The Effect of the Valsalva Maneuver on the Circulation

III. The Influence of Heart Disease on the Expected Poststraining Overshoot

By E. Elisberg, M.D., E. Singian, M.D., G. Miller, M.D., and L. N. Katz, M.D.

The normal response following sustained straining, as in a Valsalva maneuver, is a rise in systemic arterial pressure and a reflex slowing of the heart rate. This response was found to be abolished in some patients with mitral stenosis, with organic heart disease, with pulmonary vascular disease (including kyphoscoliosis), or with congenital heart disease. It was abolished in all three instances of pericardial disease studied. The relationship of the presence or absence of a poststraining bradycardia to cardiovascular dynamics at rest and on exercise as determined by right heart catheterization is presented, and the suggestion is made that the Valsalva maneuver, when performed under electrocardiographic control, may be a simple, dynamic stress test of the circulation. This suggestion is waiting for further exploration.

Normally, an overshoot in systemic arterial blood pressure and an associated reflex bradycardia occur following cessation of sustained straining. The importance of the autonomic nervous system in its production was confirmed by us, and we have demonstrated that a significantly stenosed mitral valve will abolish it by preventing the rapid inflow of blood into the systemic circuit which occurs normally after cessation of straining.

In this report we have extended our observations to a large number of subjects, both normal and those with several types of heart and other diseases, and have established that this procedure may offer a simple stress test of the circulation.

Methods

The details of the cardiorespiratory assay prior to cardiac catheterization, the catheterization procedure and the type of steady state exercise performed have been described elsewhere. In addition to the subjects studied during cardiac catheterization, a number of others with normal cardiovascular systems performed the Valsalva maneuver while pressure recordings were made with an indwelling needle in the brachial artery.

A Valsalva maneuver was performed by the patient blowing directly into a mercury manometer while support was given to his cheeks. Intrapleural pressure under these conditions is approximately 7 mm. Hg lower than the recorded intraoral pressure. Intravascular and intracardiac pressures were measured by capacitance-type electromanometers recording on a direct writing oscillograph; a continuous electrocardiogram was simultaneously recorded.

For analysis, the Valsalva maneuver is customarily divided into four phases and a preceding control period. For convenience of presentation we will consider in this report only the control period before straining and the period several seconds after the cessation of straining (phase 4), during which an overshoot in blood pressure and reflex bradycardia normally occur.

Results

In the course of catheterization two subjects were found to have normal cardiovascular systems.† These, together with eight subjects

* Sanborn.
† In both of these subjects the pressures, both at rest and during exercise, were in the normal range in all vascular structures entered, including the pulmon ary artery. One of them had a low resting arteriovenous oxygen difference and a high resting cardiac index (6.1 L. per min. per MP). This changed little on exercise, a response associated with anxiety. * Stroke output index at rest (82 cc. per beat per
without cardiovascular disease on whom a Valsalva maneuver was done and an intra-brachial arterial blood pressure was recorded, constitute the normal group.

The other subjects were classified into several clinical groups and the average changes are shown in summary fashion in table 1.

1. Normal Subjects

The poststraining overshoot in systemic blood pressure in this group averaged 45 mm. Hg systolic (range, 25 to 90 mm. Hg) and 23 mm. Hg diastolic (range, 10 to 50 mm. Hg); the pulse pressure increase was 22 mm. Hg (range, 6 to 41 mm. Hg). There was an associated average decrease in pulse rate of 19 beats per minute in the poststraining period (in one case the drop was only 4 beats per minute, but this patient had a very slow initial rate; in another the drop was 40 beats per minute but this patient had a very slow initial rate; in another the drop was 40 beats per minute; in the others the fall was between 10 and 25 beats per minute). In the two cases tested with the catheter in the pulmonary artery, the average overshoot in pulmonary artery pressure was much less, being 11 mm. Hg systolic, 8 mm. Hg diastolic, and 3 mm. Hg pulse pressure.

These average values, obtained in normal subjects, served as our controls. Since most of this data was obtained through the brachial artery needle, and only two normal subjects were studied during cardiac catheterization, the above findings should be compared with cardiac indexes at rest and exercise in normal subjects as presented in the literature.

A recent survey of the literature[^6] showed that the low normal limit for the cardiac index at rest is 2.6 liters per minute per square meter of body surface. It increases by 50 to 75 per cent with the amount of exercise performed in these studies. Assuming normal pulse rates at rest and a normal response to exercise, the low normal limit for stroke index is approximately 40 cc. per beat per square meter of surface area at rest, rising approximately to at least 50 cc. per beat per square meter on exercise.

2. Rheumatic Mitral Stenosis

All patients in this group had acoustic evidence of mitral stenosis, but in many cases there was other valvular involvement. Of the 22 cases with rheumatic mitral stenosis studied, only six showed an overshoot in systemic blood pressure and a bradycardia following straining, our group 1. Five of these patients had a rise, within the normal range, of cardiac and stroke index on exercise; the exception was a patient with auricular fibrillation. Furthermore, pulmonary artery pressure rose only slightly on exercise in five patients; in the sixth, the pulmonary artery pressure, elevated at rest, rose significantly on exercise. In this last patient, the overshoot of pulmonary artery pressure after straining was quite marked, exceeding the rise with exercise, a unique occurrence in this series.

The remaining 16 patients failed to respond with a normal overshoot of systemic arterial pressure and a bradycardia following straining. These patients were divisible into two groups, viz., those in whom the cardiac index rose at least 1 liter per minute per square meter of surface area with the exercise performed (group 2), and those in which it did not (group 3). In group 2 it may be considered that a dynamically significant mitral stenosis was present but with an opening functionally adequate for the amount of exercise performed. In group 3 it may be considered that a more severe mitral stenosis was present, with an opening functionally inadequate to permit the unrestricted passage through the narrowed valve of the extra blood brought to the lungs during the mild exercise performed.

There were five cases in group 2. One of these patients had no systemic arterial overshoot or bradycardia but otherwise showed essentially normal cardiodynamics. The other four patients in this group developed a marked rise of pulmonary artery pressure on exercise, associated with an abnormally small rise in stroke index. In two, there was neither an overshoot nor bradycardia; in the other two, there was a slight to moderate overshoot in

[^6]: ELISBERG, SINGIAN, MILLER AND KATZ
Table 1.—Summary of values found in the various groups of patients studied. (Control pressures in brachial and pulmonary arteries are recorded in mm. Hg prior to straining, and during maximum overshoot period after straining. These values are compared with other pertinent catheterization findings. (See text))

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<td>Normals (10 cases)</td>
<td>115 66 78</td>
<td>160 89 59</td>
<td>18 7 29 15</td>
<td>20 8 14* 23* 11 17 2* 65</td>
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<td>Mitral stenosis§ group 1 (6 cases)</td>
<td>109 65 88</td>
<td>138 77 70</td>
<td>24 9 33 13</td>
<td>26 11 17 33 15 26 1 42</td>
<td>2.7 4.2 35 42</td>
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<td>Mitral stenosis§ group 2 (5 cases)</td>
<td>103 61 87</td>
<td>117 73 90</td>
<td>45 20 54 29</td>
<td>39 17 28 72 34 53 1 43</td>
<td>3.1 4.4 36 37</td>
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<td>Mitral stenosis§ group 3 (11 cases)</td>
<td>121 73 85</td>
<td>120 74 97</td>
<td>55 25 62 28</td>
<td>52 25 35 78 51 60 3 54</td>
<td>2.6 2.9 32 25</td>
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<td>Pericarditis (3 cases)</td>
<td>114 68 98</td>
<td>115 69 102</td>
<td>26 15 26 15</td>
<td>30 14 23 49 37 5 13 5 2 1 2.1 3.9 27 30*</td>
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<td>Pulmonary vasc. dis. (Hypertension (1 case))</td>
<td>208 118 72</td>
<td>232 132 68</td>
<td>19 9 25 9</td>
<td>20 7 8 43 28 23 4 60 2.1 3.7 31 41</td>
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<td>Pulmonary vasc. dis. (marked) (2 cases)</td>
<td>109 69 91</td>
<td>111 69 92</td>
<td>54 26 56 28</td>
<td>47 17 33 6 52 3.5 — 39 —</td>
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<td>Kyphoscoliosis—early (2 cases)</td>
<td>118 78 107</td>
<td>152 101 84</td>
<td>17 4 25 8</td>
<td>13 5 10 3 0 35 4.1 6.1↑ 45 49↓</td>
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<td>Kyphoscoliosis—severe (3 cases)</td>
<td>99* 63* 86</td>
<td>106* 68* 86</td>
<td>45* 20* 30* 29* 22* 48* — — —</td>
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<td>Organic heart dis. No failure (3 cases)</td>
<td>227 119 74</td>
<td>271 139 66</td>
<td>21 7 27 13</td>
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<td>Organic heart dis. History congen. fail. (6 cases)</td>
<td>170 97 81</td>
<td>173 98 89</td>
<td>42 18 40 17 43 18 29 — — — — 6 49 1.7 — 24 —</td>
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<td>Congen. heart dis.§ group 1 (6 cases)</td>
<td>115 64 84</td>
<td>140 76 71</td>
<td>25* 9 28* 10* 29 9 16 33* 15* 25* 3 56* 3.6 5.0* 44 54*</td>
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<td>Congen. heart dis.§ group 2 (3 cases)</td>
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B. A. = Brachial artery pressure  
P. A. = Pulmonary artery pressure  
S = Systolic pressure  
D = Diastolic pressure  
R = Heart rate, beats per minute  
E. D. P. = End diastolic pressure (mm. Hg).  
R. V. = Right ventricle  
I. O. P. = Intracava pressure (mm. Hg).  
C. I. = Cardiac index (liters per minute)

S. I. = Stroke index (cc. per minute)  
Re. = Rest  
Ex. = Exercise  
* 2 cases.  
† See text for literature summary.  
↑↑ 1 case.  
§ See text.  
|| 4 cases.  
¶ Output of the left ventricle, systemic + left to right shunt.
systemic pressure, but no bradycardia, following straining. Furthermore, in the last two cases the overshoot began from the eighth to the twelfth beat following cessation of straining instead of after the fourth beat, as occurs normally.

There were 11 cases in group 3. These patients had a high resting pulmonary artery pressure which increased markedly on exercise, they showed an abnormally small increase in cardiac index on exercise, and generally a fall instead of a rise in stroke index on exercise.

In comparing groups 1, 2, and 3, it was found that in general a progression existed. The clinical condition was worse in patients in group 2 than in those in group 1, and worse in patients in group 3 than in those in group 2. The number of valves involved increased, on the average, on comparing groups 1, 2, and 3, and the incidence of auricular fibrillation increased, the resting pulmonary artery pressure became higher, and the resting cardiac and stroke indexes decreased.

3. Pericarditis

Three proven cases of pericarditis were studied; one of these was constrictive in nature and the other two were associated with effusion. In none was there an overshoot in systemic blood pressure or a bradycardia in the poststraining period, despite normal increases in the cardiac index on exercise in the two cases in which this was measured. The stroke index rose moderately with the exercise performed. In all three cases the cardiac index and stroke index were low at rest.

Resting right ventricular and diastolic pressure was elevated in all three cases. Exercise caused a considerable rise in pulmonary artery pressure in one of the two cases.

4. Other Organic Heart Disease

In all, nine such patients were studied, three with arteriosclerotic heart disease, five with hypertensive cardiovascular disease, and one with aortic valve stenosis. In three of the patients a normal response was noted after straining.* Two of these had hypertensive cardiovascular disease without a history of failure. The third had arteriosclerotic heart disease and auricular fibrillation, well compensated at the time of study, but with a past history of mild heart failure. The cardiac indexes in these three patients were well above average, except in the third patient at the time of auricular fibrillation. However, in him, conversion to regular sinus rhythm considerably increased his output. This last patient was studied both before and after conversion of the auricular fibrillation to regular sinus rhythm. A definite systemic pressure overshoot occurred with both rhythms. During auricular fibrillation, however, the heart rate was inconstant in the poststraining period, but on the average, the rate was slightly slower than the control. After conversion to regular sinus rhythm the usual bradycardia was noted in the poststraining period.

The other six patients were found to have no bradycardia or overshoot in systemic pressure following straining. Pulmonary artery pressure after straining also showed no rise; it remained unchanged or fell. All but one of these patients had long histories of congestive heart failure and elevated resting pulmonary artery pressure at the time of study. The exception had a severe systemic hypertension with exertional dyspnea and a low resting cardiac index. The cardiac index in these patients was reduced (one just on the border-
line), and two of them exhibited elevated right ventricular and diastolic pressures. One of these patients was studied before and after conversion of an auricular fibrillation to regular sinus rhythm. Essentially no change was noted in his response to straining. No observations during exercise were made on these patients so that a comparison cannot be made between the exercise and Valsalva maneuver stresses.

5. Pulmonary Vascular Disease

Three patients with chronic pulmonary disease were studied. One of these, a patient with associated systemic hypertension, showed a poststraining overshoot in both the systemic and pulmonary arterial pressures as well as a bradycardia. The other two showed no systemic pressure overshoot or bradycardia in the post-straining period, and in the one in which pulmonary artery pressure was recorded a negligible rise in this pressure was found after straining. The assay of pulmonary pressure was difficult in this patient because there was considerable respiratory variation in pressure. In this case the end-diastolic pressure in the right ventricle was 10 mm. Hg.

In addition, five patients with kyphoscoliosis were studied. Two responded normally to straining. In these, resting and exercise cardiovascular measurements were also within normal limits. After straining, the other three showed no overshoot in systemic pressure or an insignificant one and no bradycardia. One of these patients had a normal resting pulmonary artery pressure which rose somewhat on exercise. The resting pulmonary artery pressures in the other two patients were elevated. One had a normal right ventricular end-diastolic pressure and developed a rise in pulmonary artery pressure and cardiac index on exercise; he had a normal rise in pulmonary artery pressure after straining, despite a lack of systemic pressure overshoot. The second of these patients had an end-diastolic pressure in the right ventricle of 7 mm. Hg and showed no overshoot in his pulmonary artery pressure as in his systemic pressure, after straining.

6. Congenital Heart Disease

Three of the patients were diagnosed as having idiopathic dilatation of the pulmonary artery. Two of these showed an overshoot of systemic pressure after straining with a marked bradycardia. The third had no bradycardia or overshoot. This patient had normal pressures in all chambers examined and a high resting cardiac index which declined on exercise, a response associated with anxiety.9

Two patients with interventricular septal defects and one patient with a tetralogy of Fallot responded to straining in a normal manner. One patient with a patent ductus arteriosus in congestive failure, as well as two patients with large interventricular septal defects failed to respond in a normal manner. Each of the latter two cases had a combined left ventricular cardiac index (systemic flow plus left to right shunt) of about 11 liters per minute per square meter of surface area.

In the patient with the overriding complex the brachial artery pressure was at control level two beats after straining instead of the expected four beats seen normally. This was also observed in a patient with an Eisenmenger complex observed after this study was completed.

Discussion

The use of straining as a functional test has been employed for some time. Sustained straining against 40 mm. Hg of pressure was suggested by Flack10 as a test of fitness for aviators. The changes were attributed by him to hypercapnia and hypoxia acting on medullary centers, and to alterations in "abdominal pooling." Hamilton and his colleagues8, 11 studied the effect of the Valsalva maneuver on intra-arterial pressures as well as on extra-vascular structures. Recently Mills12 investigated the pressure relationships in various body cavities during straining. McGuire and associates13 recently correlated straining with bedpan deaths. Lauson and co-workers14 separated the net from the recorded blood pressure changes during straining. The influence of the autonomic nervous system on the response to this maneuver2-4 has been analyzed.

Our own previous studies with the Valsalva maneuver suggest that: (1) During straining intraoral pressure is about 7 mm. higher than intrapleural pressure.7 (2) The autonomic nerv-
ous system must be intact for the occurrence of a normal response.  

(3) It is the blood which is dammed back in the systemic venous system during straining that rushes into a constricted systemic arterial system with the cessation of straining and produces the overshoot in arterial pressure and reflexly sets up the bradycardia.  

(4) A significant mitral stenosis prevents a normal response by preventing blood in the poststraining period from rapidly reaching the systemic arterial system.  

Our present study has permitted us to extend and amplify these observations and to correlate them more closely with previous observations of others.

Of prime consideration in the group of patients with mitral stenosis is the principle that flow depends upon cross-sectional area and difference of pressure on either side of an orifice. This is the basis for the formulation of Gorlin and Gorlin for mitral valve area, and the stenotic index of this department.

We have been able to divide the mitral stenosis patients into three groups. In group 1, all patients responded with increased systemic flow, a systemic pressure rise, and a bradycardia after straining. Since the hemodynamics at rest as well as on exercise in five of these patients were also normal, it is unlikely that the mitral valve offered any significant hindrance to flow of blood. In the sixth case, the cardiac index increased normally on exercise, but unlike the others in this group, was associated with a marked rise of pulmonary artery pressure. Therefore this patient, in spite of the stenotic valve, was able to increase peripheral flow apparently as a result of the increase in pressure in back of the valve. The marked pulmonary pressure overshoot after straining in this patient was also apparently able to increase systemic flow sufficiently to produce an overshoot in systemic arterial pressure and a reflex bradycardia. This marked overshoot in pulmonary artery pressure following straining was unique in this series.

In group 2, the elevation of the pulmonary artery pressure found in all the patients on exercise was also sufficient to increase cardiac output through the stenotic mitral valve. But this was not true following straining, where the pulmonary artery pressure overshoot was in the normal range and insufficient to cause the usual increase in flow past the valve. In a few cases a moderate systemic overshoot did develop. However, the obstruction offered by the mitral valve caused the systemic pressure to increase more gradually and later than normal. This type of overshoot response was insufficient to stimulate the pressor receptors to cause a reflex bradycardia.

In group 3, the mitral stenosis was so marked that even the marked rise in pressure in the pulmonary artery on exercise was incapable of increasing the flow past the valve. It likewise prevented the overshoot in pulmonary artery pressure, which was in the high normal range, from leading to a systemic arterial overshoot or bradycardia after straining.

The similarity in the amount of pulmonary artery pressure rise after straining in all three groups of mitral stenosis (with the exception of one patient), regardless of resting pressure, was reported previously. This was attributed to the large capacity of the pulmonary vascular bed, emptied during straining, permitting it to readily accommodate the blood after straining without an undue pressure increase. It should be borne in mind that the elevation of pulmonary artery pressure is the result of a "back pressure" dependent upon resistance offered at the mitral valve, and transmitted backward from the left auricle to the pulmonary capillaries and pulmonary artery.  

With the exception of one case with auricular fibrillation, stroke index on exercise increased considerably in all cases of group 1, changed little in group 2, and fell in group 3. This is evidence that with increasing severity of mitral stenosis, ventricular filling becomes more and more unable to keep up with the increasing heart rate occurring during exercise.

None of the patients with pericardial disease showed an overshoot in systemic pressure or bradycardia following straining despite definite increases in cardiac and stroke indices on exercise. Apparently the limited cardiac filling following pericardial constriction or effusion prevents sufficient blood from being pumped into the periphery rapidly enough to produce an overshoot in systemic pressure after straining even though mild exercise showed no devia-
tion from the normal response. Obviously straining is a much more abrupt and severe strain than the exercise performed by these patients. The explanation suggested by Hamilton and associates\textsuperscript{11} for failure of systemic overshoot in patients with congestive heart failure (namely that the intrapleural pressure on straining did not rise as high as the elevated venous pressure, and thus did not prevent blood from flowing into the thorax during the Valsalva maneuver) may be considered to apply in the pericarditic patients. However, against this latter explanation is the fact that the intrapleural pressure (as judged by the intraoral pressure) was far in excess of the right ventricular end diastolic pressure. The development of an "empty" pulse\textsuperscript{8} during straining and a marked fall in pulse pressure immediately after straining is also opposed to this last explanation and favors the first one. Moreover, in one case in which it was measured, intravenous pressure rose markedly, but slowly, during straining, indicating that the rise in intrapleural pressure is an effective barrier to venous inflow in these patients as it is in normal subjects. For these reasons we feel that limitation of the stroke volume rather than continued flow during straining explains the lack of systemic pressure overshoot.

Since the same results may be seen in pericardial disease as in the enlarged hearts or in congestive failure, the Valsalva maneuver cannot be used to help in the differentiation between these conditions.

In patients with organic heart disease, but with no history of congestive failure, the response to straining is normal. However, when congestive failure is, or has been, conspicuous there is no systemic overshoot or bradycardia. This appears to depend on the inability of the impaired heart to increase its output when exposed to an increased input load. The alternate explanation offered for the failure of systemic pressure overshoot in these patients by Hamilton and co-workers,\textsuperscript{11} mentioned above, is not considered as valid. We assume that in these patients the elevation in venous pressure during straining is not greater than the rise in intrapleural pressure and therefore that flow into the thorax declines during straining. Furthermore, since four of the six cases studied had a normal resting right ventricular end diastolic pressure, it would appear that elevated systemic venous pressure was not a factor in the failure of these patients to develop a systemic overshoot in the poststraining period. While a few of the patients in this group showed only a slight fall in pulse pressure during straining, most of them had a pronounced fall and/or an "empty" pulse during this period.

None of the patients with organic heart disease without a systemic pressure overshoot had a definite pulmonary pressure overshoot in the period following straining. In two of these patients right ventricular end diastolic pressure was elevated, evidence of failure of this chamber, so that a rise in pulmonary pressure would not be expected after straining. But even in those patients in whom the right ventricular end diastolic pressure was normal, no pulmonary artery pressure overshoot was present after straining. This would suggest that while the right heart was able to handle the reduced cardiac resting output in these patients without rise in venous pressure, it was not sufficiently competent to increase its output in response to the increased venous inflow following straining to raise its output enough to produce a rise in pulmonary artery pressure.

Slonim and associates\textsuperscript{18} found a marked overshoot in the pulmonary artery pressure in the poststraining period in a group of patients with pulmonary vascular disease. Our data in a group of three patients with pulmonary vascular disease and five kyphoscoliotics were more variable. One of the former and two of the latter we found had a normal systemic and pulmonary artery pressure overshoot after straining. The one patient with pulmonary vascular disease who showed the normal overshoot after straining also showed an increase in his cardiac index on exercise, but this was associated with, and apparently depended upon, a rise in pulmonary artery pressure, as occurred in some of the mitral stenosis cases.

Two patients with pulmonary vascular disease and three with kyphoscoliosis failed to develop a systemic pressure overshoot after straining. Two of these (one from each group)
had an elevated right ventricular end diastolic pressure at rest and an insignificant or no pulmonary artery pressure overshoot after straining. The failing right ventricle in these cases was probably responsible for the abnormal response to straining as it was in the patients with congestive heart failure. The other three patients had normal right ventricular end diastolic pressures and in the only one of these in which it was measured, the pulmonary artery pressure overshoot after straining was in the normal range. This pulmonary pressure response in the absence of a systemic pressure overshoot reflected the inability of the reduced pulmonary vascular bed with its increased resistance to permit the extra blood, accumulated after straining and poorly accommodated within it, to pass through. All three patients with a normal right ventricular end diastolic pressure at rest and absent systemic pressure overshoot after straining showed a marked rise in pulmonary artery pressure on exercise. Because of this ability of the heart to increase its output on exercise consequent to the considerable elevation of the pulmonary artery pressure, it would appear that the reduced pulmonary vascular capacity and its greater resistance to sudden changes in its flow was often solely responsible for the lack of systemic pressure overshoot after straining.

We wish to express a word of caution in the study of these patients. Care must be exerted in interpreting a slightly or moderately elevated right ventricular end diastolic pressure as an indication of right heart failure, since in emphysematous patients the associated elevated intrathoracic pressure is transmitted to the right ventricle and so raises the end diastolic pressure. Only when it can be shown that net end diastolic pressure, that is, the actual pressure minus intrapleural pressure, is elevated can it be concluded that right heart failure exists.

Furthermore, our analysis shows that the period during which the pressure overshoot is exhibited after straining should be critically examined. This is a period of deep and forced breathing after the apnea of straining. Respiratory blood pressure variations in these patients are likely to be marked and should be carefully evaluated. Furthermore, trapping of air during this period of rapid breathing might raise intrathoracic pressure which in turn could be mechanically transmitted to the pulmonary artery and aorta. This would produce an elevation of the measured pressure without any rise in the net pressure.

A varied group of patients with congenital heart disease had an overshoot in systemic blood pressure and an associated bradycardia after straining. This group included a case of tetralogy of Fallot where, because of pulmonic stenosis, the overshoot probably came directly from the right ventricle into the overriding aorta. One patient with a patent ductus arteriosus, in congestive failure, one with an interventricular septal defect and an overriding of the aorta, and one with a large interventricular septal defect failed to show the expected overshoot in systemic pressure. In the last two cases, the total outflow of the left ventricle (including the shunt) was large and was apparently approaching the maximum output of this chamber, so that the left heart was unable to increase its output further even after straining. Each of these cases had an elevated pulmonary artery pressure, either accompanying the large pulmonary blood flow or as an evidence of left heart failure.

One patient in the group with an idiopathic dilatation of the pulmonary artery and with normal resting and exercise pulmonary artery pressure, failed to respond to straining in a normal fashion. The cardiac index dropped on exercise in this patient, an event usually associated with anxiety. Many patients are quite anxious at the time of catheterization, yet this lack of normal response is quite rare. What might have been the exact effect of the anxiety experienced by this patient upon the autonomic nervous system cannot be determined from the present studies. However, the necessity for the integrity of this system in the production of normal response has been shown and marked changes in the autonomic nervous system with anxiety might possibly be a factor accounting for the rare failure of a healthy subject who does not respond normally to straining.
The systemic pressure overshoot may occur a few beats sooner in congenital anomalies associated with overriding of the aorta as blood enters the aorta directly from the right ventricle without necessarily traversing the lungs.

The test and results described in this report have been performed during cardiac catheterization in most of the patients. In the remainder it was accomplished with a needle in place in the brachial artery. The information derived should be of interest to catheterization teams; the test is simple to carry out and is now routinely employed by us during catheterizations.

The test may also be used clinically with the electrocardiograph to follow the changes in heart rate since the reflex bradycardia after straining is usually as significant as the systemic blood pressure overshoot. The reasons for this are: (1) the poststraining bradycardia has never been found by us in the absence of a systemic blood pressure overshoot; and (2) bradycardia does not occur after straining when the systemic pressure overshoot is of a mild degree, or delayed, and minimal in extent. Patients exhibiting this latter overshoot response are abnormal and have significant lesions as illustrated in some of our patients with mitral stenosis in group 2. The reflex bradycardia will occur with sinus tachycardia; it is more difficult to evaluate in auricular fibrillation. It is well seen when the resting heart rate is as low as 80 beats per minute; but may be difficult to detect if the control rate is slow, below 60. The test can be performed by having the patient blow directly into an ordinary sphygmomanometer detached from its cuff, or simply by “bearing down” against a closed glottis for approximately 30 seconds. The heart rate is observed before straining, throughout the maneuver, and in the period after straining.

With few exceptions, as noted above, the definite development of a bradycardia in the poststraining period implies that: (1) the heart can respond to an increased input by increasing its output; (2) the autonomic nervous system is intact functionally, and (3) there is no significant obstruction to flow at any valve orifice or within the pulmonary circuit.

The failure of a bradycardia to develop after straining in most instances suggests the presence of obstructing lesions, inability of the heart to increase its output, or an autonomic nervous system impairment.

Of course, there are infrequent exceptions. As a laboratory procedure the Valsalva maneuver makes available to the clinician in his office a rather good physiological and dynamic stress with which to determine circulatory response. Further studies are needed to define the limits and errors of this test.

**Summary**

A study has been made of the circulatory responses in the poststraining period to the Valsalva maneuver. Normal patients and patients with a variety of heart diseases have been studied. A normal response is an overshoot in systemic artery pressure and a bradycardia in the poststraining period. Normally there is a small overshoot in pulmonary artery pressure. It has been shown that this normal response is abolished in patients with pericarditis; with organic heart disease when congestive heart failure has been conspicuous in the past, or is present at the time of testing; in some patients with pulmonary vascular disease; and with some types of congenital heart disease. Pulmonary artery pressure does not rise after straining where there has been failure of the right ventricle.

By observing the development of the bradycardia in the poststraining period, with the use of an electrocardiograph, the clinician has a simple and useful tool for appraising the integrity of the cardiovascular system, since the presence of bradycardia suggests that overshoot in the systemic arterial pressure occurs in the poststraining period and the absence of bradycardia, when the autonomic nervous system is intact, suggests that an overshoot of systemic arterial pressure does not occur. Bradycardia in the poststraining period of the Valsalva maneuver, therefore, is an index of the response of a patient to a simply executed stress test.

**Acknowledgment**

We are indebted to the physicians in charge of these patients for permission to study them and to
the members of the catheterization team for help in obtaining some of the data.

**Sumario Español**

La repuesta normal a un esfuerzo sostenido, como en la maniobra de Valsalva, es un incremento en la presión arterial sistémica y una desaceleración cardiaca refleja. Esta repuesta se encontró abolida en algunos pacientes con estenosis mitral, con enfermedad cardiaca orgánica, con enfermedad pulmonar vascular (incluyendo quíbo-escoliosis), o con enfermedad del corazón congénita. Se encontró abolida en tres casos de enfermedad del pericardio estudiados. La relación de la presencia o ausencia de bradicardia post-esfuerzo a la dinámica cardiovascular durante reposo y ejercicio según se ha determinado mediante catarización del lado derecho del corazón se demuestra y la sugerencia se hace de que la maniobra de Valsalva cuando se ejecuta bajo control electrocardiográfico puede ser una prueba sencilla de esfuerzo dinámico de la circulación. Esta sugerencia está en espera de más exploración.

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