Pronus Angina (Angina Pectoris Induced by Stooping or Crouching)*
A Proposed Mechanism

Elieser Kaplinsky, M.D.; and Elio DiSegni, M.D.†

Patients with severe coronary artery disease (CAD) sometimes complain of chest pressure upon crouching or bending-forward (pronus angina). The factors that trigger pronus angina are not clear. We therefore investigated 28 patients with CAD and 26 normal subjects in the sitting, knee-chest, stooping, and squatting positions. Systolic and diastolic blood pressures were found to increase by 13.5 and 19.5 percent (p<0.005) in the stooping position. In addition, left ventricular (LV) ejection time index (LVETI) also increased (p<0.005). Despite the acute rise in aortic pressure, which is expected to lengthen the pre-ejection period index (PEPI), the latter shortened slightly in 10 of 14 (71 percent) patients tested, suggesting augmentation in contractile force during the isovolumic phase. Finally, left atrial size increased (p<0.005) during the knee-chest maneuver, suggesting that the LV size also increases upon bending forward. The effect of stooping on blood pressure was similar in magnitude to that of squatting. It is concluded that the triggering factor for "pronus angina" in severe CAD may be explained as a combination of hemodynamic events which acutely increase myocardial oxygen requirements. (Chest 1993; 104:65-70)

Patients with severe coronary artery disease (CAD) sometimes volunteer the information that crouching or stooping (eg, when tying shoelaces) produces a sensation of definite pressure in the chest or throat, similar to that which they experience during effort angina.† Precordial ST segment depression also appears in some of these patients during crouching (personal observation). This particular type of angina pectoris is intriguing: it is evidently related to the change in body position but the usual mechanisms that trigger angina pectoris, eg, increased myocardial oxygen consumption (MV O2) or decreased myocardial oxygen delivery,1-7 would not appear to be operative in this setting. Why such an apparently benign endeavor should be accompanied by angina is thus not clear. We therefore investigated various hemodynamic changes that could be seen with stooping and could conceivably evoke this type of angina, which may be called "pronus angina" (from the Latin "pronus"— leaning forward, stooping, bending down†), in a fashion analogous to "decubitus angina."

METHODS

Patients

Twenty-eight consecutive patients with CAD (group 1) with moderate to severe angina pectoris and 26 healthy men without any clinical evidence of CAD (group 2) were studied. All group 1 patients (ages 50 to 70) had typical, stable effort angina of at least 6 months’ duration, relieved by nitroglycerin. Selection was not influenced by the presence or absence of pronus angina. In none of the patients except one, was the blood pressure over 160/100 mm Hg. Five had previously sustained a myocardial infarction; none had congestive heart failure. Group 2 included 20 adolescents (18 to 19 years) and 6 older men (50 to 70 years). Both groups were subdivided as lean and obese according to body habitus.

The Stooping Maneuver

Each subject was studied first while sitting; blood pressure (BP) and heart rate (HR) were measured twice after a period of rest of approximately 10 min. The subject was then instructed to bend the torso forward while sitting, so that his hands reached his toes. The BP and HR were then again measured twice. A third set of measurements was obtained 1 min after the subject sat up again. A similar set of measurements was also obtained during squatting in 8 patients with angina and in all 26 normal subjects. The BP was accurately measured each time using a mercury gauge sphygmomanometer according to recommended procedures.†† The arm was supported in order to prevent isometric exercise of the arm muscles, and care was taken that the position of the arm remain constant with respect to the heart to avoid systematic measurement errors.

Korotkoff sounds I and V were used as systolic and diastolic BP. The HR was obtained from the electrocardiogram.

Systolic Time Intervals

Systolic time intervals were measured while sitting and stooping in 14 of the group 1 patients randomly selected on the basis of availability and using standard techniques.11,12 The phonocardiogram was recorded where aortic closure (A0) was heard best. The carotid pulse was recorded according to previously described techniques.11,12 The brachial external pulse was recorded in five patients in whom the carotid pulse could not adequately be recorded while stooping. All recordings were registered on photographic paper at a speed of 100 mm/s.

The following systolic time interval indices were obtained: left ventricular ejection time index (LVETI), electromechanical systolic time index (QAI), and pre-ejection period index (PEPI) according to Weissler.11

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FIGURE 1. Systolic BP responses (mm Hg) to stooping in obese (circles) and lean (squares) control subjects (left panel) and CAD patients (right panel). Bars at right of each set of values represent mean ± 1 standard deviation. BEND = bending forward; CAD = coronary artery disease; NL = normal subjects; SBP = systolic blood pressure.

LVETI = LVET (ms) + 1.7 × HR
QA1 = QA1 (ms) + 2.1 × HR
PEPI = PEPI (ms) + 0.4 × HR

The double product (HR × systolic BP) and triple product (HR × systolic BP × LVET) were measured to obtain an indication of the direction of changes in MVO₂.

M-mode Echocardiogram

Attempts to define the effects of stooping on LV size by echocardiographic measurements were unsuccessful since it was impossible to obtain reliable serial measurements of LV size because of gross changes in the orientation of the transducer vis-a-vis the LV. An attempt to obtain this measurement by leg bending in the supine position (knee-chest) was also less than uniformly successful. However, it was noted that the echoes emanating from the left atrial (LA) wall, aortic valve, and aortic walls were consistently recorded both at rest and during the knee-chest maneuver. The LA dimension was therefore used as an indirect index of changes in LV diastolic dimensions, since the LA and LV are essentially two continuous parts of one single chamber when the mitral valve is open in diastole, and diastolic dimensions must change in the same direction in both (although presumably not with the same magnitude because of differences in wall stiffness).

Statistical Analysis

All data were analyzed by the Student’s paired and unpaired t-test and by analysis of variance and covariance for repeated measurements.

RESULTS

BP and HR Response to Stooping

This maneuver produced significant increases in systolic and diastolic BP in both groups (Fig 1 and 2; Table 1). Mean systolic BP for all 54 subjects increased by 13.5 percent from 126 ± 16 to 143 ± 17 mm Hg (p < 0.005) and decreased again to 123 ± 16 mm Hg after sitting up. Mean diastolic BP also increased by 19.5 percent from 82 ± 11 to 98 ± 15 mm Hg (p < 0.0005) and decreased again to 82.5 ± 10 mm Hg after sitting up. This rise in BP was seen in all four

Table 1 — Systolic and Diastolic Blood Pressure (mm Hg) in the Four Subsets of Subjects

<table>
<thead>
<tr>
<th></th>
<th>Sitting</th>
<th>Stooping</th>
<th>% Change</th>
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<tbody>
<tr>
<td>Lean CAD (N = 15)</td>
<td></td>
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</tr>
<tr>
<td>SBP</td>
<td>127 ± 10</td>
<td>138 ± 16*</td>
<td>8</td>
</tr>
<tr>
<td>DBP</td>
<td>79 ± 10</td>
<td>98 ± 16†</td>
<td>20</td>
</tr>
<tr>
<td>Obese CAD (N = 13)</td>
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<tr>
<td>SBP</td>
<td>133 ± 20</td>
<td>148 ± 20*</td>
<td>11</td>
</tr>
<tr>
<td>DBP</td>
<td>87 ± 13</td>
<td>106 ± 17</td>
<td>23</td>
</tr>
<tr>
<td>Lean control subjects (N = 16)</td>
<td></td>
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<tr>
<td>SBP</td>
<td>121 ± 16</td>
<td>142 ± 14*</td>
<td>17</td>
</tr>
<tr>
<td>DBP</td>
<td>80 ± 10</td>
<td>98 ± 11†</td>
<td>22</td>
</tr>
<tr>
<td>Obese control subjects (N = 10)</td>
<td></td>
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<tr>
<td>SBP</td>
<td>121 ± 12</td>
<td>143 ± 15†</td>
<td>18</td>
</tr>
<tr>
<td>DBP</td>
<td>82 ± 8</td>
<td>102 ± 9*</td>
<td>25</td>
</tr>
</tbody>
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*p < 0.005.
†p < 0.001.
‡p < 0.05.
subgroups of subjects (Table 1).

Mean HR did not change from sitting to stooping (75.9 ± 11 vs 75.3 ± 10 beats per minute, NS) or back to sitting (77.4 ± 10 beats per minute). Individual HR responses to stooping were variable. The double product increased significantly in all four subgroups (Fig 3). Pronus angina was provoked by this maneuver in two patients.

Response to Squatting

Mean systolic and diastolic BP also increased significantly during squatting, the values achieved being similar to those obtained while stooping (Table 2). The double-product increased to the same extent during squatting (from 9,568 ± 1,382 to 12,044 ± 1,835) and stooping (from 9,669 ± 1,764 to 10,078 ± 1,764).

Systolic Time Intervals With Stooping

LVETI increased in 12 of 14 (85 percent) patients, and mean LVETI increased significantly (from 397 ± 36 to 414 ± 32, p<0.005). Mean QA,I remained essentially unchanged (532 ± 29 vs 539 ± 32, NS). Individual QA,I increased in seven patients and remained unchanged or decreased in seven. Individual PEPI decreased in 10 of 14 (71 percent) patients and increased in 4. Mean PEPI decreased from 153 ± 53 to 143 ± 55 during the bending-forward maneuver (not significant, p<0.1). The mean ratio of PEP/LVET decreased slightly but significantly from 0.42 ± 0.25 to 0.39 ± 0.20 (p<0.05). Reduction in PEP/LVET ratio was observed in 12 of 14 patients (85 percent). The triple product rose in 12 of 14 (85 percent) patients from a mean of 2.7 ± 0.6 × 10⁶ to 3.6 ± 0.6 × 10⁶ (32 percent; p<0.005) (Fig 4).

Left Atrial Dimension During Knee-Chest Position

Individual LA dimensions increased by 5 to 20 percent in 21 of 24 (87 percent) patients. The mean increase in LA diameter was statistically significant (from 38.5 ± 8.5 mm to 43.0 ± 10.0 mm, 10 percent; p<0.005). In 11 of 21 patients, both aorta and LA shifted back toward the vertebral column, but the shift of the LA posterior wall was always more prominent than that of the aorta (Fig 5A, top). In the other ten patients, the aorta remained fixed in position (Fig 5B, bottom), and only the posterior atrial wall shifted backward. In all cases, the increase in LA size began within three to four beats after bending and gradually reached a maximum valve within seven to ten beats.

![Figure 3. Double product response to stooping in the four subsets of subjects. Symbols and abbreviations as in Figure 1. HR = heart rate; ob = obese subjects.](image-url)
The size of the aorta always remained unchanged. The LA resembles an elongated sphere in shape but for the sake of simplicity the formula for a simple sphere \( V = \pi \text{diameter}^3/6 \) was used in order to obtain the estimated LA volume. This value increased by 44 percent (from 28.5 ± 20 ml to 41 ± 40 ml; \( p < 0.005 \)).

**DISCUSSION**

**Factors That Trigger Angina Pectoris**

Angina is at times triggered by a primary diminution in myocardial oxygen supply, but an excessive increase in M\( \text{VO}_2 \) constitutes by far the more frequent trigger for angina. Accordingly, it is readily apparent why brisk walking and isometric exercise trigger typical angina pectoris, but it is not so readily understood why stooping should be able to provoke similar episodes of angina. Dyspnea upon stooping is conceivably explained by impaired inspiratory descent of the diaphragm in this position; however, angina pectoris is clearly distinct from dyspnea and is not so readily explained. Smith and Papp first called attention to this phenomenon in 1962 and noted that blood pressure increased in two men who experienced chest pain on stooping to tie their shoe laces, to lift trays, or while gardening. The present study sheds additional light on the probable trigger of pronus angina: BP increases consistently and significantly immediately upon stooping and remains elevated until the subjects sit back. The effect of stooping on BP is similar to that seen with squatting. While HR behaves inconsistently, double and triple products (reflecting the increased systolic BP and LVET) also increase, indicating that M\( \text{VO}_2 \) definitely increases during stooping.

Furthermore, the distinct increase in LA dimension during the knee-chest position suggests that the LV volume also increases during stooping. The increase in LV radius is in all probability less marked than the increase in LA size because wall stiffness of these two chambers differs. However, any increase in LV radius, no matter how modest, would also independently increase the intramyocardial tension that is required to produce a given ventricular pressure, in accordance with the Laplace formula, adding further to the factors that increase M\( \text{VO}_2 \).

**Afterload and Preload**

The immediacy of the rise in BP that is observed with stooping indicates that stooping acutely increases impedance to LV ejection (increased afterload) in a way that is similar in nature, if not in degree, to that associated with aortic clamping. Stooping leads to kinking of the femoral arteries, and even more significantly, to a large increase in intra-abdominal pressure. The aortic compression caused by the latter and the kinking of femoral arteries readily account for the increased afterload.

The effects of stooping on preload are more complex since stooping sequesters the blood contained in the leg veins but transiently increases venous return from the inferior vena cava and abdominal viscera by increasing intra-abdominal pressure. The ultimate effect on preload represents the sum-total of these two opposite actions. The increase in LA size (and in LV size by inference) could thus result from either increased venous inflow or from decreased LV emptying, or a combination of both. The prompt change in LA size within three to four beats from the supine to the knee-chest position strongly points to the immediate rise in peripheral resistance (afterload), as being the most important mechanism for the increased volume of the left sided chambers; increased venous return would require several seconds to manifest itself on the left side of the heart.
Study Limitations

Systolic time intervals were examined only in the patients with CAD. Likewise, no effort was made at blinding the examiners as to which group the subjects belonged. These two points could be construed as deficiencies of the study. However, the goal of this observational study was to determine whether stooping produces hemodynamic alterations in general. Thus, the group 2 subjects are not really a “control” group but rather represent one end of the spectrum of individuals tested, whereas group 1 represents the other end. Blinding was not considered to be necessary. Finally, since the patients with angina pectoris were those who were of primary interest to us, it was natural that these would be selected for the more tedious and complex part (systolic time interval measurements) of the study.

Only two patients experienced angina while performing the stooping maneuver in the laboratory, although several more had reported experiencing it at times. This is not surprising, since the threshold for angina in general is typically somewhat variable and was not reached during this study for a number of reasons, such as the quieting effect of a doctor in attendance or the short period of assumption of the stooping position. Furthermore, the patients were not selected specifically because of the history of pronus angina, since these patients are relatively uncommon.

It is possible that if more such patients had been included, the results might have been different. Nevertheless, the hemodynamic responses to stooping are so consistent across the entire spectrum of examined subjects, be they young and healthy, or elderly and suffering from typical angina, that it is fair to say that they represent a universal and reproducible physiologic response of the cardiovascular system to stooping. Clearly, this response represents a threat to the ischemic heart.

Conclusions

The following mechanism for pronus angina may thus be advanced. Stooping is accompanied by a sharp, mechanically induced, increase in systemic arterial resistance. The increased afterload (raised systemic vascular resistance and intramyocardial tension) and the prolongation in systolic ejection time lead to a significant elevation of $\text{MVO}_2$. Increased force of contraction, as reflected by the decrease in PEPI which was seen in 10 of 14 (71 percent) patients, may also contribute to further augmentation of $\text{MVO}_2$.

Stooping therefore induces far more pronounced hemodynamic effects than would superficially appear from the modest change in body position per se. The disproportionate increase in $\text{MVO}_2$ thus appears to be a very important trigger for pronus angina in patients with critical coronary artery lesions.
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World Association of Sarcoidosis and Other Granulomatous Disorders (WASOG)

The XIV International Conference on Sarcoidosis and Other Granulomatous Disorders, and the 23rd meeting of the WASOG will be held September 8-11 at the Ritz-Carlton Huntington Hotel, in Pasadena, California.

For information contact Dr. Om P. Sharma, Room 11-900, LAC/USC Medical Center, 1200 North State Street, Los Angeles 90033 (213:226-7923).