Pressure Wave Transmission along the Human Aorta

CHANGES WITH AGE AND IN ARTERIAL DEGENERATIVE DISEASE

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ABSTRACT
Pressure waves were recorded in the proximal aorta, iliac artery, and intermediate sites in 39 patients, aged 6 to 69 years, during diagnostic catheterization. In children, amplitude of the pressure wave increased progressively along the aorta, and a prominent diastolic wave appeared in the distal aorta and iliac artery. These changes in contour were associated with fluctuations in modulus and phase of the pressure wave harmonics. Alterations in the pressure wave during transmission became progressively less with increasing age, and the wave was transmitted virtually unchanged in the older patients with arterial degenerative disease. Percentage amplification (A) of the wave between aortic arch and iliac artery and age (x) were inversely related (A = 58.9 - 0.90x; P < 0.001). Transmission time (T, msec) from diaphragm to iliac artery shortened with age (T = 63.5 - 0.62x; P < 0.001) indicating a decrease in arterial distensibility. The relationship between amplification and transmission time in the abdominal aorta and iliac artery was A = 1.04T - 16.7 (P < 0.001). Findings are attributed to a decline in peripheral reflection coefficient resulting from decreased distensibility of peripheral arteries with age and in arterial degenerative disease. The functional effects of decreased distensibility are to impair the efficiency of the arterial system in accepting pulsatile flow, and so to increase the load presented to the left ventricle.

ADDITIONAL KEY WORDS
impedance wave reflection heart work arteriosclerosis blood pressure harmonic analysis windkessel pulse pressure wave velocity
proximal aorta to iliac artery in human subjects, to demonstrate the changes in wave transmission that occur with age and accompany arterial degenerative disease, and to attempt an explanation of these changes on the basis of previous studies on models and experimental animals.

Methods

Forty-three patients whose ages ranged from 6 to 71 years were studied during diagnostic cardiac catheterization or during diagnostic arteriography. All patients were conscious but sedated and lying supine and flat. Adults were premedicated with pentobarbital, 50 to 100 mg, meperidine, 50 to 100 mg, and atropine, 0.2 to 0.4 mg, and children with meperidine, 2.75 mg/kg, promethazine, 0.7 mg/kg, and chlorpromazine, 0.7 mg/kg.

In 38 patients the catheter was inserted percutaneously into the femoral artery in the groin and advanced to the aortic arch or ascending aorta. In one patient, the catheter was inserted into the right brachial artery and threaded down to the iliac artery. Catheter positions were determined fluoroscopically. Records were taken at short time intervals in the aortic arch, descending thoracic aorta at the level of the dome of the diaphragm (tenth thoracic vertebra or bottom of ninth thoracic vertebra), abdominal aorta at the second lumbar vertebra, and in the iliac artery at the level of the sacroiliac joint. The whole procedure was completed in 3 to 5 minutes. In three patients, difficulties were encountered in inserting the catheter, and records were taken only at the peripheral sites. In 19 patients pressure records were taken in the ascending aorta as well as at the other four sites. The catheter was the one used for the diagnostic studies; it was 1.2 to 3.0 mm o.d., was made of barium-impregnated polyethylene or dacron, and usually had two or more side holes within 1 cm of its open tip. Catheters used for selective celiac or renal arteriography had a curved end to engage the vessel. Frequency response of the catheter-manometer system was determined at the completion of each study by the pressure-step or "pop" method (9). Damped natural frequency was 11 to 36 cps (average 21 cps), and damping coefficient varied between 0.15 and 0.40. Pressure was recorded by a Statham P23Db transducer on an Electronics for Medicine photographic recorder. Comparisons were made between pressure waves which corresponded as closely as possible with regard to cycle length, mean blood pressure, and timing in the respiratory cycle. Selected pressure waves were digitized manually at 0.02-second intervals. Harmonic content of the waves was determined by Fourier analysis (9) using a digital computer; appropriate corrections were applied to the moduli.
and phases of the harmonics for manometer frequency characteristics.

In most patients, the distance between recording positions was measured as the distance the catheter was withdrawn. This distance did not always represent arterial length between the recording sites when the catheter took a direct course around the aortic arch or when the curved end (used in selective renal or celiac arteriographic studies) straightened out during withdrawal. Because of the uncertainty of these measurements, and because pressure was measured at relatively constant anatomical positions in all patients, transmission time was used as an (inverse) index of the velocity of the pulse wave when making comparisons within the whole group. Transmission time between two pressure-recording sites was measured by subtracting the delay between the R-wave peak in the electrocardiogram and the foot of the wave at one position (averaged over 5 to 10 beats) from the delay between the R-wave peak and the foot of the wave at the second position (again averaged over 5 to 10 beats).

In all adults and in one child, pressure records were taken after the diagnostic procedure, 6 to 20 minutes after injection of radiopaque contrast material. Injection of hypertonic radiographic contrast material is known to alter circulatory dynamics (15, 16). To investigate the influence of contrast injection of pulse wave transmission, pressure records were taken at the different sites before and after injection in 19 patients.

At the completion of this study, films from all patients were reviewed by one of us (C.M.), who was at the time unaware of the pressure transmission findings. The radiological evidence of arterial degeneration (tortuosity of the vessel, narrowing and irregularity of the lumen) in the thoracic aorta, abdominal aorta, and iliac artery was arbitrarily graded 0 to 4. Five patients had definite clinical evidence of arterial disease, either giving a history of major arterial occlusion or manifesting signs of arterial narrowing or obstruction. All but one (whose films are not available) had radiological evidence of degenerative disease affecting the distal aorta, iliac artery, or both.

**Results**

Figure 1 shows pressure waves recorded at different positions between the proximal aorta and iliac artery in three children aged 13, 11, and 10. The first child (R.C.) had aortic stenosis, the second (K.S.) had a normal aortic valve, and the third (D.H.) had aortic incompetence. All presumably had healthy arteries. Despite differences in the pressure wave generated in the proximal aorta, changes in the amplitude and contour of the wave during transmission were similar in all.

In the 11 children studied, pulse pressure increased by an average value of 55% between the aortic arch and iliac artery. A diastolic pressure wave was obvious in the abdominal aorta and iliac artery of 10 of the 11 children. The greatest difference from these normal childhood patterns was seen in patients with arterial degenerative disease. Figure 2 shows pressure waves recorded between the proximal aorta and iliac artery in three adults aged 69, 59, and 67 who had clinical and radiological evidence of arterial degeneration. In contrast to the children, there was little or no alteration in amplitude or contour of the wave.
between the proximal aorta and the iliac artery. At all positions, pressure rose to a sharp systolic peak then fell in an almost exponential fashion during late systole and all of diastole.

Progressive changes were noted in pressure wave transmission with age in the absence of any clinical or radiological evidence of arterial degeneration. In older patients, the pressure wave was transmitted virtually unaltered, as it was in the patients with degenerative disease (Fig. 2); in younger adults, wave transmission characteristics were intermediate between those of children and these patients. While a diastolic wave was obvious in the distal aorta and iliac artery of 10 of the 11 children, it was not seen under control conditions in any vessel of the other 32 patients. The diastolic part of the wave was concave upwards in all vessels of the older patients and flat in the younger adults.

The records presented here were obtained with the same transducer and connections as used for routine diagnostic studies. Natural frequency of the manometer assembly was relatively low (11 to 36 cps). Some of the pressure fluctuations seen were obvious artifacts due to manometer resonance. This is illustrated in Figure 3, which shows waves recorded at two points with a relatively low frequency manometer and the same waves resynthesized from the first ten harmonics after correction for manometer artifacts, using a digital computer. Distortion of the pressure wave was relatively minor and easily identifiable at this heart rate; this would not have been so had the natural frequency been any lower, the heart rate much faster, or the manometer been used to record pressure waves (such as in the atrium) that have a relatively high frequency content (17).

It is instructive to compare the original and resynthesized waves in Figure 3 (after manometer correction had been applied) with similar data published by Warner (Distortion of pressure waves by arterial walls, Circulation Res. 5: 79, 1957) and reproduced in Physiology and Biophysics by Buch and Paton (Philadelphia, W. B. Saunders, 1965, p. 604). In the published figure, lack of correspondence between the original and resynthesized waves is apparently due to an error in the analysis or resynthesis, since it is possible to obtain a better fit to Warner’s original wave with 10 harmonics than he showed.

The revised version of Warner’s figure and two tables giving specific data on the patients in our study have been deposited as Document number NAPS-00079 with the National Auxiliary Publications Service of the American Society for Information Science, c/o CCM Information Sciences, Inc., 22 West 34th Street, New York, N. Y. 10001. A copy may be secured by citing the Document number and remitting either $1.00 for microfiche copies or $3.00 for photo copies. Advance payment is required. Make checks or money orders payable to ASIS-NAPS.
Changes in the contour of the wave during transmission were always associated with alterations in amplitude, and the degree of amplification of the wave between the aortic arch and iliac artery (i.e., the ratio of pulse pressure in the iliac artery to pulse pressure in the aortic arch) was related to the amount of change in wave shape. When amplification was high, changes in contour were similar to those in Figure 1, when amplification was low, changes in contour were similar to those in Figure 2. The relationship between amplification and age is shown in Figure 4. Amplification was highest in the children and fell with increasing age. The adults in this series included a disproportionately large number referred because of real or suspected vascular lesions, but the same fall in amplification was seen even when these patients were not considered. The fall in pressure wave amplification with age was obvious only because of the wide range in age of the patients studied. The most obvious change with age was in the younger patients; for patients aged 6 to 35 years, the regression line fell steeply (\( Y = 68.6 - 1.37X; r = -0.67; P < 0.01 \)), while for patients over 35 there was no significant correlation between amplification and age. For all ages, the regression line is given by the equation \( Y = 58.9 - 0.90X; r = -0.78; P < 0.001 \). In patients with clinical or radiological evidence of arterial disease, amplification tended to be lower than in others of the same age. Of the patients over 35 years of age, seven were classified as having grades 2 to 4 radiological evidence of arterial degeneration in the abdominal aorta or iliac artery, and in these amplification averaged 4.7%, whereas in the patients with grades 0 to 1, amplification averaged 11.7%. In the five patients with definite clinical evidence of arterial degenerative disease, amplification averaged 2%, whereas in the three patients without such evidence in the same age group, amplification averaged 17%. There was a significant correlation (\( r = 0.55; P < 0.01 \)) between amplification and the radiological assessment of arterial degeneration.

Besides the fall in amplification, increasing age was associated with a decrease in transmission time from the aortic arch to iliac artery (Fig. 5). The greatest changes were in the distal aorta and iliac artery, transmission time being little altered in the thoracic aorta. This fall in transmission time with age reflects an increase in pulse wave velocity. A fall in transmission time between the lower thoracic aorta and iliac artery from an average value of 61 msec in patients under 15 to an average value of 28 msec in the patients over 45 indicates a twofold increase in wave velocity over this interval. Actually, wave
velocity probably increased more than this because of the shorter distance between the two sites in the younger patients (average height of the patients under 15 was 57 inches and of the patients over 45, 68 inches). The increase in pulse wave velocity with age is attributable to a decrease in arterial distensibility (9, 12).

In Figure 6 amplification of the pressure wave between arch and iliac artery is plotted against transmission time over the same interval (A) and against transmission time over the distal aorta and iliac artery (B). For A, the equation for the regression line is $Y = 0.836X - 32.0; \ r = 0.65; \ P < 0.001; \ B$, $Y = 1.039X - 16.7; \ r = 0.69; \ P < 0.001$. One might expect that correlation would have been better had amplification been related to wave velocity rather than to transmission time because the two outlying points on the graph with unusually low transmission times are from two children who were less than 4 feet tall. The correlation for the relationship between amplification and (transmission time + body height) was 0.75 ($P < 0.001$) for (A) and 0.78 ($P < 0.001$) for (B).

Figure 7 shows the changes in moduli (amplitudes) of the first five harmonics of the pressure wave between the proximal aorta and iliac artery of the three children whose waves appear in Figure 1, and contrasts these with the alterations in pressure moduli of the three adults with arterial disease whose waves are shown in Figure 2. In the three children, there were fluctuations in pressure moduli along the aorta, with all tending to increase toward the periphery. No such fluctuations were seen in the patients with arterial disease; in these, pressure moduli remained relatively constant or increased only slightly between the proximal aorta and iliac artery.

Figure 8 shows differences in pressure phase between recording sites plotted against harmonic number in a child (left) and in an adult with arteriosclerosis (right). If the pressure wave was transmitted unchanged, phase
Amplification of the pressure wave between aortic arch and iliac artery plotted against transmission time between aortic arch and iliac artery (A) and against transmission time between lower thoracic aorta and iliac artery (B). Open circles denote patients without clinical evidence of vascular lesions; solid circles, patients with known vascular lesions.

Amplification would be expected to vary linearly with harmonic number. Phase difference did vary approximately linearly with harmonic number in the adult patient. The fluctuations in phase difference in the child reflect the differences in shape of the pressure wave at the various recording sites. If the distances between recording positions were known and if the pressure waves at the different sites had been sampled with a fixed relationship to the electrocardiogram, one would be able to describe these phase differences in terms of apparent phase velocity. There would have been large fluctuations in apparent phase velocity with harmonic number over the various arterial segments in the child, whereas apparent phase velocity would have remained relatively constant with harmonic number in the adult.

Fluctuations in pressure modulus with distance and in apparent phase velocity (or phase difference) with frequency indicate the presence of reflected waves in the arterial pulse. The absence of large fluctuations indicates that reflected waves are small or absent.

In 19 patients (14 adults and 5 children), pressure records were taken before and 6 to 20 minutes after injection of radiopaque contrast material. In the adults, the average difference in amplification over corresponding arterial segments was less than 1%. In the children, amplification of the pressure wave averaged 60% before and 47% after contrast injection. The data presented in this paper on pressure wave transmission in adults were obtained at the conclusion of the diagnostic procedure following contrast injection. Data from children were obtained in all but one case before any contrast was injected. While it is obvious that injection of radiopaque contrast material can alter wave transmission, it...
At top, changes in moduli of the first five harmonics (numbered at top center) of the pressure waves in the three children whose waves are shown in Figure 1. At bottom, changes in moduli of the first five harmonics of the pressure waves in the three patients with arteriosclerosis whose waves are shown in Figure 2. Recording positions 1 through 5 are the same as in Figures 1 and 2. Mean heart rates: R.C., 95/min; K.J., 75/min; D.H., 90/min; E.C., 73/min; C.N., 75/min; O.G., 60/min.

Discussion

PRESSURE WAVES IN YOUNG HUMANS AND OTHER MAMMALS

The alterations in amplitude and contour of the pressure wave during transmission along the aorta in children were similar to but less marked than those normally seen in dogs and other mammals (7-11). In dogs, the amplitude of the iliac pressure wave is usually about twice that in the aortic arch, i.e., amplification is approximately 100%, whereas is highly unlikely that the experimental results presented here were significantly affected by this.
HUMAN AORTIC PRESSURE WAVE TRANSMISSION

FIGURE 8

Phase difference between corresponding harmonics of pressure waves recorded at two different sites plotted against harmonic number in a child (left) and in a patient with arteriosclerosis (right). Line with crosses is interval between ascending aorta and aortic arch; with solid circles is interval between arch and lower thoracic aorta at the diaphragm; with open circles is interval between thoracic aorta at the diaphragm, and abdominal aorta at the second lumbar vertebra; with asterisks is interval between abdominal aorta at the L2 level and the distal common iliac artery.

in the 11 children reported here amplification averaged 55%. The changes in pressure modulus and phase along the aorta and iliac artery of these children were also similar to but less marked than those found in dogs and other mammals (9-11).

In young adults, alterations in the pressure wave during transmission were smaller than in children. The data on wave transmission in young adults presented here is in agreement with that already in the literature. Kroeker and Wood (3) found in a group of 12 healthy physicians aged 27 to 41 that the pressure wave was amplified by an average value of 39% between the aortic arch and femoral artery. This figure compares with our average amplification of 32% between the aortic arch and iliac artery in six patients aged 22 to 40 who were being investigated for reasons other than vascular disease. The changes in modulus and phase of pressure waves along the aorta in these six patients were similar to those reported by Luchsinger et al. (6).

The principal factors responsible for alterations in the pressure wave during transmission are wave reflection (3, 6, 7, 9, 11) and the progressive increase in stiffness of peripheral arteries relative to the proximal aorta (6, 10). With regard to wave reflection, the systemic arterial system of mammals behaves like a tube with two closed ends, between which the pressure wave bounces back and forth before being damped to extinction (7-11). In this model, one closed end represents the summation of all reflection sites in the head, neck, and upper limbs and the other closed end represents the summation of all reflecting sites in the trunk and lower limbs (11). The amount of reflection seen in the whole system depends both on the reflection coefficient at each individual reflecting site (9, 10) and on the degree of spatial dispersion of the many individual reflecting sites (11, 18).

Studies on experimental animals (8, 9, 19) indicate that the arterioles are the most important sites of wave reflection in the vascular system. The reflection coefficient at an arteriole depends on the impedance mismatch between the arteriole and artery of supply, being given by the formula

$$R_t = \frac{Z_t - Z_0}{Z_t + Z_0},$$

where $Z_t$ is the impedance of the arteriole to steady flow and $Z_0$ is the characteristic impedance of the small artery of supply (9). (Characteristic impedance is impedance in the absence of wave reflection.)

The differences in wave transmission between children and dogs may be explained as being due to a lower apparent reflection coefficient in the former. It is tempting to suggest that this results from the greater degree of dispersion of reflecting sites in children, because the limbs are longer. This explanation has been advanced to explain differences in wave contour between dogs and Australian wombats (which have relatively
long bodies and short stubby limbs) and the changes in wave transmission in dogs when peripheral arteries are occluded (11). However, the differences between children and dogs could have been due to a lower impedance mismatch at individual arterial terminations or even to smaller changes in arterial distensibility between the proximal aorta and iliac artery.

While differences in wave transmission between dogs and children may be explained (at least in part) on the basis of differences in reflection coefficient secondary to differences in body shape, it is hardly possible to invoke the same explanation for the differences in wave transmission between children and young adults. This might be reasonable if one were comparing infants (whose arms and legs are short in relation to the trunk) with adults. However, the children in this series were all over 6 years of age; 7 of the 11 were over 5 feet tall and all appeared to have attained relative adult dimensions if not full adult size. Here one can only suggest that the reflection coefficient at each individual arteriole was decreased. This could have resulted from a fall in terminal impedance $Z_t$ (which is highly improbable) or to an increase in characteristic impedance $Z_o$. This is more likely. Pressure wave transmission time over the abdominal aorta and iliac artery decreased with age, indicating that the vessels had become stiffer and that their characteristic impedance had increased (9-12). Similar changes occurring further downstream would explain a fall with age in the reflection coefficient at each individual reflecting site.

No data are available on characteristic impedance of peripheral arteries at different ages in man. However, results of vascular impedance in the human femoral artery have been reported (20), and it is instructive to compare these with corresponding results obtained from dogs (19, 21). Patel et al. (20) measured femoral vascular impedance in 11 patients aged 15 to 62 and found in the whole group that impedance modulus above 3 cps settled about a value which was 15% to 20% of the terminal impedance. In dogs, impedance modulus at high frequencies settled about a constant value which was only 3% to 5% of the terminal impedance (19, 21). This constant value of impedance modulus is the characteristic impedance (18). The difference in the ratio of characteristic to terminal impedance between these patients and our dogs was no doubt responsible for differences in reflection coefficient at each arteriole in the vascular bed. A low reflection coefficient in the femoral vascular bed of patients reported by Patel et al. was probably responsible for the small or absent diastolic pressure and flow waves in their records, for the frequent absence of backflow, and for the absence of any distinct minimum of impedance modulus.

One would suspect that there is little difference in the ratio of characteristic to terminal impedance between children and dogs and that the difference only becomes apparent as the human arteries become stiffer with age.

It was surprising to find that decreasing distensibility of the distal aorta and iliac artery relative to the proximal aorta with increasing age (Fig. 5) was associated with a fall in amplification of the pressure wave along the aorta (Fig. 6). One of the explanations for the increase in amplitude of the pressure wave between the heart and periphery is the progressive decrease in arterial distensibility between the proximal aorta and peripheral arteries (6, 9, 10). If this nonuniform elasticity were an important factor in determining alterations in the pressure wave along the aorta in humans, one would expect that amplification of the pressure wave would increase, not decrease, as the distal aorta and peripheral arteries became stiffer with age.

**WAVE TRANSMISSION IN OLDER HUMANS**

The similarities in wave transmission between young healthy humans and other mammals became less distinct with increasing age. In this series, a continuous spectrum was seen, with children at one end and elderly patients with arterial degenerative disease at the other. In the former there was abundant evidence of wave reflection in the arterial...
pulse. In the latter there was practically none. In the elderly patients with arterial degeneration, pressure fell almost exponentially from its systolic peak and there was no secondary wave in any vessel; the wave had the same, or almost the same, contour in the iliac artery as in the ascending aorta or arch; pressure moduli remained relatively constant along the aorta, and the phase difference between pressure waves at different locations varied almost linearly with frequency. The reason for the progressive fall in reflection coefficient with age is probably the increase in stiffness and in characteristic impedance of the peripheral arteries. A further possible mechanism in arterial degenerative disease is the increased spatial dispersion of reflecting sites that would result when new reflecting sites are created at localized regions of narrowing or induration.

In the elderly patients with arterial degeneration, the contour of the arterial pressure wave in different arteries is best explained by likening the arterial system to a different model than used for children, dogs, and other mammals. The model is not a tube in which the wave is travelling back and forth at a finite velocity, with reflection at both ends, but an elastic chamber from which rigid pipes carry blood to the tissues. The elastic chamber represents the thoracic aorta and the pipes the relatively inextensible peripheral vessels. This model is a windkessel and is that first proposed by Stephen Hales (22). The major objection to the windkessel as a model has been that it ignores the finite time taken for the pulse to travel along the arteries and it ignores wave reflection. It is appropriate here only because wave reflection is so small and wave velocity so high. While the windkessel is not a satisfactory model of the systemic arterial system in mammals, it does accurately describe the arterial system in birds (domestic goose and turkey [10, 23]). In these animals, the proximal thoracic aorta is relatively distensible and terminates in relatively inextensible peripheral vessels. The wave shape is little altered between the ascending aorta and iliac artery and the changes in pressure harmonic moduli and phase are almost identical to those in the patients with arterial degenerative disease.

If the alterations in aortic wave transmission that occur with age are due to arterial degeneration, it is conceivable that one might be able to infer the degree of degeneration from studies of wave transmission. Bramwell and Hill (24) suggested that the amount of arterial degenerative disease may be gauged from measurements of pulse wave velocity. This has never gained popularity, probably because the foot of the pressure wave is not easy to identify in records taken routinely in the cardiovascular diagnostic laboratory, where a high natural frequency of manometer systems is usually not attained. It is, however, relatively easy to measure the amplitude of arterial pressure waves accurately, and it should be possible to determine the degree of amplification of the wave quite accurately. If it can be confirmed that amplification of the wave is an index of the degree of arterial degeneration, this might prove clinically useful not only for determining the amount of degeneration in individual patients but also perhaps for assessing the influence of aging, diet, and drugs on the natural history of arterial degeneration during life.

The evidence presented here that suggests a relationship between pressure wave amplification and arterial degeneration is purely circumstantial. To establish the relationship definitely it will be necessary to compare wave transmission data obtained during life to the physical characteristics of the artery determined after death.

**Relevance to Blood Pressure Determination in Peripheral Arteries**

Because amplification of the pulse wave varies with age and is altered by arterial stiffening, the systolic and diastolic pressure levels recorded in the lower limbs do not bear a constant relationship to corresponding levels of the ascending aorta. In a child, a pulse pressure of 40 mm Hg in the femoral or iliac artery would be associated with a pulse pressure of approximately 26 mm Hg in the...
ascending aorta. With advancing years, as the distal aorta and iliac arteries become progressively stiffer, the femoral pulse pressure may remain at 40 mm Hg while pulse pressure in the ascending aorta increases to 40 mm Hg (i.e., by 54%). A high pulse pressure in peripheral arteries is often taken as a sign of decreased arterial elasticity (25), but the foregoing considerations make it clear that it may be a late sign. A better sign would be an increased pulse pressure in the ascending aorta, and this may increase more than 50% before any change in pulse pressure is obvious in the lower limb vessel. These points might appear to have little clinical relevance because blood pressures are rarely taken in the leg. However, the same principles apply when considering wave transmission between the ascending aorta and brachial artery. No data are available on changes in wave transmission with age in the upper limb. Preliminary observations indicate that the changes with age are similar to those described here for wave transmission between the heart and iliac artery. The subject of wave transmission between the ascending aorta and brachial artery is obviously of more relevance to routine blood pressure measurements than that of wave transmission to the lower limbs.

FUNCTIONAL EFFECTS OF ARTERIAL DEGENERATION

The data presented here may help to illustrate the effects of arterial degeneration on the cardiovascular system. The functions of the arteries are twofold: first, to carry blood to the tissues in sufficient quantities for their needs at any time, and second, to damp out with minimal energy losses the flow oscillations which result from intermittent left ventricular ejection. Both functions may be affected by degeneration, the first by narrowing or occlusion of the arterial lumen and the second either by narrowing of the lumen or by stiffening of the arterial wall. The efficiency of the second function may be estimated by calculating the amount of pulsatile energy lost in the systemic arteries (26). No data are available on patients with arterial disease, but from pressure and flow waves in the ascending aorta of young subjects this has been calculated to be approximately 12% of external heart work. An index of the pulsatile energy lost in the circulation may be obtained by measuring the difference between mean systolic pressure in the ascending aorta and mean pressure over a full heart cycle. In the three patients with arterial degenerative disease whose records are shown in Figure 2, mean systolic pressure in the aortic arch was 106, 142, and 174 mm Hg, and mean pressures over the whole cycle were 87, 125, and 142 mm Hg, respectively. The ratio of mean systolic to mean pressure averaged 1.20, indicating that something like 17% of external heart work was lost in pulsatile phenomena. The comparable figure in the five children with normal aortic valves and normal arteries was 8%. These figures indicate only the changes in pulsatile energy losses because they take no account of the shape of the aortic flow wave and the phase relationships between pressure and flow. It is possible that pulsatile energy losses are increased more by arterial disease than these figures suggest, and it is probable that they become even greater still when cardiac output rises, when blood pressure falls, or when heart rate slows (26).

Acknowledgments

We acknowledge with thanks the assistance of Drs. W. R. Milnor, R. Elkins, and R. Gaylor. We also thank the staff of the Cardiovascular Diagnostic Laboratories of the Johns Hopkins Hospital.

References


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Circ Res. 1968;23:567-579
doi: 10.1161/01.RES.23.4.567

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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