a femoral artery by cannulating a side branch just above the knee. In other experiments, a no. 10 cardiac catheter was introduced into the femoral artery in the midthigh region and passed centrally so as to record pressures from successive sites in the aortic-femoral system. Accurate timing of pulse complexes was facilitated by simultaneously recording the oscillations of a 50 cycle tuning fork.

After control recordings, the animals were progressively cooled by either an arteriovenous shunt, passed through a coil immersed in an ice bath and/or plastic bags filled with ice cubes, inserted into the abdominal cavity through a midline incision. To minimize the intense shivering observed in the neighborhood of 30 to 34 C, animals were either given additional doses of barbital or were immobilized with d-tubocurarine superimposed on light barbiturate anesthesia. Such precautions ceased to be necessary after temperature dropped below this range. Rectal temperatures were registered continuously with a thermistor thermometer. The control temperatures reported for these animals are somewhat subnormal for the dog because of the fact that no precautions were taken to maintain normal temperatures during the period of preparation of the animal.

RESULTS

Figure 1 illustrates the changes in pulse form associated with hypothermia. In this experiment the alterations in pulse form were essentially independent of changes in pressure, since the aortic arch pressure of 113/88 mm. Hg at 36.7 C. was maintained at 110/82 with a rectal temperature of 27.0 C. approximately 4 hours later.

To proceed with a dynamic analysis of these pulses, evidence must be presented to establish that the transformations observed are not to be attributed to alterations in the elastic properties of the aortic system as the temperature is lowered. One indication of this was obtained from measurements of pulse transmission time. In 5 animals in which recordings were made at identical sites while rectal temperature was lowered from an average value of 36.3 to 24.4 C, the average transmission time between the foot of the pulse wave in the aortic arch and the foot of the pulse wave in the femoral artery exhibited a minimal change of from 72 to 77 msec. This slight slowing can be attributed to some reduction in central diastolic pressure at the lower temperatures, which fell from an average of 111 mm. Hg to a level of 85 mm. Hg. These data offer no evidence of any marked change in the properties of the aortic system.

A similar observation of greater relevance to the present analysis is shown in figure 2. In this experiment a study was made of the progressive prolongation of the duration of systole as measured by the time interval between the start of the upstroke of the aortic pulse and the incisura. These values are compared with the time interval between the start
of the aortic upstroke and the bottom of the dicrotic dip observed in the femoral pulse. (For reasons which will be clarified below, the latter measurement was confined to the first dip in those pulses where a double dicrotic notch was exhibited.) As seen in figure 2, although a marked prolongation of the interval to the aortic incisura was observed as temperature fell below 30 C, there was only a minimal prolongation of the interval to the diastolic dip. In agreement with the standing-wave hypothesis, this demonstrates that the dicrotic dip cannot be attributed to a transmission of the incisura. Furthermore, insofar as the timing of the dicrotic dip may be used as an index to the frequency of the standing wave, figure 2 indicates that this wave frequency is not altered appreciably as hypothermia develops.

With this relationship in mind, we may now return to consider more closely the standing-wave components in figure 1. In the upper left hand tracing are the aortic and femoral pulses prior to initiation of artificial cooling. A recording in which the optical beams overlapped was purposely selected to emphasize that the femoral peak, which corresponds roughly to the standing-wave peak, occurs at the same time as the central incisura in the typical normothermic animal. Precise interpretation of such pulses is obviously extremely difficult. In the lower left hand tracing are the pulses recorded after rectal temperature had been lowered to 30 C. This pair of tracings reveals distinctive changes in the form of the aortic arch pulse. The sharp, late systolic drop in aortic pressure has been replaced by a rolling plateau, while the incisura is now riding the crest of a wave instead of following it, as in the initial recording. The pulse in the upper right, recorded at a still lower temperature, shows a continuation of this trend, with the incisura clearly following an upward surge of pressure in the aortic arch. A new feature has appeared in the femoral pulse in the form of a double undulation which replaces the "dicrotic notch." The first and lesser of these 2 dips occurs almost coincident with the central incisura, while the second and more prominent dip follows the incisura by 109 msec. The latter interval agrees closely with the transmission time for the foot of the pulse wave of 101 msec, and thus identifies this second dip as the transmitted incisura.

The recording in the lower right of figure
ARTERIAL PULSES OF HYPOTHERMIC DOGS

Fig. 3 Left. Hypothermic pulses recorded from two other animals by technique used for figure 1.

Fig. 4 Right. Superimposed tracings obtained from successive recordings at 24.2 C. as catheter was withdrawn from region of diaphragm down abdominal aorta and into femoral artery. Figures to the left of each pulse indicate distance in centimeters of catheter tip below diaphragm; lowest tracing is pulse recorded simultaneously from aortic arch.

1 shows a fuller development of the hypothermic pulse pattern. In the aortic tracing, it is now evident that there is a small negative undulation following the initial anacrotic crest which occurs simultaneously with the femoral peak. This is followed by a rolling summit in the central pulse occurring simultaneously with the first of the dicrotic valleys in the femoral pulse. The second dicrotic valley continues to occur at a point in time which corresponds with the conduction time to be expected of a transmitted incisura.

Figure 3 illustrates pulses from 2 other animals to indicate both the variability in the pulse forms observed as well as the common features which may be distinguished in them. The tracings on the left were obtained from an animal in which central pulse pressure became less as hypothermia developed, with a corresponding diminution of the magnitude of standing-wave components, as has been described. In this circumstance the standing-wave undulations are barely discernible in the aortic pulse tracing at 25.8 C. and are greatly reduced in the femoral pulse, although the same relationships as described for figure 1 can still be identified.

The right hand tracings, by contrast, show an animal which maintained a vigorous pulse throughout the experiment. The initial recording was not made until the animal had been cooled down to 32.6 C., and it is already evident that the aortic incisura is riding the crest of a positive wave which follows a negative dip on the systolic summit. At 24 C. there is a definite trough in the anacrotic rise of the aortic arch pulse, well in advance of the systolic peak, a trough which occurs simultaneously with the initial femoral peak.

Another method of identifying the components in the hypothermic pulses is by a mapping experiment of the type illustrated in figure 4. The temperature of this animal was lowered to 24.2 C. and then pulses were recorded from the aortic arch (lowest tracing) and from the aorta at the level of the diaphragm (uppermost tracing), the latter pres-
sure being obtained through a catheter passed up the aorta from the femoral artery. It will be noted that this uppermost tracing exhibits a smooth summit lacking any indication of standing-wave undulations, and hence it is assumed that this corresponds approximately with the node of the standing-wave system. The catheter was then withdrawn in 5 cm. steps and successive recordings obtained from the abdominal aorta and iliac system. These successive tracings have been superimposed in figure 4, using the foot of the simultaneously recorded aortic arch pulses to synchronize the time relations of the individual tracings. Although there was some minor variation in cardiac dynamics from beat to beat, the main features of the standing-wave system can be traced through the system with such recordings. The second tracing from the top, recorded 5 cm. below the assumed node, still demonstrates no distinct waves, but the peak has obviously been distorted from the symmetrically rounded summit observed in the top tracing. Scanning records downward indicates that this distortion develops progressively into an augmented peak in early systole, followed by a distinct trough in midsystole. The “standing” characteristic of these undulations is indicated by the fact that, although the foot of each successive pulse is displaced further in time, as would be expected of a traveling wave, the systolic peaks and valleys are essentially simultaneous, with no progressive temporal displacement at the more distal recording positions. Since the foot of each successive pulse starts its rise at a progressively later point in time, while the peak remains “stationary” in time, the result is a progressively earlier peaking of the pulse wave. In contrast to the timing of these peaks and troughs recorded below the node, the aortic arch recording at the bottom reveals the predicted phase shift of 180°. There is a retardation in the rapid anacrotic upstroke which occurs synchronously with peak of pressure below the node. This wave system therefore agrees well with the predicted characteristics of the standing wave, the prolongation of systole produced by the hypothermia merely serving to delay the incisural pressure fluctuations so that they do not confuse the picture.

**Discussion**

It is beyond the scope of this report to review all the evidence bearing on the standing-wave hypothesis, or to discuss the theories which have been proposed to account for its genesis. Nevertheless, widespread confusion as to the connotations of the standing-wave interpretation prompts a restatement of the basic concept. Some confusion may relate to the fact that in their original publication, Hamilton and Dow made use of the unfortunate analogy of an organ pipe. The phenomenon of sound wave generation in an organ pipe fails to parallel the situation in the aortic system on two important counts. The walls of an organ pipe are rigid; the oscillatory phenomena are dependent primarily upon the elasticity of the gas in the lumen. In the aorta, it is the incompressible fluid in the lumen which is effectively rigid, while the vascular wall contributes the elastic component. More important, the organ pipe is used to produce a sustained, driven oscillation in which the fundamental and associated harmonics are continuously regenerated. This analogy has led to the serious error of assuming the heart rate to be the fundamental frequency of the standing wave, and the dicrotic oscillations the corresponding harmonics. Were this the case, changes in heart rate should radically alter the frequency of the standing wave and the ability of the aortic system to contribute harmonic resonance. The fact is that alterations in heart rate do not in themselves have any influence on the standing wave frequency, as might be deduced from an inspection of figure 1.

Although it is difficult to cite an adequate analogy, a more fitting comparison would be the striking of a tubular orchestral chime. Here, the frequency of the initial driving force is essentially irrelevant to the frequency
ARTERIAL PULSES OF HYPOTHERMIC DOGS

response of the chime. Once the elastic component in such a system has been suddenly strained by the striking process, there arises an oscillation whose characteristics are dependent upon the elastic properties of the chime; the frequency of striking is dictated by the rhythm of the music and does not influence the pitch of the chime. Similarly, the impact of cardiac ejection strikes the elastic aortic system, and this is thrown into an oscillation whose frequency is unrelated to heart rate, but rather is determined by the elastic properties of the vascular tree.

The hypothermic pulses support this concept in several respects. The timing of the dicrotic oscillations is not influenced by marked prolongations of systole or the consequent delays in the central incisura and other dynamic phenomena associated with the termination of systole and valve closure. These oscillations must, therefore, causally relate to the initial impact of cardiac ejection rather than to other dynamic events in the later part of the cardiac cycle. Secondly, hypothermic pulses demonstrate that there are pressure undulations in pulses recorded from the thoracic aorta which cannot be explained by any known characteristics of cardiac ejection. Analysis of the progressive development of these pulse forms as body temperature is lowered (fig. 1) indicates that these undulations do indeed correspond with the previously described "preincisural slump" and "post-incisural hump" observed in normothermic pulses, and that they do occur simultaneously in time, but 180°C. out of phase, with the peak and dicrotic dip in the femoral pulse. These relationships are explicitly predicted by the standing-wave hypothesis. It may be conceded that existing evidence is inadequate to offer substantial proof of a precise description of the causative phenomena involved in the genesis of this standing-wave system. On the other hand, uncertainty as to its exact etiology should not cloud the reality of its existence.

Recognition of the standing-wave phenomenon opens the door to considerable additional insight into further details of the dynamics of pulse waves. One example of this is seen in the transmission of the incisura in the hypothermic pulses. Comparison of the two pairs of pulses on the right of figure 1 reveals little difference in the pattern of the aortic incisura between the 29°C. recording and the 27°C. recording. Yet the femoral pulses show only a small incisural dip in the 29°C. recording but a prominent rolling transmission of the incisura at 27°C. The explanation for this difference would appear to relate to resonance of the standing-wave system with the transmitted incisura. At a temperature of 29°C., the transmitted incisura arrived at the femoral bed at about the crest of the second cycle of the standing wave; hence there tended to be interference between these two waves and the resultant pressure change was minimal. At 27°C., however, systole was prolonged to the point that the transmitted incisura did not reach the femoral bed until a point corresponding roughly with the second trough of the standing wave. There resulted a reinforcement of the standing wave by this transmitted incisural wave, with a consequent exaggeration of the incisural wave, followed by a clearly discernible persistence of the standing-wave undulation throughout the remainder of diastole.

Extrapolation of these observations back to the normothermic condition reveals that the normal incisura, while not the primary cause of the dicrotic dip, is in effect arriving in the femoral bed at about the same time and therefore serves to reinforce the standing-wave oscillation that is basically responsible for this dicrotic dip. Under different dynamic states, the degree of this reinforcement will be altered as a function of the duration of systole and the transmission rate of the pulse which, together with the natural frequency of the aortic-femoral system, determine the arrival time of the incisura in reference to the standing-wave oscillation. Some such explanation could resolve the paradox that vasodilation, as induced by high fever or amyl nitrite, acts to accentuate dicrotism, whereas vasodilation induced by acetylcholine tends to minimize dicrotism. Similarly, in so far as the pressure contour resulting from the pattern
of cardiac ejection contains frequency components which do or do not approximate the natural frequency of the aortic-femoral system, there will result a reinforcement or interference with the full development of the standing wave system.

**Summary**

This analysis of arterial pulses in hypothermic animals offers confirmatory evidence of the presence of a simultaneous pressure oscillation, or standing wave, in the lower aortic-femoral system. It also presents the first uncomplicated evidence of a similar pressure oscillation in the aortic arch. As theory predicts, these undulations in aortic arch pressure occur synchronously with the lower oscillations but with opposite phase relations. The documentation of the existence of a standing-wave system in the aortic-femoral system is therefore complete.

**Summario in Interlingua**

Le presente analyse de pulsos arterial in animales hypothermic offer provas confirmatorii pro le presentia de un simultanea oscillation de pression, i.e. un "unda stationari," in le systema aorto-femoral inferior. Ilo etiam presenta le prime non-complicate prova pro le presentia de un simile oscillation de pression in le arco aortic. Como le theoria lo predice, iste undulaciones del pression in le arco aortic occurre synchronemente con le oscillations inferior sed con opposite relationes phasic. Per consequente, le documentation del existentia de un sistema de unda stationari in le systema aorto-femoral es complete.

**References**

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