ARTERIAL COMPLIANCE IN HYPERTENSION

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Study of the viscoelastic properties of the arterial tree in man is difficult. Most previous reports on arterial compliance (distensibility) in man, in which compliance has been estimated directly from pressure-volume relationships, have involved arteries taken from cadavers, (1,2). In vivo studies in humans have usually utilized indirect estimates of compliance (3-8), such as arterial pulse wave velocity (5,8).

Arterial compliance has been a somewhat neglected subject in recent cardiovascular research; this probably reflects methodological difficulties rather than lack of interest (3-7) and does not imply that aberrations in arterial distensibility play no important role in human circulatory pathophysiology and disease. Loss of arterial compliance is seen as part of the process of arterial aging (1-4,6,9-11). In vitro studies have documented a diminution in compliance in arterial hypertension in man (1,2). Reduced arterial compliance in man has been related to systolic hypertension in the elderly (3,11) and to abnormal baroreflex function in hypertensive subjects (5,12).

A method for studying arterial compliance in vivo in man would be of considerable value. We have developed a technique for estimating arterial compliance from the intra-arterial pressure pulse contour and calculated total peripheral vasomotor resistance (13). The method is based on the relationship of the change in arterial volume (ΔV) during a selected interval of the diastolic runoff period, to the fall in pressure (ΔP) that this change in volume produces. Since this method is a direct expression of pressure-volume relationships, it carries potential advantages over other indirect estimates of arterial compliance, such as those based on

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pulse wave velocity measurements (5,8).

METHODS AND MATERIALS

Patient Groups:

Arterial compliance was estimated in 27 subjects undergoing diagnostic cardiac catheterization who had no evidence of aortic valvular disease or of intra- or extracardiac shunts. Three groups were defined: Group I, eight normotensive patients (intra-arterial blood pressure less than 140 mm Hg systolic and 90 mm Hg diastolic) (5 men (m) /3 women (w)) under the age of 40; Group II, 13 normotensive patients (9 m/4 w) over the age of 40; Group III, six hypertensive patients (5 m/1 w) under the age of 40. All hypertensive patients met at least two of the three following criteria: brachial intraarterial blood pressure $\geq$ 140 mm Hg systolic, $>$ 90 mm Hg diastolic, or $\geq$ 100 mm Hg mean.

Catheterization Procedure and Measurement of Haemodynamic Variables:

None of the hypertensives had received sympatholytical drugs. A number 6 or 7F Eppendorf [R]catheter was passed to the pulmonary artery through a venotomy. A polyethylene catheter was placed in the brachial artery and the dead space of the sampling system and the rate of withdrawal was taken into account. Indocyamine green, 2.5 mg was injected rapidly into the pulmonary artery, employing a saline flush. Three calibration points were used for the densitometer and cardiac output was calculated by the method of Hamilton et al (14).

A number 6,7 or 8F Eppendorf [R] catheter was then placed in the brachial artery and advanced to the ascending aorta where pressure-pulse contour was again recorded at 200 mm/sec. on a calibrated E for M recorder. The estimation of arterial compliance was based on measurements from 2-6 arterial pressure-pulse contours at each site.

Estimation of Arterial Compliance Index:

Windkessel theory views the (central) arterial system as an elastic chamber with a linear pressure-volume relationship between end-systole and end-diastole (7,15). However, arterial compliance which is measured as the change in volume that accompanies a unit change in pressure ($\Delta V / \Delta P$) is nonlinear (1,2) and describes the physical properties (viscoelastic) of the walls of the large and medium arteries. The present approach to measurement of systemic arterial compliance depends on the simultaneous estimation of change in arterial volume and arterial pressure during diastolic runoff.

For the estimation of change in arterial volume (ΔV), that is flow out of the arterial system into the capillary bed, of necessity an indirect method is used. The estimation of ΔV is based on the relationship between the instantaneous rate of change in arterial volume, dv/dt, during diastolic runoff, and the instantaneous arterial pressure (P). If two simplifying assumptions are made, first of a linear relation of flow to pressure at pressures above approximately 20 mm Hg (16, 17) and second that the pressures used during diastolic runoff are the same throughout the central arterial system, the change in arterial volume for the period t_1-t_2 (Fig. 1), may be calculated. This is accomplished by integrating to find the area under a designated portion of the arterial pressure pulse curve in diastole (eliminating the area of assumed physiological nonlinearity for the relationship of flow to pressure, that is below 20 mm Hg), and dividing by the resistance to flow from the central arterial system (total vasomotor resistance). The portion of the diastolic pressure pulse contour used, from t_1 (0.06-0.08 sec after the peak of the dicrotic wave) to t_2 (0.20-0.24 sec after t_1) was chosen to avoid aberrations known to occur in the brachial pressure pulse contour, when compared with the aortic pressure pulse contour during systole and the dicrotic wave (18). The corresponding change in pressure is measured directly from the pressure pulse contour.

Change in volume (ΔV) is adjusted for body surface area (BSA) to correct for differences in body size, and ACI, the mean arterial compliance index ($\Delta V/BSA/\Delta P$) calculated. (See Appendix).

RESULTS

The general characteristics and haemodynamic measurements of the 27 patients studied are shown in Table 1.

Arterial compliance index decreased with age. There was a significant inverse correlation between age and ACI in normotensive subjects; $r = -0.496$, $p<0.05$ (Fig. 2). There was also an inverse relationship between arterial compliance index and blood pressure (Fig. 3). Further, in an individual subject, ACI was dependent upon instantaneous blood pressure. With spontaneous changes in blood pressure, ACI varied in a systematic, inverse fashion with blood pressure (Fig. 3). Consequently, the ease with which the systemic arterial system could be distended (ACI) increased as blood pressure fell.

The relationship of ACI to blood pressure and to age was further evaluated by comparing ACI in two patient groups, matched in one instance for age, and in the other for blood pressure. ACI was compared in the normotensive groups I and II, matched for blood pressure (mean blood pressure 81 ± 5 mm and 82 ± 8 mm) but with a significant difference in age (32.0 ± 6.4 yrs. and 53.4 ± 8.8 yrs, $p<0.01$)* The significantly lower ACI in the older patients (0.678 ± 0.191 ml/mm Hg.m^2 and 0.975 ± 0.191 ml/mm Hg.m^2, $p<0.01$) is attributed to the effect of age on the arterial system and not to any minor differences in arterial blood pressure that may have existed. Patients of similar age (32.0 ± 6.4 yrs and 31.0 ± 6.1 yrs), but with a significant difference in blood pressure (Group I vs Group III) (mean blood pressure 81 ± 5 mm and 106 ± 11 mm, $p<0.01$) were also compared. The ACI was significantly lower in the hypertensive group (0.577 ± 0.170 and 0.975 ± 0.191 ml/mm Hg.m^2, $p<0.01$).

Thus, blood pressure and age appear to act as independent determinants of arterial compliance. When the ACI was compared in the older normotensive subjects (Group II) and the young hypertensives (Group III), no significant

* The significance levels of the difference between groups were determined by Scheffé multiple comparison confidence levels. (Scheffé, H: Analysis of Variance, New York, John Wiley & Sons, Inc., 1959, pp 66-72)

TABLE 1

General Characteristics and Haemodynamic Measurements

Patients	Sex/Age	Body Surface Area-m^2	Cardiac Output Liters/min	Blood Pressure Systolic/Diastolic/Mean mmHg	Ascending Aorta Total Peripheral Resistance Index (TPRI) mmHg.m^2/ml/sec	Arterial Compliance Index (ACI) ml/mmHg/m^2
Group I:	*Normotensive patients under 40 years of age*					
1	M/32	2.21	5.2	86/ 69/ 75	1.91	1.08
2	W/22	1.56	5.9	101/ 65/ 77	1.22	1.17
3	M/26	1.88	6.9	103/ 73/ 83	1.36	1.13
4	W/39	1.80	5.5	110/ 73/ 85	1.67	0.71
5	M/35	1.86	5.8	111/ 72/ 85	1.64	0.80
6	M/36	2.04	5.6	115/ 74/ 88	1.92	0.82
7	M/39	2.10	6.5	96/ 65/ 76	1.47	1.20
8	W/27	1.34	4.3	102/ 67/ 79	1.48	0.89
Group II:	*Normotensive patients 40 years of age and over*					
9	M/48	1.83	4.0	99/ 64/ 75	2.06	0.76
10	M/45	1.71	2.9	94/ 65/ 75	2.65	0.79
11	M/53	1.86	5.2	105/ 69/ 81	1.74	0.90
12	M/59	1.97	4.9	106/ 70/ 82	1.98	1.01
13	M/50	1.80	4.2	125/ 80/ 95	2.44	0.57
14	W/46	1.52	3.1	118/ 79/ 88	2.59	0.45
15	M/58	1.92	4.9	102/ 63/ 76	1.79	0.74
16	W/74	1.46	3.7	103/ 51/ 69	1.63	0.49
17	M/51	2.13	2.8	117/ 78/ 91	4.15	0.46
18	M/47	1.96	6.0	128/ 79/ 95	1.86	0.65
19	W/47	1.77	4.3	118/ 74/ 89	2.20	0.61
20	W/49	1.62	3.8	114/ 59/ 77	1.97	0.47
21	M/67	1.78	3.2	93/ 71/ 78	2.60	0.92
Group III:	*Hypertensive patients under 40 years of age*					
22	M/35	2.00	6.6	144/ 91/109	1.98	0.71
23	W/39	1.60	2.1	155/113/127	5.81	0.31
24	M/24	1.98	2.0	113/ 85/ 94	5.58	0.53
25	M/35	1.97	4.4	133/ 87/102	2.74	0.52
26	M/26	2.09	7.5	137/ 85/103	1.72	0.80
27	M/27	2.15	7.2	129/ 86/100	1.79	0.59

difference was noted (0.678 ± 0.191 and
0.577 ± 0.170 ml/mm Hg.m^2, n.s.). Data
obtained from the brachial artery site were
similar to those shown for the aorta.

Since calculations of
ACI involve total peripheral vasomotor
resistance, R_v (equation 10, appendix) the
contribution of R_v to the correlation of
ACI with systolic blood pressure was
evaluated. The correlation coefficient
of ACI with systolic blood pressure was
$r = -0.754$, $p<0.01$. When the contribution
of R_v to this correlation was removed by
partial correlative analysis, the correla-
tion was similar, $r = -0.626$, $p<0.01$.
This finding supports the observed decrease
in ACI with increasing blood pressure
(Fig. 3) as being real and not a spurious
mathematical relationship dependent on
any contribution of R_v to blood pressure.

To test the reliability of
the estimates of systemic arterial complia-
nce derived from pressure-pulse contours
recorded in a peripheral artery, measure-
ments were made at two arterial sites:
one peripheral, the brachial, and one cent-
ral, the ascending aorta, in 33 subjects
(6 of whom did not meet the criteria for
Groups I-III, Table 1). Measurements were
made consecutively in the brachial artery
and the aorta at approximately 30-second
intervals. Mean arterial compliance
indices derived from pressure-pulse
contours at the two sites were similar, and
the within-patient correlation coefficients
for arterial compliance index derived from
measurements at the two arterial sites
approached unity (Fig. 4).

DISCUSSION

Aterial compliance, like
total peripheral resistance, is an import-
ant part of total systemic arterial
impedance (19,20). The measurement of
arterial compliance in cardiovascular
diseases, however, has received less
attention than peripheral resistance,
probably as a consequence of the lack of
an available technique for directly
measuring compliance in man, rather than
any lack of interest. The index of
arterial compliance used most commonly is
an indirect one, not based directly on
arterial pressure-volume relationships,
namely arterial volume distensibility
calculated from the arterial pulse wave
(conduction) velocity (5,8). Most previous
reports of arterial compliance measurements
in man of necessity have been _in vitro_

studies on arteries removed from cadavers
(1,2).

In the present method arterial
compliance was calculated from arterial
pressure-pulse contours and haemodynamic
measurements. The estimation of compliance
is based on the relationship of the change
in systemic arterial volume during a
selected interval of the diastolic runoff
period, to the fall in pressure that this
change in volume produces. Since this
method is a direct expression of arterial
pressure-volume relationships, it carries
potential advantages over indirect estima-
tes of arterial compliance, such as those
based on pulse wave velocity measurements
(5,8). It should be emphasised that
what is calculated here is an overall
compliance figure, representing a weighted
mean of the differing compliances, of the
systemic arteries in different parts of
the arterial system.

In the present study, an
effect of blood pressure and of age on
systemic arterial compliance was demonst-
rated. This finding is in agreement
with previous _in vitro_ studies (1,2).
Concordance with the findings of these
studies, in which arterial compliance was
measured directly _in vitro_, provides
support for the usefulness of the present
method for _in vivo_ evaluation of systemic
arterial compliance in man. However, it
should be stressed that the accuracy of
the method presented here for the estima-
tion of overall systemic arterial complia-
nce is dependent on the acceptability of
two assumptions made to facilitate the
calculation of change in arterial volume.
First, that resistance to flow out of the
central arterial system, vasomotor resis-
tance (13), is constant above a pressure
of 20 mm Hg (16,17) and second, that the
pressures used during diastolic runoff
are the same throughout the central
arterial system. The assumption of a
linear pressure-flow relationship seems
reasonable since this is the same
assumption used in the routine calculation
of total peripheral resistance. The second
part of assumption one, that of zero flow
below 20 mm Hg, is perhaps more contentious.
The work of Whittaker and Winton (16)
and Girling (17) appears to demonstrate an
approximately linear relation of flow to
pressure in mammalian systems, except at
very low perfusing pressures, such as bel-
ow 20 mm Hg. However, there is some
dispute on this question of the linearity
of the pressure-flow relationship. Green

et al. described the variation of flow with
pressure as being an approximately expon-
ential rather than linear function (21).
We have adopted the most generally held
view, that of a linear pressure-flow
relationship above 20 mm Hg, in computing
ΔV from the arterial pressure-pulse
contours. The 20 mm Hg value is somewhat
arbitrary, but approximates the pressure
at which flow is zero (16,17) and has been
used most often by workers in this area
(22,23,24).

In the present study
systemic arterial compliance proved to be
related to the level of the blood pressure.
This relationship was of two types: a
reduction in compliance in patients with
essential hypertension, and spontaneous
changes in compliance with respect to
variation in blood pressure. This change
in arterial compliance with spontaneous
changes in blood pressure presumably
reflects the nonlinear viscoelastic
behaviour of arterial walls as they are
distended. With small spontaneous rises
in blood pressure, in the somewhat more
distended arterial tree, the compliance
was lower; conversely, with small
decreases in pressure, compliance rose.

In the hypertensive
patients, arterial compliance index was
clearly below normal. The presence of
diminished systemic arterial compliance in
hypertension could be an expression of
this phenomenon of reduced distensibility
of the arterial tree at higher distending
pressures and larger arterial volumes.
An additional mechanism, however, may be
operating. In established hypertension
structural changes develop in the arterial
wall. (1,11). The subnormal compliance
index in hypertension may be partly a
consequence of these structural changes.

Diminished arterial
compliance in hypertension may possibly
have important pathophysiological
consequences. One example may be the
subnormal arterial baroreflex sensitivity
noted in essential hypertension (25-27).
This may well be secondary to reduced
distensibility of the arterial wall at the
site of the carotid and aortic arterial
baroreceptors. Such a relationship between
decreased arterial compliance index in
essential hypertension and reduced
baroreflex sensitivity has recently been
reported (12).

A reduction in arterial

compliance with aging has been reported
here and by others (1,2,6). As in
hypertension, this loss of arterial disten-
sibility with age probably is partly based
on structural changes in the arterial
wall (1,11). This reduction of arterial
compliance with age may well have
pathophysiological consequences. Systolic
hypertension in the elderly is widely
regarded to be an expression of reduced
arterial compliance secondary to atheroscl-
erosis and also nonatherosclerotic aging
of the arterial wall (3,11).

Of importance, in terms of
the possible clinical utilization of
measurement of arterial compliance, is the
finding that estimates of arterial
compliance, as derived from the pressure-
pulse contour of a peripheral artery
such as the brachial in the antecubital
area, agree closely with values obtained
with the catheter tip in the ascending
aorta. The relative simplicity of
percutaneous brachial artery puncture
would support the brachial artery as the
site of choice for determining arterial
compliance if this measure is found to be
clinically useful. Differences between
central and peripheral wave forms were
noted, but these involved only the dicrotic
wave and systolic portion of the pressure-
pulse contour. This finding has also been
reported by O'Rourke (18). Since we
calculated arterial compliance from the
diastolic portion of the curve, distal to
the dicrotic wave, interference from such
factors as reflected waves had no
appreciable effect on this portion of the
aortic and brachial pressure-pulse contour.

We have developed a method
of estimating overall arterial compliance,
this representing a weighted mean of the
differeing compliances in the arteries
throughout the arterial system. As such,
the measurement is a value of physiological
significance because it is a measure of
one component of the impedance against
which the left ventricle must work (19,20).
Although arterial compliance is a function-
ally important component of systemic
arterial impedance, its systematic study
in human disease has been somewhat
neglected because of lack of suitable
methods. It is hoped that the described
technique for evaluating overall systemic
arterial compliance index will prove useful
in evaluating the overall status of the
cardiovascular system.

APPENDIX

The formal derivation of the equation for calculation of arterial compliance index

$$F_O(t) = \frac{1}{R_V}(P(t)-20) \qquad (1)$$

$F_O(t)$ = flow out of arterial system (ml/sec)

$P(t)$ = pressure (mm Hg)

R_V = total peripheral vasomotor resistance (mm Hg/ml/sec)

To find the value of R_V the data may be averaged over several cycles (time) $(0, T_1)$:

$$\frac{\int_0^{T_1} F_O(t)\ dt}{T_1} = \frac{1}{R_V}\frac{\int_0^{T_1}(P(t)-20)\ dt}{T_1} = \frac{1}{R_V}\frac{\int_0^{T_1}P(t)\ dt}{T_1} - \frac{20}{R_V} \qquad (2)$$

However:

$$\frac{\int_0^{T_1} F_O(t)\ dt}{T_1} = \text{average flow out of arterial system or cardiac output (CO)}$$

And:

$$\frac{\int_0^{T_1} P(t)\ dt}{T_1} = \text{average (mean) blood pressure (MBP)}$$

Therefore:

$$CO = \frac{1}{R_V}(MBP-20) \qquad (3)$$

Or:

$$R_V = \frac{MBP-20}{CO} \qquad (4)$$

For any defined volume (in this case, the central arterial system) conservation of mass requires that the rate of change of the volume is equal to the sum of all flows into the volume (F_i) less the sum of all flows out of the volume for incompressible fluid.

$$\frac{dV(t)}{dt} = \Sigma\ F_i(t) - \Sigma\ F_O(t) \qquad (5)$$

During diastole there is no flow into the system and therefore:

$$\Sigma\ F_i = 0 \qquad (6)$$

Also, for subjects with competent aortic valves and no extracardiac shunts the only flow out of the system is that flow to the capillary bed and therefore, from equations (1), (5) and (6):

$$\frac{dV(t)}{dt} = -\frac{1}{R_V}(P(t)-20) \qquad (7)$$

Compliance is a measure of change in volume in the arterial system per unit change in pressure,

$$C = \frac{\Delta V}{\Delta P} \qquad (8)$$

Over any interval of time t_1 to t_2, during diastole we may calculate the change in volume in the central arterial system by integrating equation (7):

$$\Delta V = \int_{t_1}^{t_2} \frac{dV(t)}{dt}\ dt = -\frac{1}{R_V}\int_{t_1}^{t_2}(P(t)-20)\ dt \qquad (9)$$

and therefore:

$$C \text{ or } AC = \frac{\Delta V}{\Delta P} = -\frac{\int_{t_1}^{t_2} (P(t)-20) \, dt}{R_v (P(t_2)-P(t_1))} = \frac{\int_{t_1}^{t_2} (P(t)-20) \, dt}{R_v (P(t_1)-P(t_2))} \qquad (10)$$

or:

$$ACI = \frac{\frac{\Delta V}{\Delta P}}{BSA} = \frac{\Delta V}{\Delta P} \cdot \frac{1}{BSA} = \frac{AC}{BSA}$$

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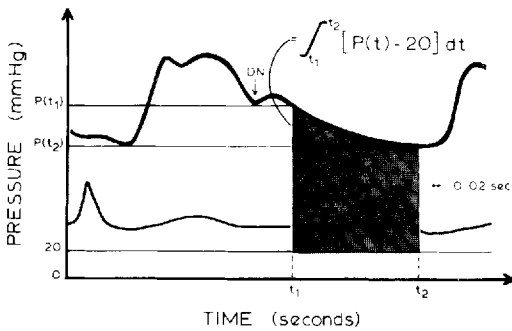
FIGURE 1. Pressure-pulse contour. The shaded area, calculated by the equation shown, is bounded on the pressure axis by a portion of the diastolic pressure-pulse contour above (P_1-P_2) and by 20 mm Hg below (see text). The lower limit of the time boundary, t_1, is 0.06-0.08 sec after the peak of the dicrotic wave and the upper limit, t_2, is 0.20-0.24 sec after t_1. DN = dicrotic notch. Lead II of an electrocardiogram is shown below.
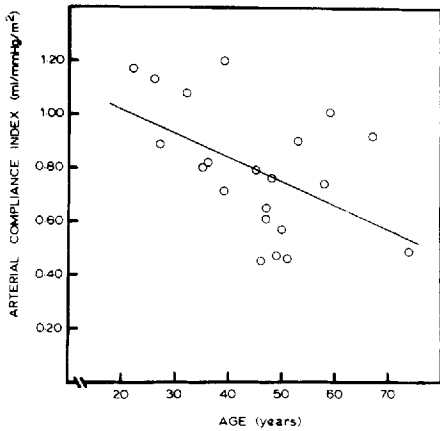

FIGURE 2. The correlation of age with systemic arterial compliance index (ACI) (determined from the ascending aorta) in 21 normotensive subjects; $r = -0.496$, $p < 0.05$. The regression equation, ACI = 1.195 - 0.009 (age), describes the linear relationship of age and ACI

FIGURE 4.
Systemic arterial compliance index as determined from the ascending aorta and brachial artery in relationship to the line of identity.
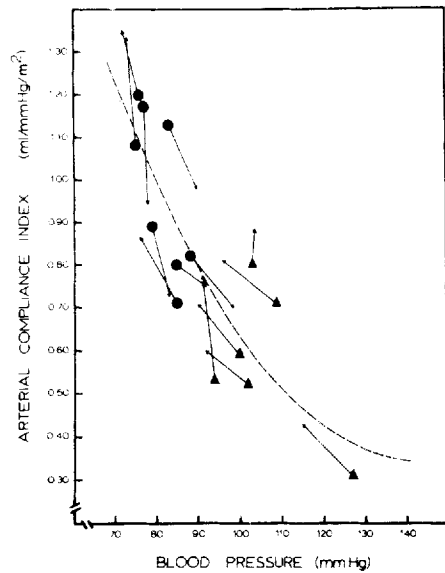

FIGURE 3. The relationship between mean arterial compliance index (ACI) and blood pressure (determined from the ascending aorta) in age-matched normotensive (closed circles) and hypertensive (closed triangles) subjects, is indicated by the curvilinear interrupted line. This quadratic equation is, ACI = 4.0045 - 5.3244 X 10^{-2} BP + 1.9507 X 10^{-4} BP^2 ($R^2 = 0.73$, p 0.001). Arrows represent instantaneous variations in compliance, dv/dP, with blood pressure in each normotensive and hypertensive subject.
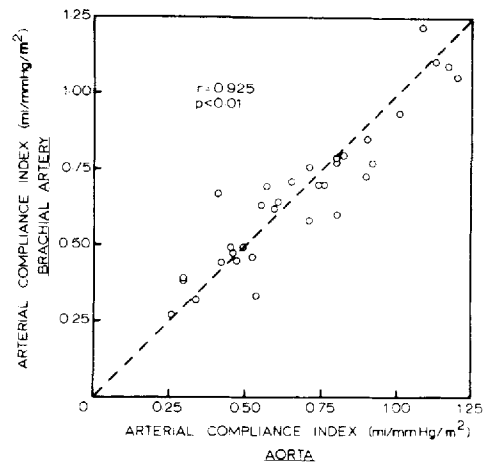
DISCUSSION - CHAIRMAN: PROFESSOR TAYLOR

DR. O'ROURKE: There are some other measurements of arterial compliance which are well established, such as measurements of pulse-wave velocity. I wonder if you have had the opportunity to measure pulse-wave velocity, to compare it with your index of compliance and to correlate it with some of these other factors that you have correlated with your own compliance method.

DR. RANDALL: I think that's important. It has to be compared with some other standard method. We think this is more reliable than pulse-wave velocity. We have not done a large number of patients, but we have at the time of cardiac catheterisation, placed a catheter in the root of the aorta and pulled it back to get distance and then calculated pulse-wave velocity. There is a weak correlation in nine patients with a significance of just over 0.05. We are at present continuing to about twenty-five or more patients to confirm this.

DR. WIKSTRAND: I wonder if you have applied any correction for initial volume? You must have both volume and wall stiffness here and with the Doppler method of O'Rourke for instance, it should be possible to apply a correction.

DR. RANDALL: Not to correct for the initial volume, I guess that's the difference in absolute and specific compliance; we are just looking at what happens to the pressure for a given change in volume for whatever volume is in that system. The only attempt to correct for initial volume we have used is body surface area to correct the differences in the total body size. But we have not done anything to look at V_0 in the calculation of compliance.

DR. GOW: The weak correlation you just showed between pulse-wave velocity and the parameter you're measuring is surprising, considering that established studies have shown that the relationship between wave velocity and arterial elasticity and therefore compliance is rather good.

DR. RANDALL: Yes, that doesn't bother me much, in that the patients that we have studied so far all were within a relatively narrow age range and blood pressure range. When we get patients of age say 20 to 80 years, or patients with wide ranges in blood pressure, I think we will see the correlation. But I am surprised that we almost have it just using patients between 40 and 55 years of age, where the pulse-wave velocity when you really look at those studies, was almost the same in all patients until you get up to a fairly old age and in all of the studies they showed good correlation. There are 60, 70 and 80 year old patients in there and I don't know whether they really had stiff vessels.

DR. HUNYOR: Can I ask whether frequency response of the pressure recording device, catheter delay etc. are relevant for your type of measurement and particularly did you take these factors into account when you were measuring the velocity changes that Dr. O'Rourke referred to?

DR. RANDALL: I would think that is important. We had a Bio-Engineer who was one of the workers in this are. We used fluid filled catheters and we have plans now to use pressure tip transducers to look at this. I think it would make a difference. I don't think it would change the overall findings of differences in groups of patients, hypertensive versus normotensive.

DR. BROD: Dr. Randall, I think it was a very nice presentation, but why was the last patient you showed us in the Intensive Care Unit with a blood pressure of 195/90mm and why did you treat him with nitroprusside?

DR. RANDALL: This patient was a 65 year old lady who presented to the Coronary Care Unit with acute myocardial infarction, and we thought she had a phaeochromocytoma. Her diastolic pressure was going from 120 to around 220 and at the time of admission she had a diastolic blood pressure of 150. We could not control it other than with nitroprusside and we put the catheters in.

DR. O'ROURKE: I am still pretty worried about this, because it is based on this assumption of a continuous fall of pressure in the aorta where you were measuring it. Under some circumstances you can have the pressure actually rising during diastole to a second peak and even on the first slide in which you showed where you were taking the measurement, you are obviously part the way up a diastolic wave. I think in people with hypertension where you have an almost exponential fall-of and you don't have any oscillations in the arterial system, this type of thing is probably pretty reasonable, but under circumstances where there are

prominent diastolic waves, I think it
becomes a little bit worrying from a basic
theoretical point of view.

DR. RANDALL: I think that's important but
I don't think it's always the case; that
first slide was the first one I made when
I first studied this about two years ago.
Now we always move a fair distance off the
dicrotic wave. I think that if you use
a small segment in the middle of the
contour that you avoid most of the
dicrotic wave. Even if you don't, I wonder
whether that increase in area is a reflec-
tion of what is going on with the pressure
volume relationship. That I don't know,
but we have some studies now planned with
the Bio-Engineers where we plan to look
at some other events in diastole; where we
can get rid of the many changes in events
in systole, such as ejection rates and
dp/dt.