SPECIAL ARTICLE

Initial Ventricular Impulse
A Potential Key to Cardiac Evaluation

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Physicians are frequently obliged to evaluate the status of the heart in patients with various types and degrees of cardiac malfunction or disease. Clinical criteria for this evaluation have been largely subjective. For example, the cardiac reserve capacity is most commonly judged by the level of exertion that patients can accomplish without symptoms. In research laboratories, functional capacity of the heart is assessed from measurements of the cardiac response to standard exercise in terms of heart rate, oxygen consumption, electrocardiograms, cardiac output, and other measures. Cardiac output is frequently normal at rest even in patients with severe cardiac disease. Knowledge concerning the maximum cardiac output that a specific patient can attain would be of great interest in evaluating cardiac reserve, but is clearly unattainable in routine clinical diagnosis and evaluation. Techniques for monitoring the clinical conditions of hospitalized patients will ultimately be based on continuous registration of reliable indicators of the performance of vital organ systems.

The value and significance of data obtained by continuously monitoring blood flow and pressure in active conscious dogs are greatly extended by relating observations to human subjects and patients. Comprehensive quantitative descriptions of the performance of the heart illustrate changes in function of the heart and can be readily followed from beat to beat during spontaneous cardiovascular adjustments. By imposition of experimental loads on such animals, immediate effects of different types of cardiac malfunction have been simulated. Studies of this type have disclosed characteristic changes in the pumping action of the ventricles in terms of altered initial impulse. Impulse is a well-accepted physical term, defined as the product of force and time (vide infra). The principal objective of this report is to present certain typical changes in cardiac performance observed in dogs during experimental procedures designed to simulate disease states in man. These criteria may have relations with available clinical procedures (i.e., arterial pulse waves, ballistocardiography, electrokymography, kinetocardiography, etc.). An experimental approach to validating these clinical tests is suggested.

Isotonic Contraction of Isolated Papillary Muscles

Ejection of blood from the ventricles requires that tension be developed and maintained during shortening of the myocardial fibers. Sonnenblick studied contraction of excised papillary muscles during shortening against constant load (isotonic contraction) (fig. 1A). Myocardial fibers shorten at a maximal rate and to a maximal extent under minimal load. As indicated by the slopes in figure 1A, the maximum rate of myocardial shortening occurs at the onset of contraction, so that the initial velocity (I.V.) is always the greatest velocity during a contraction. The initial rate of shortening is most rapid during contraction without load and progressively diminishes as the magnitude of the load is in-

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INITIAL VENTRICULAR IMPULSE

A. ISOTONIC CONTRACTION (SONNENBLICK)

B. RATE OF SHORTENING (SONNENBLICK)

C. VENTRICULAR EJECTION

Figure 1

A. Shortening of isolated papillary muscle against a constant load is most rapid and most extensive when the load is minimal. B. The initial rate of shortening is most rapid with minimal load and diminishes as the load is increased. C. In the intact heart, ventricular pressure represents the load and ejection velocity represents the rate of myocardial shortening. In contrast with isolated myocardial sample, right ventricular ejection against a small load is slower than left ventricular ejection in the face of a large load.

The rate, duration, and total amount of shortening all decrease with increasing load. The reduction in initial velocity of shortening with increasing loads is indicated in figure 1B.

The rapid initial shortening of myocardial strips finds expression in a very rapid initial ejection of blood from contracting ventricle chambers. Other properties of isolated myocardial strips are not directly reflected in the ejection properties of the intact ventricular walls. The right and left ventricles are composed of myocardial fibers that are intricately interwoven in strips and layers. The net rate of myocardial shortening determines the rate of outflow of blood from the contracting ventricle. The load is represented by the pressure developed in the ventricle. Extrapolation from characteristics of myocardial strips suggests that the right ventricular ejection should have higher initial velocity and last longer than left ventricular ejection (fig. 1A). This prediction is not confirmed by direct measurement (fig. 1C).

Right and Left Ventricular Ejection Patterns

The characteristic ejection patterns of the right and left ventricles in conscious dogs can be continuously registered in terms of in-
stantaneous flow rates through the aorta and pulmonary arteries by means of chronically indwelling ultrasonic flowmeters (fig. 1C). Right ventricular contraction develops only a slight increase in pressure (load) and ejects blood at a gradually increasing velocity to a peak value nearer mid-systole. Left ventricular ejection is characterized by a rapid acceleration of the outflow to a high peak early in systole, in spite of the much higher load than that developed by the normal right ventricle. Right ventricular outflow begins earlier and persists longer than left ventricular ejection. In spite of gross differences in ejection pattern, the stroke volumes of the right and left ventricles are remarkably well balanced, even during transitional states at the onset of exercise and during changes in posture.

The gradual rise in outflow rate from the right ventricle into the low pressure pulmonary arterial system may result from sequential excitation and contraction of the inflow and outflow tracts. The right ventricle is served by a single branch of Purkinje fibers. The lateral wall of the right ventricle is excited before the excitation wave invades the conus region (fig. 1B). As the inflow tract begins to contract, blood is displaced into the outflow tract, so that the conus region extends. Right ventricular ejection begins very early in systole because the diastolic pressure in the pulmonary artery is extremely low (7 to 10 mm. Hg). Ejection begins as soon as right ventricular pressure rises above this low level. By virtue of the sequence of right ventricular excitation, the ejection rate gradually ascends to a peak near mid-systole. Since pulmonary arterial pressure normally remains relatively low, even during systole, ejection persists after left ventricular systole has terminated.

In contrast, left ventricular excitation is achieved by rapid spread of activity along a branched Purkinje system distributed widely over the endocardial surface. As the ventricles are abruptly shortened by contraction of the inner layers of myocardium, the circumference of the ventricle rapidly expands. The deeper layers of circumferentially oriented fibers are excited just as they are being actively stretched, a condition which is conducive to production of very high myocardial tension. As the wave of excitation rapidly invades the left ventricular wall, the left ventricular pressure rises abruptly because additional layers of myocardium are rapidly recruited into the contracting mass. Ejection is delayed until the left ventricular pressure exceeds aortic diastolic pressure.

The differences in right and left ventricular ejection patterns have been confirmed by Spencer and Greiss and by Okino and Spencer, using electromagnetic flowmeters. These authors also demonstrated the mechanisms by which the high peak flow rates were developed by the left ventricle. At the onset of left ventricular systole, myocardial tension develops rapidly and ventricular pressure rises abruptly to exceed greatly the pressure at the root of the aorta (fig. 2B). A high pressure gradient rapidly imparts kinetic energy to the contained blood and outflow is accelerated rapidly to a very high peak in the first part of systole. During the remainder of systole the flow rate decelerates. This kind of ejection pattern is more like striking a piston with a mallet than squeezing an orange or milking a cow.

The Ventrices as Impulse Generators

Force may be defined as the product of mass and acceleration (F = ma). The acceleration of blood out of the ventricle is determined by the force (F) exerted by the contracting myocardium; the peak outflow is determined by this force and the time during which it acts. The product of force and time is the impulse (I = Ft). In this discussion, the initial impulse is regarded as the net force acting over the time from the beginning of ejection to the attainment of peak flow rate. Manifestations of this initial impulse are to be found in the various waveforms in figure 2. For example, the onset of systolic ejection from the left ventricle is marked by the rapid increase in pressure in the aortic arch, the rapid increase in aortic flow velocity to a peak in early systole, and the rapid increase in left ventricular pressure. Differentiation of the aortic flow velocity shows the peak ac-
Acceleration occurring as the flow rate ascends to its peak value. Similarly, differentiation indicates the rate at which ventricular pressure increases (dP/dt). All these patterns reflect, to some degree, the magnitude of the impulse generated by the contracting left ventricle during the crucial initial stage of ejection. During the later part of systole, acceleration is below the reference level (negative), indicating that the outflow of blood is slowing toward zero.

Spencer and Greiss \(^8\) related the very rapid acceleration of blood in the aorta to the driving pressure gradient, determined by comparing the pressures recorded just above and just below the aortic valves. This pressure differ-

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**Figure 2**

A. Left ventricular ejection is characterized in terms of the aortic pressure pulse, the aortic flow velocity, the acceleration of the blood through the aorta, left ventricular pressure, and the rate of change of ventricular pressure (dP/dt). B. Initial ventricular ejection produces high ejection velocities early in systole by virtue of the impulse (I = Ft), which produces a steep pressure gradient during the initial phases of ventricular systole. Right ventricular ejection accelerates more slowly and reaches a peak level near mid-systole (according to Spencer et al.).\(^8\)\(^\text{—}10\)
Simultaneous recordings of aortic and pulmonary flow illustrate the differences between left and right ventricular ejection patterns during a variety of conditions in a healthy dog. During the act of standing, three coupled beats appeared, each consisting of a premature contraction without significant ejection, and a long compensatory pause followed by an ejection with a very high ejection velocity (postextrasystolic potentiation). During exercise at 4.5 m.p.h. on a 12-per cent grade, the peak flow velocity increased and the duration of ejection diminished. The stroke volume (area under the curves) was increased only slightly.

The right ventricular impulse is not so great as that of the left, since the blood is accelerated more slowly (average 2,480 cm./sec.) to a peak flow rate of lower magnitude near mid-systole. The ventricular pressure exceeds pulmonary pressure for a longer time, but to a lesser degree than left ventricular pressure exceeds aortic. Thus, the right ventricle also acts as an impulse generator, but does not develop as much impulse, or such high pressure gradients, or such high outflow rates as the left ventricle does. In both ventricles, the initial outflow rate is determined

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**Figure 3**

Simultaneous recordings of aortic and pulmonary flow illustrate the differences between left and right ventricular ejection patterns during a variety of conditions in a healthy dog. During the act of standing, three coupled beats appeared, each consisting of a premature contraction without significant ejection, and a long compensatory pause followed by an ejection with a very high ejection velocity (postextrasystolic potentiation). During exercise at 4.5 m.p.h. on a 12-per cent grade, the peak flow velocity increased and the duration of ejection diminished. The stroke volume (area under the curves) was increased only slightly.
more by the inertial properties of the blood than by hydraulic resistance.\textsuperscript{11}

**Spontaneous Changes in Ventricular Impulse in Conscious Healthy Dogs**

The ejection patterns of the right and left ventricles undergo extensive alteration in form during cardiovascular adjustments to various activities. For example, typical differences between left and right ventricular ejection patterns were recorded from a healthy dog standing quietly (fig. 3). When he lay down spontaneously, three cardiac cycles with very long diastolic intervals terminated in ejections with very high peak outflow rates, but the distinctive differences in the right and left patterns were retained.

In another animal, treadmill exercise at 4.5 m.p.h. on a 12-per cent grade produced obvious changes in ventricular ejection including a greatly increased peak outflow rate from both ventricles. The duration of ejection was diminished. In fact, the reduction in ejection period tended to counteract the effects of high peak outflow rate, so that the stroke volume was increased only slightly or not at all. These changes in ejection pattern are the same sort that accompany stimulation of sympathetic nerves to the heart. The increased impulse must represent greater force developed by the contracting myocardium. Greater force could result from more synchronous excitation throughout the ventricular myocardium\textsuperscript{12, 13} or from more powerful myocardial contraction. However, the contractile mechanism does not seem to be closely tied to the electrical activity in the myocardial cell membranes.\textsuperscript{14, 15} The functional mechanisms involved in these changes in myocardial performance deserve a great deal more study.

**Ventricular Impulse as a Sign of Cardiac Malfunction**

The ventricles can develop a forceful im-

![Image](https://example.com/image.png)

**Figure 4**

Sinus arrhythmia produces modest changes in ejection velocity, ventricular pressure, and left ventricular diameter as the cycle lengths vary. A premature contraction with reduced ejection was characterized by a more gradual acceleration of ejection velocity to a lower peak associated with a slower rate of rise and fall of ventricular pressure.
pulse only when the myocardium in the walls is contracting synchronously. During normal everyday stress, which provokes sympathetic discharge to the heart, the acceleration and peak outflow rates of both ventricles are accentuated. This is a primary mechanism for autonomic control of the heart. On the contrary, a reduction in ventricular impulse has been repeatedly observed in a number of different conditions associated with subnormal ventricular function. Depression of ventricular impulse (Ft) results from several different mechanisms, as illustrated by some selected examples.

**Ventricular Premature Contractions**

The typical ventricular premature contraction follows closely after a normal beat and characteristically produces a markedly reduced ejection (fig. 4). At the onset of systole, the upward slope (acceleration) is more gradual and the peak rate of flow from the left ventricle is attained nearer mid-systole. The left ventricular pressure rises more gradually to a lower peak value. In other words, left ventricular ejection assumes some characteristics of the normal right ventricular ejection and the mechanisms may be related. A premature contraction from an ectopic focus in the ventricle at some distance from the conduction system produces an abnormal sequence of excitation. Instead of the normal rapid, almost synchronous, excitation of the endocardial surface of the left ventricle, the wave of excitation spreads over slowly conducting pathways, so that various portions of the ventricular wall contract sequentially. The contracting myocardium cannot rapidly elevate ventricular pressure if part of the ventricular wall is relaxed and bulges outward. This phenomenon is reminiscent of the normal sequential contraction of the right ventricle, illustrated in figure 1C. Thus, an essential requirement for a very rapid rise in ventricular pressure, rapid acceleration of blood, and high peak flow rates (large impulse) is a concerted

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*Figure 5*

Acute ligation of the circumflex coronary artery in a conscious dog with a previously ligated anterior descending coronary artery produces a rapid reduction in the rate of increase of outflow rate, a lower peak velocity, and a smaller stroke volume from both right and left ventricles.
and synchronous contraction of the entire ventricular wall during the initial stages of systole.

Experimental Coronary Occlusion

In a series of experiments on healthy, conscious dogs with indwelling flowmeters and pressure cannulae, the left circumflex coronary artery was abruptly occluded by tension on external ligatures. Since coronary atherosclerosis is usually diffuse, the anterior descending coronary artery had been ligated at a point near the junction of its upper and middle thirds when the gages were installed a week or two before the experiment (fig. 5). The typical response to sudden occlusion of the circumflex coronary artery was prompt reduction in the peak flow rates and stroke volume, becoming fully developed within 10 to 30 beats after occlusion. Changes in acceleration and ejection rate following coronary occlusion have been reported by several investigators. For example, Ohlsson, who used high-speed cineradiographic technics in dogs with myocardial infarctions, found that peak flow velocity was delayed (reduced acceleration), although it was not necessarily observed until some hours after the occlusion. Reduction in the ability of the myocardium to accelerate blood after infarction has been demonstrated with differential pressure measurements by Rudewald and with electrokytography by Dack et al. Altered ballistocardiographic patterns in patients with coronary occlusion have also been reported.

Two mechanisms could be ascribed roles in the reduction in ventricular impulse when coronary occlusion produces myocardial ischemia. If the blood flow to a portion of the left ventricular wall is interrupted, the affected myocardium may become inactive (noncontractile), so that "paradoxical" bulging of the ischemic region occurs during ventricular systole. The contraction of one segment of the ventricle with bulging of another would retard acceleration and lower peak outflow rates by the same mechanism as occurs during premature ventricular contraction (fig. 3) or in the normal right ventricular ejection (fig. 1). On the other hand, if coronary blood flow through the chamber walls is diminished diffusely, the contractile tension and the rate of shortening of major portions of the ventricular myocardium may be reduced by ischemia.

Exsanguination Hypotension

Systemic arterial pressure can be reduced by withdrawing sufficient quantities of blood from either veins or arteries. The filling pressure, diastolic distention, myocardial fiber length during diastole, and perfusion pressure in the coronary arteries are all diminished. The net result of these factors includes reduced stroke volume, slower acceleration of outflow, and lower peak flow rates from both ventricles. The effects of reduced ventricular impulse are illustrated in figure 6 in terms of the reductions in ejection velocity, acceleration, and the initial rate of rise of aortic pressure at the onset of systole (vide infra). The reduced ventricular impulse appears to result from a generalized reduction in the ability of the myocardium to shorten rapidly while developing contractile tension. Reduction in the perfusion pressure in the coronary arteries and diminished coronary flow is a potential etiologic mechanism that deserves full investigation under these conditions.

Effects of General Anesthesia

The anesthetic agents administered to induce analgesia, relaxation, or unconsciousness in human subjects and in animals also depress cardiovascular reflexes. Sodium pentobarbital, perhaps the most popular anesthetic agent employed in physiologic research, has profound effects on ventricular performance. The peak flow velocity, acceleration, and stroke volume of both ventricles are greatly reduced (see fig. 4 in ref. 24). Since the ventricular impulse is depressed by barbiturates, ventricular performance is undoubtedly subnormal during many physiologic experiments on anesthetized dogs.

Other anesthetic agents have drastic effects on ventricular impulse. For example, the effects of a halothane anesthetic (Fluothane) administered to a conscious, healthy dog are shown in figure 7. The aortic pressure in-
Increased transiently during induction of anesthesia, but soon became very stable at a lower mean with a diminished pulse pressure. The initial upslope in aortic pressure became more gradual. The heart rate became very constant at a slightly elevated level. The aortic flow rate demonstrated that the acceleration was more gradual, the peak rate flow was lower and delayed to about mid-systole. Stroke volume was reduced, but the computed peripheral resistance was essentially unchanged. The duration of systole remained essentially unchanged.

**Increased Right Ventricular Impulse with Increased Pulmonary Resistance**

The patterns of right ventricular ejection illustrated in figures 1 and 2 are characteristic of the healthiest dogs in the series. In three animals the wave forms of outflow from the right ventricle were similar to normal left ventricular ejection, with peak flow rates in early systole (fig. 8). All these animals had severe pleural effusions and pulmonary atelectasis. In one of them, tilting of the implanted flow section also produced obstruction, which was manifested by a pressure gradient of more than 30 mm. Hg measured as a catheter was withdrawn through the main pulmonary artery. All three of these animals had intrathoracic abnormalities leading to increased pulmonary resistance and elevated right ventricular pressure. The right vent-

*Figure 6*

Removing blood from the region of the right atrium results in a reduction in peak flow velocity, acceleration of the outflow rate, lower aortic pressure, and lower rate of change of aortic pressure, as evidenced by fast paper records obtained at the time indicated by the arrow.
Figure 7

Administration of halothane anesthesia to a healthy animal produced rather drastic transient changes in cardiovascular performance during induction. During surgical anesthesia, the aortic pressure stabilized at a somewhat lower level, the heart rate became regular at a higher level, and the aortic peak flow and stroke volume were diminished. The cardiac output and peripheral resistance remained essentially unchanged. The ejection pattern resembles normal right ventricular outflow wave form.
Left Ventricle

Standing Erect

Right Ventricle

Ejection Velocity

Figure 8

In a few dogs with pulmonary hypertension accompanying atelectasis and pleural effusion, the right ventricular ejection pattern of both ventricles tends to attain a peak value in early systole. Right ventricular ejection pattern assumed characteristics like normal left ventricular ejection in the presence of pulmonary hypertension, even during changes induced by changes in position.

tricle must have become converted into a high pressure pump, assuming some of the anatomic characteristics of the left ventricle.

The time required for the right ventricular ejection to acquire the pattern normal to the left ventricle has not been established. Okino and Spencer, however, have observed the same kind of increased right ventricular impulse in a dog with dirofilariasis. The “heart” worms were obstructing the pulmonary arteries, and the pulmonary arterial pressure was found to range about 81/34. The maximal acceleration of right ventricular outflow was much greater in this animal than in any other dog in their series.

Clinical Significance of Ventricular Impulse

Patterns of ventricular ejection that can be characterized in terms of altered initial ventricular impulse have potential predictive value in assessing performance of the heart in the experiments illustrated here. From these experiments on dogs, many different pathologic conditions may lead to diminished left ventricular impulse. The fact that similar changes in ventricular impulse can occur in diverse conditions means that they have little or no significance as a diagnostic sign. If, on the other hand, reduced work capacity or impaired left ventricular competence can be evaluated in terms of the signs of reduced ventricular impulse, these signs could have great clinical value in estimating cardiac status.

Observations on dogs cannot be safely extrapolated to human subjects or patients, nor can direct-recording flowmeters (e.g., electromagnetic or ultrasonic) routinely be employed in conscious humans for direct indication of altered ventricular impulse in terms of continuous outflow rates. On the other hand, changes in ventricular impulse may be reflected in numerous other recordable variables. For example, the rate of change of ventricular pressure is diminished when peak outflow rates are slowed (e.g., ventricular premature contraction as in fig. 3). Gleason and Braunwald have reported that the first derivative of the ventricular pressure pulse
(rate of change of pressure) in man is altered by exercise, isopropylterenol or nor-
epinephrine just as in the dog (fig. 3).

Using electromagnetic flowmeters, Spencer and his co-workers have demonstrated a greatly reduced acceleration and peak flow velocity during experimentally induced constriction of the aorta simulating coarctation of the aorta. In this case, the retarded acceleration and low outflow rate did not result from a reduced left ventricular impulse but from an obstruction intervening between the heart and the flowmeter. This type of observation indicates the hazard of employing a measure of ventricular impulse as a diagnostic test. Such conditions must also be taken into account in employing signs of ventricular impulse to evaluate the status of the ventricles.

**Indirect Methods for Detecting Altered Ventricular Impulse**

A number of technics developed to evaluate the dynamic function of the heart have failed to achieve anything like universal acceptance. Analysis of the arterial pressure pulses, ballistocardiography, electrokymography, and analysis of precordial movements have demonstrated relationships with known types of cardiac disease. In each instance empirical relationships have been established by relating observed alterations in wave form to specific types of cardiac disease at postmortem examination. Although empirical relationships may have demonstrable utility, a more logical and scientific approach to recognition and assessment of disease can be achieved through greater understanding of the fundamental processes and by direct objective measurements. The number of measurements that can now be made on human patients is so limited that every effort should be made to determine by careful validation experiments the areas in which each is useful.

Since normal autonomic control is manifest in part through a very large increase in initial ventricular impulse (fig. 3), and since numerous abnormal states tend to depress ventricular impulse, clinical tests that are consistent indicators of the acceleration and peak flow rates in early ventricular systole could be most valuable in assessing the state of the ventricular myocardium. Since velocity, acceleration, and rate of change of pressure are all time derivatives, they can be indicated by slopes.

The initial abrupt upslope in the first portion of the aortic pressure pulse occurs during the period of most rapid acceleration of blood and could conceivably contain valuable information regarding the ventricular impulse. If the wave form of the arterial pulse is not too greatly deformed in its passage to the carotid arteries, a pulse wave recorded from within the carotid artery, or even by an external recording capsule, may have an initial slope that could be correlated with simultaneously recorded direct measures of ventricular impulse (fig. 9). If the initial arterial pressure upslope can be established as a valid indicator of the rate of pressure rise and the rate of ejection into the aorta, a simple recording pressure capsule with a differentiating circuit may have value as a tool ancillary to electrocardiograms in cardiology laboratories. It would not be difficult to add an indirect measure of isovolumic contraction time\(^{26}\) or ejection time\(^{27}\) to the procedure, and both have value in some circumstances.

Electrokymography can be applied to most patients suspected of having heart disease in the course of routine fluoroscopic examination.\(^ {28}\) When the photocell is positioned over an appropriate border of the heart, the changes in light intensity reflect the movements of that wall. To the extent that the electrokymographic record corresponds to actual changes in ventricular dimensions,\(^ {29}\) the early systolic downslope is a sign of the rate of ejection from the ventricle. An evaluation of the downslope by means of differentiation to indicate the rate of change of dimension seems worthy of critical examination as a sign of changing ventricular impulse.

If the rate of change of dimensions is confirmed as a reliable sign of ventricular impulse, the technic of ultrasonic echo-ranging\(^ {30-32}\) may then be applied as a much simpler means of obtaining information regarding the rate of displacement of ventricular walls. Since
An experimental design is schematically indicated for the purpose of determining the extent to which certain indirect clinical measures (arterial pulse waves, electrokyromograms, precordial displacement records, and ballistocardiograms) might be employed to assess changes in initial ventricular impulse as an indicator of ventricular competence or status in human subjects or patients. By means of a double-lumen catheter carefully positioned at two sites in the aorta a large number of the characteristics of left ventricular ejection can be derived as indicated on the left. Indirect methods could be evaluated in terms of changes in initial ventricular impulse by cross correlations between these critical variables and the initial deflections on wave forms derived by the indirect methods indicated in the column on the right.

the output from an echo-ranging device can easily be developed as an analog voltage, differentiation of the signal by electronic analogs is very simple.

The initial movements of the heart at the onset of systole have been recorded by transducers applied to the precordium (i.e., kinetocardiograms, apex cardiograms, etc.). As the coupling of the heart to the thoracic wall is very imperfect, the recorded deflections cannot be expected to be any consistent sample of the energy expended during the initial ventricular impulse. Time relations between the deflections recorded at various sites over the precordium have been reported, but the variability of the wave forms suggests that the technic will have limited value for definitive studies of ventricular function. There would be some merit, however, in directly comparing simultaneous recordings of direct indicators of initial ventricular impulse and precordial displacement recordings. The amplitudes and slopes of the wave forms recorded during initial systolic ejection could then be subjected to a comprehensive statistical analysis. This appears to be a logical approach.
to determining the reliability of precordial displacements as indicators of ventricular dynamics.

Ballistocardiography has evoked waves of interest and popularity, but has never achieved generalized clinical application. Talbot emphasized the fact that the approach to ballistocardiography has been largely "exploratory and correlational" without an understanding of the detailed origins of the record. The ballistic forces accompanying initial ventricular ejection are undoubtedly dampened and distorted by the spring-like suspension of the heart within the thorax and by the elastic properties of the supporting tissues between the body and the recording platform. Theoretically, the ballistocardiograph might achieve a much more useful position in cardiology as a tool to indicate changes in initial ventricular impulse. A statistical analysis of the relations between the direct indicators of initial ventricular impulse and simultaneous ballistocardiographic wave forms from devices sensing displacement, velocity, and acceleration (fig. 9) might disclose signs of prognostic value even if the ballistocardiograph proves a slender reed as a diagnostic tool.

Proposed Experiment for Validation of Ventricular Impulse as a Clinical Test of Cardiac Status

Detection of changes in left ventricular impulse in human subjects requires continuous registration of the acceleration of the blood, peak outflow rates, and ventricular and aortic pressure, as in figure 2. A large number of potentially significant variables referable to properties of ventricular ejection can be recorded in human subjects by means of double-lumen catheters placed in two positions (fig. 9). When the distal opening is in the left ventricle, the left ventricular pressure and its rate of change can be recorded. The proximal lumen provides an opportunity to record the initial rise and rate of change of aortic pressure. The difference between ventricular and aortic pressure could be registered just as was described by Spencer and Greiss (fig. 2). Withdrawal of the catheter until its distal tip is just above the aortic valve would permit registration of the pressure difference between points along the aorta. Techniques for deriving instantaneous flow velocities by analog computer analysis of this pressure difference have been reported by Fry, by Rudewald, and by Barnett et al. The wave forms derived from this analysis of pressure difference per unit of distance along the aorta (dP/dx) are very similar to those recorded by direct application of flowmeters to aortas in patients at surgery or from chronically implanted flowmeters in dogs. Both the acceleration of blood and the peak flow velocity can be indicated by this method. If these measurements were made on human subjects during spontaneous changes in ventricular impulse, or those induced by administration of pharmacologic agents, while recordings were obtained by electrokymography, ballistocardiography, apex cardiography, and carotid pulse wave forms, cross correlations and factor analysis would provide definitive information concerning the validity of the indirect recording techniques as indicators of the changes in ventricular impulse (fig. 9). Those indirect measures proved valid in such an experiment could then be employed as indices of ventricular performance in patients with heart diseases to establish its prognostic value on a scientific basis.

Summary

During the early stages of ventricular systole, myocardial tension develops very rapidly, ventricular pressure rises steeply to exceed arterial pressure, and momentum is then rapidly imparted to the blood flowing out of the ventricles. The acceleration of blood out of the ventricles is an expression of the force being applied by the contracting myocardium, and the peak flow velocity can be regarded as the product of the net force applied over the time from the onset of ejection to the peak. The product of force and time is designated by a well-established physical term: impulse (I = Ft). Thus, "initial ventricular impulse" appears to be an appropriately descriptive term for the dynamic properties of ventricular ejection. Direct and continuous records of acceleration, the outflow rate of blood, and the rates of change of ventricular and arterial
pressure demonstrate that ejection of blood from both ventricles (particularly the left) is more like striking a piston with a mallet than like squeezing blood out of a chamber. These dynamic characteristics of ventricular ejection are greatly altered by autonomic control and by simulated disease conditions.

Initial ventricular impulse is greatly augmented during exercise, by stimulation of sympathetic nerves to the heart, and following long diastolic intervals. On the contrary, initial ventricular impulse is markedly depressed during premature ventricular contractions, acute coronary occlusion, exsanguination hypotension, general anesthesia, etc. Thus, signs of changing ventricular impulse may have significant value in assessing the performance capability of the heart. For this purpose, certain clinical tests of cardiac performance (e.g., arterial pressure pulses, electrokymograms, precordial displacements, ballistocardiograms) may have unsuspected importance. Experiments on animals have considerable value in suggesting measurements that might be appropriately employed on human subjects and patients, but because of species differences, the validation of initial ventricular impulse as clinical test of value must ultimately be accomplished by appropriate measurements conducted on human subjects. For this reason, a specific experimental design is suggested for the evaluation of certain indirect indicators that may have potential value in routine assessment of the cardiac status. By means of modern high-speed computers, cross correlations, and factor analysis could bring to light the aspects of peripheral arterial pulse waves, electrokymograms, and ballistocardiograms of reliable predictive value in assessing initial ventricular impulse in man.

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