Influence of Aortic Insufficiency on the Hemodynamic Significance of a Coronary Artery Narrowing

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SUMMARY The coronary hemodynamic effects of controlled aortic insufficiency (Al) were studied in 10 dogs. Coronary blood flow (CBF), before and during reactive hyperemia (RH) with graded coronary diameter narrowings (CN), aortic (Ao) and left ventricular (LV) pressures (P), and aortic blood flow (AoF) were recorded. Opening an adjustable basket catheter, positioned across the aortic valve, created reversible Al quantitated from phasic AoF. Al was regulated so that mean CBF was similar with or without Al. During Al, heart rate and systolic AoP were unchanged, but diastolic AoP declined 14 mm Hg (mean) and end-diastolic LVP increased 8 mm Hg, both p < 0.05. With CN ≥ 85%, mean CBF decreased with or without Al. Coronary resistance was similar with or without Al. During Al with no CN, peak RH CBF declined significantly and was similar to peak RH with 70% CN without Al. Furthermore, Al with 60% CN caused additional reduction in peak RH and was similar to peak RH with 80% CN without Al.

These data suggest that CBF reserve, exposed during RH, is decreased during AI. With Al, a given CN has coronary hemodynamic properties similar to higher degrees of CN without AI. These results may relate to clinical findings of ischemia in patients with AI and no or moderate CN.

AORTIC INSUFFICIENCY (AI) is sometimes accompanied by clinical findings that suggest ischemia, including angina pectoris. 1-4 Although these findings may imply severe valvular disease and a poor prognosis, they do not necessarily indicate accompanying severe coronary artery disease. 1-4 Why angina accompanies Al without severe coronary disease is unclear. 5-9 Altered coronary blood flow (CBF), 3, 4, 6-9 with or without an associated increase in myocardial work, 3, 10 was suggested from previous experimental observations. The possible influence of these AI-related hemodynamic alterations on the coronary hemodynamic properties of a given coronary artery narrowing have, to our knowledge, not been described.

The purpose of this investigation was to study coronary hemodynamics and aortic and left ventricular pressures during reversible AI in an animal model. CBF with graded coronary artery narrowings was examined before and during reactive hyperemia. The magnitude of AI was controlled using a basket-catheter instrument that was positioned across the aortic valve and did not damage the aortic valve cusps. Thus, the magnitude of AI could be controlled so that large CBF and systemic hemodynamic changes were avoided. Furthermore, the aortic valve could be made transiently insufficient, permitting measurements with or without AI in close temporal relation in any given dog.
Methods

Animal Preparation

Ten large, healthy mongrel dogs (average weight 27 kg, range 20-30 kg) were studied. After an overnight fast and premedication with morphine sulphate (1 mg/kg), the dogs were anesthetized with alphachlorolose (100 mg/kg) and intubated. Respiration was controlled (Harvard apparatus #623) to maintain arterial blood gases and pH within the physiologic range. A left thoracotomy was done in the fifth intercostal space and the heart was supported by a pericardial cradle. The circumflex coronary artery and ascending aorta were isolated, the sinus node was crushed, and the left carotid and right femoral arteries were exposed.

Instrumentation

Appropriately sized electromagnetic flow probes (Biotronex, series 6000 and 5000) were positioned on the proximal circumflex artery and ascending aorta and connected to a two-channel sine wave flowmeter (Biotronex BL-613) to measure circumflex and aortic blood flow. The probes were carefully matched to the diameter of each artery. A probe that would neither slide loosely nor visibly constrict the vessel was chosen. The flowmeter was operated at its highest nominal frequency setting (100 Hz, -3db), which provided, according to calibration in our laboratory, a constant amplitude response within ± 5% from 0-47 Hz and a linear phase shift. Static calibrations of the flow probes were performed in vitro by means of a hydraulic system similar to that used by Malooly et al.1 Since hematocrit changes have been shown to have little effect on the flowmeter voltage output for a given flow,12 physiological saline and a section of artery were used during calibration. A series of different flow rates was used for each probe, and the flowmeter output was linear (bidirectional) over a wide range of flows. Catheter-tip micromanometers (Millar) were positioned in the ascending aorta from the femoral artery and the left ventricle from the left atrial appendage to measure high-fidelity aortic and left ventricular pressures. A snare, made from 00 Tevdek suture material attached to a machinist’s micrometer (Starrett), was used for calibrated reduction of circumflex diameter. This technique created very short narrowings, approximately 1 mm long, and its use in our laboratory has been described in detail elsewhere.13 Reversible AI was created using a technique similar to that previously used by other investigators.5, 14, 18

An open-end #8F cardiac catheter was inserted into a carotid artery and advanced, while continuously monitoring pressure, until the tip was in the left ventricular cavity. A collapsible wire basket (Dotter Retriever, Cooke Catheter Co) was then threaded through the catheter so that the tip of the basket was just protruding from the catheter. As the wire basket passed through the magnetic field of the aortic flow probe there was a small shift in the aortic flow baseline without distortion of the signal. This new baseline was steady as long as the basket catheter was through the aortic flow probe. The basket catheter was then opened in the left ventricle. The open basket was then positioned at the level of the aortic valve, creating acute AI. The magnitude of AI was assessed by observing changes in the phasic aortic flow signal, aortic diastolic pressure and left ventricular end-diastolic pressure. The magnitude of AI was limited with small amounts of catheter manipulation so that mean CBF was similar to that recorded without AI (control). Further withdrawal of the basket or reinsertion of the basket into the left ventricle reversed the acute AI.

Recordings

Pulsatile and mean circumflex and aortic blood flows and aortic and left ventricular pressures were continuously monitored and recorded at a paper speed of 2.5 mm/sec (Electronics for Medicine DR8) and stored on magnetic tape (Hewlett Packard 3960). Recordings of the phasic flow and pressure signals at 50 and 100 mm/sec were made at each degree of circumflex narrowing, with and without AI. The circumflex artery was occluded for 10 seconds. Recording continued after release of occlusion during reactive hyperemia. These reactive hyperemic responses were repeated at each degree of circumflex narrowing.

Measurements and Calculations

Circumflex blood flow before the 10-second occlusion was used as an index of CBF. Peak reactive hyperemic circumflex blood flow was taken as an index of CBF reserve. For each dog, a normalized peak reactive hyperemic CBF value was calculated as the ratio of peak reactive hyperemic CBF to CBF at no narrowing without AI. This was the value with which hyperemic CBF responses with and without AI and with and without circumflex narrowing were compared. Measurements of CBF, aortic flow and aortic and left ventricular pressures were made over 10 heart beats. At each degree of circumflex narrowing with and without AI these measurements were averaged.

Percent coronary artery diameter reduction (0-100%) was calculated as percent circumflex narrowing = (micrometer reading at no narrowing minus reading at each step)/total micrometer excursion × 100%. Measurements were made with diameter reductions of 0, 40, 60, 70, 80, 85, 90, 95 and 100%. These degrees of narrowing were chosen because previous work13, 16, 17 suggested that CBF declines with snare-type narrowings > 80%. During reactive hyperemia, CBF declines with snare narrowings > 40% compared with responses without narrowing.13, 16-18

An index of mean coronary artery resistance was calculated as CRm = mean aortic pressure (mm Hg)/mean CBF (ml/min). The method of Denison and colleagues19 was used to calculate end-diastolic coronary resistance as CRed = end-diastolic aortic pressure (mm Hg)/end-diastolic CBF (ml/min). This value is thought to represent an index of coronary
arteriolar tone, because extravascular compression is minimal at end-diastole and is undergoing only minimal change with respect to time. Because the pressure gradient across the myocardium in diastole may be altered with AI, an estimate of coronary resistance (CR) that included the myocardial pressure gradient was also derived. CR = [end-diastolic aortic pressure (mm Hg) minus end-diastolic left ventricular pressure (mm Hg)]/end-diastolic CBF (ml/min).

The area under the systolic and diastolic phasic CBF signal was quantitated by planimetry. Zero CBF was reconfirmed during each 10-second complete circumflex occlusion used to evoke the hyperemic response. Similarly, the area under the aortic flow signal was also determined by planimetry. The portion of the diastolic flow signal at the onset of the R wave, without AI and the basket closed in the left ventricle, was taken as zero aortic flow. The aortic flow signal above this zero baseline was considered forward flow and that below regurgitant flow. The magnitude of AI was quantitated as the ratio of the regurgitant and forward components of pulsatile ascending aortic flow. This value was termed the regurgitant fraction.

Typical Experiment

Control: Each experiment consisted of three experimental periods beginning with a first control sequence. A sequence was defined as duplicate recordings of CBF, aortic flow and aortic and left ventricular pressures before and after a 10-second complete coronary artery occlusion. This procedure was repeated with a series of circumflex narrowings to complete the first control sequence. During this control sequence, the basket-catheter remained closed (basket withdrawn into the end of the catheter) within the left ventricle.

Aortic insufficiency: Next the basket was opened and slowly withdrawn, monitoring circumflex and aortic blood flows, until AI occurred as indicated by downward displacement of the aortic blood flow signal in diastole. The magnitude of AI was regulated by advancing or withdrawing the basket so that mean CBF was similar (± 10%) to that observed without AI. After flow and pressure stabilization (± 5%), which occurred in less than 5 minutes, the AI sequence of recordings were made in the same manner as described above for the control sequence. The series of circumflex narrowings, with complete 10-second occlusions, was repeated.

Second control: The basket was then withdrawn further or readvanced into the left ventricle reversing the acute AI, and measurements were repeated again according to the sequence outlined above. This was the second control sequence.

Confirmation Studies

Our ability to produce AI reversibly (fig. 1) and regulate the magnitude of AI was indicated by recording a stable aortic blood flow pattern in diastole and confirmed by in vivo aortic root angiography (fig. 2). Aortic valve competence with the basket closed and the catheter positioned in the left ventricle was evaluated during aortography. With the closed basket catheter positioned within the left ventricle, each experiment was ended with an overdose of intravenous potassium. The electrical zero aortic flow and CBF baselines were checked with the circulation arrested.

Each dog included in this report also had a detailed
postmortem examination of the heart and ascending aorta to exclude damage to the aortic root or valve. Postmortem aortic valve competence was checked by filling the aortic root with saline and inspecting the left ventricle for leakage. The aortic valve leaflets were then carefully inspected.

Analysis of Results

Only experiments in which CBF and the peak reactive hyperemic response were stable (± 10%) comparing the first and final control sequence were accepted for analysis. The control sequence with the lower reactive hyperemic response was compared with the response observed during Al. When there was a small (≤ 10%) response variation between duplicate recordings, the higher CBF and/or reactive hyperemic response was always used for that degree of circumflex narrowing. If repeat recordings at any degree of circumflex narrowing varied by more than ± 10% the experiment was rejected.

Mean values and standard errors were calculated. An analysis of variance, appropriate multiple comparison procedures and a paired t test were used for statistical comparisons. A p value ≤ 0.05 was considered statistically significant.

Results

No Al was seen during aortic angiography when the basket-catheter was inside the left ventricle, and no damage to the aortic valve leaflets was seen on post-mortem examination of the heart and ascending aorta.
mortem examination. All dogs included in this report had competent aortic valves when the aortic root was filled with saline. The electrical zero aortic flow and CBF baselines set during the experiments agreed with those found with the circulation arrested after completion of the experiments.

**Aortic and Ventricular Hemodynamic Responses**

The regurgitant fraction averaged 33 ± 5% (range 13–66%). Ascending aortic and left ventricular pressures and heart rate during control and AI sequences for all dogs are shown in figure 3. Heart rate (135 ± 3 to 130 ± 2 beats/min, control to AI) and peak systolic pressure (140 ± 3 to 141 ± 2 mm Hg) were similar during both periods. During AI, however, aortic diastolic pressure declined (101 ± 4 to 87 ± 7 mm Hg, p < 0.05) and left ventricular end-diastolic pressure increased (8 ± 2 to 16 ± 2 mm Hg, p < 0.05) (table 1).

**Coronary Hemodynamic Responses (table 1)**

Mean circumflex blood flow with no narrowing was 43 ± 5 ml/min and was similar during AI (45 ± 7 ml/min, NS). This similarity in mean CBF with and without AI was apparent at all degrees of coronary narrowing (fig. 4). As circumflex narrowing increased

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### TABLE 1. Coronary Hemodynamic Responses

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* p < 0.05, control vs AI, by paired t test.

Abbreviations: C = control; CBF = mean coronary blood flow; CBFed = end-diastolic coronary blood flow; RH CBF = peak reactive hyperemia coronary blood flow; AoP = mean aortic pressure; Aodp = aortic end-diastolic pressure; LVEDP = left ventricular end-diastolic pressure.

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**FIGURE 4. Circumflex blood flow during control and aortic insufficiency (AI) at different degrees of coronary artery diameter reduction. CBF was similar with and without AI and decreased during both control and AI with coronary narrowings > 80%**.
The fraction of CBF during systole and diastole was different during control and AI (fig. 5). The systolic fraction of CBF increased from 16 ± 2% to 29 ± 2% (p < 0.05) during AI. The diastolic fraction declined from 84 ± 2% to 71 ± 2% (p < 0.05) during AI. End-diastolic CBF was lower during AI, decreasing from 67 ± 8 ml/min to 53 ± 8 ml/min (p < 0.05) during AI.

Coronary artery resistance was not significantly changed during AI. CRm was 3.07 ± 0.39 mm Hg/ml/min during control and 2.6 ± 0.27 mm Hg/ml/min during AI. CRed was 1.69 ± 0.16 mm Hg/ml/min during control and 2.04 ± 0.30 mm Hg/ml/min during AI. CR (1.55 ± 0.16 mm Hg/ml/min) was also similar during AI (1.60 ± 0.19 mm Hg/ml/min).

Peak reactive hyperemic flow in each experiment was reduced during AI. Peak reactive hyperemic CBF declined from 156 ± 23 to 118 ± 18 ml/min during AI with no circumflex narrowing (p < 0.05). Without coronary narrowing, normalized peak reactive hyperemic flow decreased from 3.6 ± 0.2 to 2.6 ± 0.2 with AI (p < 0.05). A significant decrement in this value was noted at each degree of narrowing < 90% during AI (fig. 6). A significant decrease in both control and AI hyperemic responses was observed with narrowing > 40% compared with no narrowing during control or AI, respectively (fig. 6).

**Discussion**

Reversible AI was created and CBF responses were studied with graded coronary artery narrowing in dogs. We found that coronary reserve, defined as the increase in CBF induced by a hyperemic stimulus, is limited during AI. This reduction in hyperemic flow...
responses occurred with AI of only moderate magnitude. Thus, coronary reserve was reduced without coronary artery narrowing during AI, and significant reduction in reserve was observed with all degrees of narrowing studied. Because the basket-catheter was manipulated so that mean circumflex flow with AI was not significantly changed, the magnitude of AI was usually only mild or moderate (angiographic 2+). Different results might have been found with AI of greater magnitude.

Several possible limitations of this study deserve comment. First, although heart rate and aortic systolic pressure were unchanged during AI periods, significant differences between aortic diastolic and left ventricular end-diastolic pressures occurred. Aortic diastolic pressure decreased and left ventricular end-diastolic pressure increased. These pressure differences with AI were probably not due to marked deterioration in ventricular function related to the duration of the open-chest experiment, anesthesia, etc. In all instances these values returned to previously recorded levels when the basket was closed. Second, because average heart rate in these studies was rapid (130–135 beats/min), the full hemodynamic effect of AI on the coronary circulation and myocardium might have been diminished. A third consideration related to the length of the coronary narrowings created. We have previously shown that narrowing length has a pronounced effect on coronary hemodynamics.13, 17, 22 Snare narrowings, however, are short (≤ 1 mm); therefore, care should be taken when attempting to equate the percent diameter narrowing necessary to alter CBF with snare-type narrowings to that necessary to alter CBF with longer narrowings. We have not studied the effects of longer narrowings on CBF in AI. Fourth, the absolute magnitude of peak coronary flow after a 10-second occlusion should not be taken as the maximum possible circumflex arterial bed flow. We used reactive hypemic CBF after a 10-second coronary occlusion as an index of coronary reserve because we found that, in the absence of acute interventions, it is a reproducible response.13, 17, 22 in most canine coronary beds. This study was not designed to evaluate the maximum reserve of the circumflex arterial bed. Further investigation may be indicated to evaluate the effect of AI or other acute interventions on CBF after longer periods of coronary occlusion or pharmacologic stimulation. Fifth, both AI and coronary narrowings were maintained for a relatively short period of time, and the possible occurrence of compensatory left ventricular or coronary hemodynamic alterations over time was not addressed. Attempts to extrapolate these data from an acute animal model to patients with chronic AI must be made with caution.

Although the calibrated snare technique to create coronary narrowing has been used by several investigators,13, 16–18 the precise degree of narrowing created is open to question. In this study, the estimated degree of narrowing was not verified by independent techniques, e.g., coronary angiography or casts. Because we created multiple narrowings both

with and without AI, it was not feasible to check our estimates of coronary narrowing with postmortem coronary casts. In vivo coronary angiography could have been done; however, perceptual problems make it difficult to measure short narrowings accurately.22 In spite of these limitations our estimates of degree of narrowing are not arbitrary. Previous work from our laboratory using both in vivo cineangiography and postmortem casts has shown close agreement (± 10%) with the estimated degree of narrowing.13, 17, 22 Even if the above reasoning about the degree of coronary narrowing is rejected, the observations of this study are still valid. Coronary reserve was usually lower with AI at all micrometer settings.

The fraction of CBF during systole was small without AI and increased with AI (fig. 5). Increased systolic CBF during AI has been noted by others.3, 6–9 The systolic CBF seen during control observations was in agreement with previous phasic CBF observations.24–26 These changes in the phasic CBF pattern during AI may be mediated through alterations in aortic and ventricular pressures. Because mean CBF was similar with and without AI (fig. 4), diastolic CBF decreased while systolic CBF increased. Diastolic coronary perfusion pressure (aortic diastolic pressure) decreased during AI, probably without significant alteration of diastolic filling time, because the heart was not influenced. Left ventricular end-diastolic pressure increased, probably because of the volume load due to AI. This increased preload, within the range of left ventricular diastolic pressures observed, would not be expected to affect diastolic CBF or its distribution.27 The net effect of the aortic and ventricular pressure changes during AI should be a decrease in diastolic CBF. Systolic coronary perfusion pressure (aortic systolic pressure) was unchanged with AI. Archie29 found that increased preload increased systolic CBF in the epicardial myocardial layers in animals with competent aortic valves. It is possible that the increased preload during AI effected an increase in systolic CBF. Mean CBF could be maintained during AI through an increase in the systolic component of CBF, while the diastolic component of CBF decreased. Inspection of phasic CBF flow signals also revealed that the continuous decline in amplitude that usually occurs throughout diastole begins earlier and becomes steeper during AI (figs. 7 and 8). This alteration of diastolic CBF resulted in lower total diastolic and end-diastolic CBF components during AI.

Previous investigation of directional mean CBF change during AI has produced some conflicting results.3, 4, 6–10 In our opinion, these observations are not necessarily discordant. Manipulation of the magnitude of AI markedly altered left ventricular and aortic pressures in several dogs. Concomitantly, mean circumflex flow could change in either direction. In most of the other reported studies of acute severe AI (regurgitant fraction > 60%) without hypotension and marked elevation of left ventricular diastolic pressure, mean CBF usually increased,3, 4 while in this study with a lesser magnitude of acute AI (regurgitant frac-
% CORONARY NARROWING

Figure 7. A typical example of coronary blood flow (CBF, ml/min) responses at different percents of snare circumflex narrowing during control (above) and mild aortic insufficiency (AI) (below). The aortic flow (AoF, ml/sec), aortic pressure (PAo, mm Hg), and left ventricular pressure (PLV, mm Hg), responses during control and AI are shown. Note the increased systolic CBF and the decreased diastolic CBF during AI. Reactive hyperemic responses during mild AI were similar to those during control with a higher percentage of coronary narrowing, e.g., zero AI to 60% control. S = systole; D = diastole.

Figure 8. An example of coronary blood flow (CBF, ml/min) responses at different percents of snare circumflex narrowing during control (above) and severe aortic insufficiency (AI) (below). The aortic flow (AoF, ml/sec), aortic pressure (PAo, mm Hg) and left ventricular pressure (PLV, mm Hg) responses during control and severe AI are shown. With severe AI, CBF is predominantly systolic and a rapid decline in diastolic CBF occurs, becoming negative in end-diastole. Reactive hyperemic responses during severe AI were similar to those during control with a very high percentage of narrowing, e.g., zero AI to 80% control. S = systole; D = diastole.
appropriate with groups of mild, moderate and severe AI.

Coronary resistance did not change significantly during mild-to-moderate AI. These results probably also depend on the magnitude of AI. As AI increases in magnitude, generally larger decreases in mean and diastolic arterial pressure occur, while mean CBF may increase. These changes in coronary perfusion pressure and flow would be expected to decrease mean resistance. End-diastolic CBF, however, decreases and may even be negative with severe AI (fig. 8), suggesting that resistance at this time must be high. These coronary artery pressure-flow changes during diastole are complex and difficult to quantitate with simple ratios, using either mean pressure and flow or pressure and flow at only one time in the cardiac cycle. Further investigation of the influence of AI on pulsatile coronary artery dynamics using a pulse-wave velocity technique recently developed in our laboratory may be helpful in evaluating these changes.

Coronary reserve with AI was uniformly reduced compared with reserve without AI. With the addition of a given coronary narrowing during AI, an additional reduction in coronary reserve occurred. Previous studies showed a reduced coronary reserve with 40–60% snare-type coronary artery narrowings in animals without AI. Furthermore, AI with a 60% coronary narrowing caused reductions in CBF reserve similar to those caused by an 80% narrowing without AI. Thus, during stress such coronary flow limitation could result in inadequate myocardial perfusion. An example of the reduction in CBF reserve with mild AI is shown in figure 7. With severe AI (fig. 8), CBF reserve may be even more limited than suggested from our studies with lesser magnitudes of AI. Alterations of the phasic CBF pattern could also influence the transmural distribution of CBF. Although transmural CBF was not measured in this study, reduced diastolic CBF could result in decreased subendocardial CBF during AI and contribute to inadequate myocardial perfusion. Preliminary observations on transmural CBF distribution during AI have been reported by two groups. Severe AI may decrease endocardial perfusion, while lesser magnitudes of AI do not appear to affect transmural CBF distribution.

In conclusion, CBF reserve in AI is potentially limited, even without coronary narrowing, and can be severely decreased with moderate coronary narrowing. This coronary reserve limitation is comparable to that observed with higher degrees of narrowing without AI. The mechanism of this decreased CBF reserve during reactive hyperemia with AI remains unclear. These findings may relate to clinical observations of myocardial ischemia in patients with AI and without severe coronary artery narrowings.

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The Prognostic Value of Systolic Time Intervals in Angina Pectoris Patients

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SUMMARY Eighty-five subjects with stable angina pectoris and proven obstructive coronary disease were followed prospectively (mean follow-up 4.2 \pm 2.0 years) to assess the value of various predictors of longevity. After patients with congestive heart failure, hypertension, left bundle branch block, valvular heart disease or recent propranolol therapy were excluded, subjects were followed until a major cardiac event (new acute myocardial infarction, cardiac surgery or death) occurred. During follow-up, 22 patients died, 49 survived without events, nine underwent coronary bypass surgery, and five had nonfatal myocardial infarctions. The measurements made at the onset of the study, including cardiothoracic ratio (C/T) on chest x-ray, resting electrocardiographic abnormalities, maximum exercise tolerance testing (METT) data, systolic time interval (STI) measurements (before exercise and 3-4 minutes after METT), and results of cardiac catheterization (55 patients), were analyzed at its conclusion to determine the best predictor of subsequent mortality. Of these measurements, left ventricular ejection fraction, endurance time on METT and C/T were shown to be useful prognostic indicators of subseqent mortality. However, the pre-ejection phase-to-left ventricular ejection time (PEP/LVET) ratio (0.40 \pm 0.05 in survivors vs 0.50 \pm 0.09 in nonsurvivors) in the resting pre-exercise state was significantly more predictive of mortality than the other measurements. On life-table analysis, the difference in survival between subjects with a resting PEP/LVET < 0.50 and those with a resting PEP/LVET \geq 0.50 was highly significant. The measurement of the STIs after maximal exercise testing failed to improve upon the prognostic ability of the simple determination of PEP/LVET in the resting, supine state. STIs provided highly specific noninvasive prognostic information in this group of patients with stable angina pectoris.

DEPRESSED LEFT VENTRICULAR function, particularly as reflected in a reduced angiographic ejection fraction (EF) determined at cardiac catheterization, has been shown to affect adversely the prognosis of both medically and surgically treated patients with coronary heart disease.\(^1\)\(^2\) Measurement of systolic time intervals (STIs) is a noninvasive technique that has shown a close correlation with the angiographic assessment of left ventricular performance in most forms of heart disease, although the correlation of the ratio of pre-ejection phase to left ventricular ejection time (PEP/LVET) with EF is not as close in coronary heart disease as it is in certain other forms of heart disease.\(^3\)\(^4\) While several investigators have found the evaluation of ventricular function by STIs to be useful in patients with coronary disease, there has been no prospective study of the relationship between STIs and subsequent mortality in patients with angina pectoris.\(^5\)\(^6\) This study was designed to determine whether STIs, measured before and 3-4 minutes after a maximal exercise treadmill test, have prognostic value in subjects with stable angina pectoris and proven obstructive coronary artery disease.

Methods

Patient Population

Eighty-five patients (80 men and five women), mean age 56 \pm 7 years, who were considered by two cardiologists to have stable effort-related angina pectoris, participated in this prospective study of the prognostic value of STI measurements. Stable angina pectoris was diagnosed when recurrent attacks of substernal

\(^{1}\)\(^2\) From the Cardiovascular Service and Laboratories, U.S. Public Health Service Hospital, Baltimore, Maryland.

\(^{3}\)\(^4\) Supported by Base Research Program Support of the Federal Health Programs Service.

\(^{5}\)\(^6\) Preliminary results of these data were given in a poster presentation at the 51st Annual Scientific Sessions, American Heart Association, November 14, 1978.

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