Evaluation of Operative Left Ventricular Outflow Tract Lesions with a Fluid Impedance Plot

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SUMMARY
A hydraulic fluid impedance plot is produced by plotting instantaneous differential pressure against phasic blood flow across a vascular segment. This simple plot quantitatively and compactly describes the changes in flow resistance (impedance) throughout the entire cardiac cycle. Impedance plots were obtained before and after corrective surgery in patients with a variety of left ventricular outflow tract and aortic valve lesions. Although present techniques are still limited, impedance plots now provide quantitative measurement of flow resistance throughout the cardiac cycle in a variety of cardiac and great vessel lesions.

Additional Indexing Words: Aortic valve lesions, Flow resistance, Differential pressure, Phasic blood flow, Pulsatile pressure, Valvular regurgitation

PRECISE EVALUATION of the hydraulic resistance of vascular channels carrying pulsatile flow requires simultaneous measurement of the pulsatile pressure difference and phasic blood flow. Since the electromagnetic blood flowmeter senses both directional and phasic changes in blood flow, these data, when plotted against simultaneous differential pressure measurements across a vascular lesion, permit quantitative measurement of flow resistance throughout the entire cardiac cycle.

Ohm's law, applied to hemodynamics, relates resistance, flow, and differential pressure across a vascular segment. A more general expression for flow resistance or impedance may be written: \( Z = \frac{\Delta P}{F} \), where \( Z \) is the instantaneous impedance. \( \Delta P \) is the instantaneous pressure difference \( (P_1 - P_2) \), and \( F \) is the instantaneous flow through the vascular segment. If flow and differential pressure are continuously plotted by an X-Y recorder, impedance, the sum of all resistive and reactive forces impeding blood flow through a given channel, is continuously displayed throughout the cardiac cycle. In the present study, the capability of this display to more precisely measure hydraulic resistance has been used to evaluate lesions of the aortic valve and left ventricular outflow tract. The impedance plots of these lesions are presented with interpretations, and the advantages, disadvantages, and limitations of the method are discussed.

**Methods**
Blood flow was measured with an appropriately fitted square-wave electromagnetic flowmeter.

*The instantaneous impedance summed for all points in time may be termed "mean impedance" and may be analogous to the "characteristic impedance" of electrical terminology.

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differential pressure was measured via individual pressures by connecting the output of one half of each strain gauge bridge to one side of a third d-c amplifier which was connected through a third driver amplifier to the recorder. Balance of the differential pressure channel was obtained by increasing the amplifier gain above the expected range and trimming the excitation voltage for one or both individual pressure gauges until simultaneously increased static pressures produced minimal deflection on the differential pressure channel. Dynamic balance was checked by rapidly and equally changing the pressure on both strain gauges simultaneously.

Zero reference for gauges was obtained by opening gauge stopcocks to atmospheric pressure. Zero reference for the needle tips was not taken because positional changes in placement of both needles caused gravitational differences in the differential pressure measurements. Differential pressure measurements were accurate to less than 0.5 mm Hg.

The strip chart recorder displayed the phasic blood flow, proximal and distal pressures, and differential pressure (fig. 1). All parameters were also recorded on a magnetic instrumentation recorder†† for playback and subsequent analysis. The frequency response of the flowmeter extended to approximately 50 Hz while the pressure system was limited to approximately 10 Hz.

In the operating room, impedance plots were displayed on an X-Y cathode ray recorder‡‡ and photographed§§ for immediate analysis. Time relationships during the cardiac cycle were represented by the density or faintness of the tracings. During playback, time relationships were represented by the spacing between Z axis modulations of 5 msec with the direction of movement of the trace indicated by the arrow points.

††Ampex Corporation, Redwood City, California.
‡‡Tektronix, Inc., Beaverton, Oregon.
§§Polaroid camera available through Tektronix, Inc., Beaverton, Oregon.

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*Carolina Medical Electronics, Inc., Winston-Salem, North Carolina.
†Beckman Instruments, Inc., Offner Division, Schiller Park, Illinois.
‡Statham Instruments, Hato Rey, Puerto Rico.
§Endevco Corporation, Pasadena, California.
**Dana Laboratories, Inc., Irvine, California.

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A full interpretation of the impedance plot requires knowledge of three passive impedances referred to here as resistance (R), compliance (C), and iner- tance (L). Compliance, for practical purposes, expresses the stiffness of the involved vessel wall and valve, and iner- tance is related to the blood mass in the segment. Each of these parameters may influence the impedance plot tracing, although resistance is by far the major practical consideration at the time of surgery.

Over a 3-yr period, selected patients were studied at the time of operation, both before and after intracardiac surgery was performed. Prior to patient studies, left ventricular outflow impedances of normal sheep and dogs and those with experimentally produced aortic valve lesions were measured. Data from six patients and one animal were selected to illustrate the value and interpretation of the impedance plot.

Results

Normal Aortic Valve

Figure 1 presents the basic parameters of aortic blood flow, left ventricular and aortic pressures, and differential pressures from one patient after closure of a small ventricular septal defect. In figure 2, the impedance plot of the differential pressure versus flow for the data presented in figure 1 is reproduced. Most of the information in figure 1 is con- densed into the impedance plot in a more usable and easily understood form. To facilitate description of the changing events during the cardiac cycle, the impedance plot has been arbitrarily divided into six phases. These are illustrated in figure 3.

Phase 1 denotes the beginning of systole when “isovolumic” ventricular contraction occurs. The exact beginning of phase 1 is often obscured by the tracing made during mid and late diastole, but may be indicated by the sudden increase in velocity of ΔP as it moves toward the Y axis. Phase 2 begins when left ventricular pressure exceeds aortic pressure. Early in phase 2 the impedance plot describes an “acceleration transient” which is due to the acceleration of the static mass of blood.7,8 During the remainder of phase 2, blood flow increases rapidly and differential pressure approaches zero. Phase 3 begins at peak forward blood flow and ends when the tracing crosses X zero on the Y axis. Forward blood flow decreases during phase 3, but again, differential pressure is very small.

Phase 4 appears in the second quadrant and denotes the period between X zero and

![Figure 2](image-url)

*Figure 2*

Impedance plot of same data presented in figure 1. Flow is plotted on Y axis and differential pressure is plotted on X axis. Plus sign on Y axis indicates forward blood flow. Plus sign on X axis indicates that left ventricular pressure exceeds the simultaneously recorded aortic pressure. Arrows are spaced 5 msec apart and indicate the direction of the impedance plot sweep.

![Figure 3](image-url)

*Figure 3*

Impedance plot during one cardiac cycle divided into six arbitrary phases to facilitate description (see text).
Y zero during late systole and early diastole. During phase 4, blood flow is forward, but is decelerating, and aortic pressure exceeds left ventricular pressure. With a normal valve and outflow tract, this negative differential pressure gradient is small. We believe this second quadrant tracing may be due to initial relaxation of the ventricle at the time inertia of the blood mass continues forward flow.

Phase 5 is one of negative or backward blood flow which causes the valve to close, and then to descend slightly or seat (seating transient). Phase 5 ends at the beginning of isometric relaxation which is a poorly delineated point occurring after valve seating and after negative blood flow returns toward Y zero. Due to coronary artery filling, the flowmeter on the ascending aorta often senses a slight negative flow throughout phase 6. Phase 6 represents diastole as can be seen by the maximum negative differential pressure near Y zero, as the ventricle begins to contract (phase 1).

Aortic Regurgitation

Experimental aortic valve incompetence without stenosis was produced in anesthetized sheep and dogs by puncture with a probe passed via the common carotid artery. Blood flow and pressures were measured with the chest open as described under “Methods.” Figure 4 discloses the impedance plot taken before and after puncture of the aortic valve. Although calibrations were not available, the ΔP and F scales were identical before and after producing the lesion.

Regurgitation is manifest by the depression of phases 6 and 1 into the third quadrant because of reversed flow during diastole. Other characteristics of aortic regurgitation apparent on the plot are the decreased aortic-left ventricular pressure difference during diastole, increased systolic flow apparent from the higher peak flow, longer duration of forward blood flow, and the more prolonged acceleration transient. In addition, the positive slope during phase 1 is a unique feature of the regurgitant valve which is not apparent from classical pressure and flow tracings.

Since the elasticity of the valve was little affected, the valve closing transient of phase 5 proceeds to the same depth as it did in the control. However, because of regurgitation, blood flow does not approach the 0 flow base line during phase 6.

Supravalvular Aortic Stenosis with Normal Aortic Valve

A 12-year-old asymptomatic female with mental retardation, partial deafness, and strabismus was found to have a peak systolic gradient across her aortic outflow tract of 100 mm Hg at cardiac catheterization. Angiograms demonstrated supravalvular aortic stenosis which was seen at operation to narrow the aortic lumen to approximately one fifth of its cross sectional area, 1.5 cm above the aortic annulus. An elliptical pericardial gusset, 2.5 by 3.5 cm, was placed across the stenotic area. The aortic valve and outflow tract were otherwise normal. Impedance plots made before and after repair are presented in figure 5.

Phases 2 and 3 of the pre-repair impedance plot demonstrate a marked increase in resistance during accelerating blood flow. During decelerating forward blood flow (phase 3), instantaneous resistance was less than that observed during the acceleration phase. The
Supravalvular aortic stenosis with normal aortic valve. Pre-repair loop shows marked hysteresis, a significant increase in mean systolic and peak flow impedance and a prominent second quadrant component (phase 4). The valve seating transient is normal. After repair a small pressure gradient remains, but the loop shows reduced hysteresis and a normal second quadrant component.

Impedance loop has marked hysteresis which may be due, in part, to technical factors of current methodology (see “Discussion”) but also may be due to increased momentum of the high blood velocity in the presence of significant, fixed orifice resistance. With repair, some residual stenosis remained (peak differential pressure of 30 mm Hg), but the marked delay in reversing forward blood flow (phase 4) disappeared.

Since the impedance plot correlates instantaneous flow and differential pressure, instantaneous impedance may be measured at each point. Instantaneous impedance varies as the cotangent of the angle ($\theta$) between the X axis and a vector drawn from the point of origin (zero flow and zero pressure differential) is estimated as point on the Y axis where impedance loop crosses X zero during early systole. A vector drawn through this point to the point of peak forward flow makes an acute angle, $\theta$, with the X axis. The cotangent $\theta$ equals $\Delta P/F$ or $Z$, the instantaneous impedance at peak flow. The factor by which peak flow impedance (a useful quick measurement) is altered by surgery is obtained by dividing cot $\theta$ before repair by cot $\theta$ after repair ($\cot 40^\circ = 1.1918$; $\cot 73^\circ = 0.3057$; $1.1918 / 0.3057 = 3.9$).

Point of origin, for practical purposes, may be estimated as the point between phase 1 and phase 2 when differential pressure is zero. Forward aortic flow has not begun and coronary artery flow has practically ceased when systolic left ventricular pressure equals aortic pressure. In all impedance plots when a zero flow reference was established, correlation between this estimated point of zero blood flow and true zero blood flow was excellent.

Since the gain of the flowmeter recorder was not changed during operation, peak resistance fell in proportion to the change in the cotangent of the angle theta ($\theta$; fig. 6). In the example illustrated in figure 6, before repair, $\theta$ was 40° with a cotangent of 1.1918 and after repair, $\theta$ was 73° with a cotangent of 0.3057. Peak systolic resistance therefore decreased by a factor of 3.9. The flat phase 1 and the good valve seating transient (phase 5) indicated a competent, flexible aortic valve.

Supravalvular Aortic Stenosis with Bicuspid Aortic Valve

A 12-year-old boy with an aortic systolic murmur fainted once after extreme exertion.
Supravalvular stenosis with bicuspid aortic valve. Pre-repair loop shows marked hysteresis, increased systolic impedance, and prominent second quadrant component. The valve seating transient is abnormally small. After repair of supravalvular stenosis, the pressure gradient was reduced to 25 mm Hg, but peak forward flow was also reduced. Instantaneous impedance at peak flow was reduced by a factor of only 2.8. Note prominent second quadrant component (phase 4) and absent valve seating transient in the post-repair plot.

A peak systolic gradient of 95 mm Hg was measured across the aortic outflow tract at cardiac catheterization. At surgery he was found to have a bicuspid aortic valve with both annular and marked supravalvular stenosis. A pericardial gusset, 3.5 by 1.5 cm, was placed in the wall of the aorta across the supravalvular stenotic area and down to the annular stenosis. The bicuspid aortic valve was not disturbed.

The impedance plots and cotangents of θ taken during systole in figure 7 indicate that resistance between the left ventricle and distal part of the ascending aorta decreased by a factor of 2.8. The presence of a bicuspid valve and annular stenosis probably explains the remaining outflow tract resistance. The valve seating transient was reduced both before and after outflow tract repair, and is probably related to the bicuspid valve which was competent but stiff. A somewhat greater than normal negative gradient during phase 4 was present both before and after repair.

Calcific Aortic Stenosis Treated by a Cutter-Smeloff Prosthesis

A 53-year-old symptomatic male had pure aortic stenosis with poststenotic aortic dilatation and a peak systolic gradient of 95 mm Hg. A bicuspid, heavily calcified valve was replaced with a no. 5 Cutter-Smeloff prosthesis (Teflon ball, diameter 16.7 mm and
Aortic stenosis and regurgitation: Starr-Edwards prosthesis, paravalvular leak. Pre-replacement impedance plot shows both fixed orifice stenosis and regurgitation. The first quadrant loop has less hysteresis than the loops of valves with pure fixed orifice stenosis and the second quadrant component is small. Loop remains well below Y zero during diastole and early systole (phases 6 and 1) indicating regurgitation. After replacement of the valve with a Starr-Edwards prosthesis (postrepair), the gyrations during phase 2 probably indicate turbulence. The depressed loop during phases 6 and 1 clearly indicates regurgitation, possibly due to poor valve seating, but proved at reoperation to be due to paravalvular leak.

Idiopathic hypertrophic subaortic stenosis. The clockwise first quadrant impedance loop before aortotomy indicates dynamic resistance which increases during mid and late systole. Cotangent \( \theta \) at peak \( \Delta P \) is 11 times cotangent \( \theta \) at peak forward flow. As the muscular resistance relaxes, \( \Delta P \) falls rapidly and after crossing X zero, the remainder of the impedance plot is normal. After incision of the hypertrophic mass, a small initial pressure gradient (phase 2) remained as an exaggerated acceleration transient. Small clockwise loop at peak flow indicates slight residual muscular obstruction. Although the entire plot appears shifted slightly to the right of X zero, no discrepancy in X zero was found during playback tape analysis.

weight 3.04 g). The impedance plots are reproduced in figure 8.

Before replacement, the impedance loop showed characteristic hysteresis and marked outflow resistance. No valve seating transient or aortic regurgitation was recognized and again a prominent phase 4 was present. After replacement, outflow resistance during accelerating and peak blood flow was considerably reduced. During the deceleration phase, left ventricular pressure was reduced below aortic pressure, making a prominent phase 4, second quadrant tracing. Whether this portion of the impedance loop is characteristic of this prosthesis, is related to the ball mass, is due to slight impaction of the ball on the outflow cage prongs, or is an individual finding cannot be concluded with certainty from this case. A ball seating transient occurred in phase 5.

Aortic Stenosis and Regurgitation Treated by a Starr-Edwards Prosthesis: Paravalvular Regurgitation

A 52-year-old symptomatic male was found
to have aortic valvular stenosis and regurgitation by cardiac catheterization and angiography. At surgery, a tricuspid, fibrotic, stenotic, and incompetent aortic valve was excised and replaced with a Starr-Edwards ball-valve prosthesis (model 9A). Paravalvular aortic regurgitation was noted after placement of the prosthesis, but could not be corrected (fig. 9). Ten months later, the original prosthesis was replaced, but differential pressure and flow data were not obtained.

The impedance plot before repair (fig. 9) demonstrated aortic stenosis and regurgitation. The second quadrant trace (phase 4) was small, but negative flow occurred throughout diastole. A valve seating transient was not well delineated. The forward to reverse resistance ratio was 1.5:7.5, indicating very poor overall valve function. After valve replacement, the prosthesis appeared to offer resistance to accelerating forward blood flow. The erratic changes in differential pressure in phase 2 probably represent turbulence from ball vibration within the cage. This has been a singular finding in our experience with Starr-Edwards prostheses, as other valves have performed without this phenomenon. The paravalvular aortic regurgitation was apparent during phases 1 and 6 since negative blood flow did not approach Y axis zero. Overall improvement in outflow resistance represented by a change in cotangent $\theta$ was 5.2 times and the forward to reverse valve resistance ratio improved to 0.3:6.

**Idiopathic Hypertrophic Subaortic Stenosis**

A 34-year-old woman described in detail previously was found to have a peak systolic gradient of 85 mm Hg across the aortic outflow tract and an abnormal interventricular septal mass. Impedance plots made before and after incision are presented in figure 10.

During systole, the impedance plot displayed a clockwise loop in the first quadrant. Some increased resistance was present throughout systole. However, between peak forward flow and peak differential pressure, outflow tract resistance increased by a factor of 11.0 ($\cot 20^\circ / \cot 70^\circ$). Following incision, a small amount of residual dynamic systolic resistance remained. Normal valve compliance was indicated by a deep phase 5. A reduction in the reverse gradient of phase 4 followed removal of the stenosing outflow tract hypertrophy.

**Discussion**

This report presents a new technique for quantitative measurement of the impedance of operative cardiovascular lesions, particularly left ventricular outflow tract lesions.

Impedance plots combine all of the dynamic changes in flow, differential pressure, and resistance during the cardiac cycle into a single record. These plots provide a quantitative measure of impedance or flow resistance, and thus permit quantitative evaluation of the hemodynamic resistance of cardiovascular lesions before and after surgical repair. The presence of valve regurgitation does not interfere with precise measurement of outflow tract impedance. This objective hemodynamic evaluation is applicable to a variety of surgical procedures, particularly those in which the surgical repair does not restore normal anatomy. Measurement of pressure gradient alone may be misleading since blood flow after repair may differ considerably from pre-repair flow. Pressure difference and flow may decrease proportionally without a change in flow resistance.

The advantages of the impedance plot over classical tracings of blood flow and blood pressure are as follows: (1) The most essential information regarding hemodynamic impedance before and after surgery is condensed into a single graph for objective evaluation and clinical report. (2) The data are immediately available to the surgeon and may influence operative decisions regarding the adequacy of the surgical procedure. Tape recording permits later analysis and opportunities to improve signal to noise ratio. (3) The technique quantitates forward and reverse impedance of a valve and both valve and wall compliance and mass acceleration phenomena. (4) Estimation of a zero flow...
reference is often possible under highly pulsatile conditions of blood flow across a
valve without the necessity of occluding the
blood vessel or stopping the heart. (5) The
transient loops of the reactive elements of
the vascular segment are preserved without
impairing immediate measurement of peak or
average flow resistance.

A few disadvantages of present techniques of
measuring impedance remain: (1) The
present system is limited by phase errors due
to placement of the flowmeter slightly away
from the site of the lesion and transmission
of pressure changes through needles and
catheters to the transducers. (2) The method
is applicable only during surgery and ex-
tends operative time by approximately 10 min.
However, no increase in the duration of
cardiopulmonary bypass is required. (3) Pre-
sent equipment and techniques are not applic-
able to the study of atrioventricular valves,
but are applicable to right ventricular out-
flow tract lesions if the flowmeter can be
satisfactorily placed on the main pulmonary
artery.

The future development of an impedance
catheter, which simultaneously measures ΔP
and F, will allow comparison of serial mea-
surements taken at intervals during the entire
period of medical and surgical management
of cardiac patients. Many of the disadvan-
tages and limitations of present techniques
will be overcome by the development of this
catheter. Future incorporation of sensors into
cardiovascular prostheses will permit pre-
cision monitoring of the mechanical perform-
ance of these devices under all conditions
of activity.

The reverse gradient across the aortic out-
flow tract during phase 4 is greatly increased
in the presence of stenosis. This increase
and the enlarged hysteresis loop in phases 2 and
3 may be caused by increased momentum of the blood due to increased velocity through
the stenosed area.

The impedance diagram of the case of
idiopathic hypertrophic subaortic stenosis
(IHSS) shows that the major resistance to
forward flow reaches maximum. This resis-
tance during mid and late systole causes the
characteristic diphasic flow tracing which is seen in patients with IHSS. The
clockwise loop of the impedance diagram in
the first quadrant indicates that resistance
increases during systole, even after forward
flow begins to decrease. Thus, the impedance
diagram in the first quadrant indicates that
resistance increases during systole, even after forward flow begins to decrease. Therefore,
the impedance diagram clearly indicates that
dynamic muscular resistance in IHSS is max-
imal in mid or late systole, as opposed to the
resistance of fixed orifice lesions.

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... that within very wide limits the output of the heart is independent of the resistance to the output. Thus the heart possesses in a high degree the power of adaptation to changed conditions, which is the essential characteristic of living organisms; and we have now to see how far we can refer this power to the physiological mechanisms with which the heart is endowed.

... Excitation of certain of the cardiac branches of the sympathetic causes a constant increase in the strength of each ventricular beat, and therefore raises the output at each beat. This augmentation of the ventricular contraction may or may not be accompanied by acceleration.

... None of these [reflex] mechanisms, therefore, seem to be directed for the purpose of maintaining that cardiac adaptation which is the necessary condition of compensation in heart disease; and this conclusion is confirmed by the fact that we may divide all the nerves, afferent or efferent, which supply the heart without diminishing to the slightest extent its power of compensation.

... [In experimental induced aortic stenosis] this increased diastolic distension exercises a strong augmenting effect on the ventricular contraction, so that not only the power but also the extent of the contraction is increased above normal, and this increase might be sufficient to empty the ventricle to its usual degree even against the higher resistance offered by the stenosis.

... Owing to the arrangement of the muscular fibres of the heart there is a strong circular band of fibres round each of the auriculo-ventricular orifices. This ring contracts with the rest of the ventricular muscle, thus acting as a sphincter and practically closing these orifices. It is not until fatigue of the cardiac muscle sets in, or the orifices are held permanently open by development of hard connective tissue around them, that mitral or tricuspid regurgitation can occur to any considerable extent. ... If the strain on the cardiac muscles becomes too great or too prolonged, or the reserve power of the muscle itself is diminished in consequence of malnutrition, we get failure of compensation and the appearance of all those symptoms characteristic of defective working of the heart pump. There is no doubt that cardiac muscle, just like skeletal muscle, is susceptible of fatigue, which, as in the latter, may be caused either by too great a strain on it during its contraction or by too great a stretching or distension between the contraction. ... A constant recurrence of such condition of overstrain must lead to a steady deterioration of the contractile powers of the heart and the production of the chronic results of the failure of compensation. ... —ERNEST H. STARLING: Some Points in the Pathology of Heart Disease. Lancet 1: 569, 1897.
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