The Hemodynamics of the University of Cape Town Aortic Prosthetic Valve


Several reports have appeared in the literature indicating that ball valve aortic prostheses usually produce some residual obstruction.1-4 It has often been assumed that the residual pressure differences across these valves is due to the small orifice area of the prosthetic valve ring compared to that of a normal aortic valve.

In contrast to this concept, our results indicate that even when the orifice area of a ball valve approaches normal values, obstruction may exist and that for all valve sizes the resistance to flow is greater than might be expected from a knowledge of its orifice area.

We would also like to emphasize the not infrequent occurrence of an obscure form of myocardial insufficiency which is in many cases responsible for the abnormal exercise response noted in these patients.

Group Studied

To date 28 patients with severe aortic valve disease have been operated on. Eleven (cases 1 to 11) of these have now survived 1 year following the operation and have been restudied to assess their hemodynamic state and capacity.

All patients had grade 3 or 4 clinical disability preoperatively. Ten patients had rheumatic or congenital valve disease, seven with combined stenosis and incompetence, two with pure incompetence and one with pure stenosis. The remaining patient (case 6) suffered from syphilitic aortic incompetence. In four patients the mitral valve was mildly involved and did not require surgical interference, but in two the mitral lesion was severe so that both mitral and aortic valves were replaced (cases 9 and 10).

The aortic valve used was the U.C.T. aortic valve previously described5 and illustrated in figure 1. This valve is available in six sizes (table 1). The size 3 and size 4 valves were each used in five patients and the size 5 valve in one patient.

The U.C.T. mitral valve6,7 was used in the two patients who required double valve replacement.

Methods

Preoperatively, all patients underwent right and left heart catheterization. Mitral valve gradients were usually assessed by simultaneous recordings of wedged pulmonary artery pressure and left ventricular diastolic pressure.

The aortic gradient was calculated from a withdrawal tracing across the aortic valve; the degree of mitral and aortic insufficiency was assessed from left ventricular and aortic angiography.

Postoperatively the right heart pressures were recorded by catheterization of the median basilic vein; the ascending aorta was reached retrogradely from the femoral or brachial artery. In seven cases the left ventricle was entered via the transeptal route from the femoral vein.

In the two cases with prosthetic mitral and aortic valves direct percutaneous puncture of the left ventricle was employed. In one of these a fine polyethylene catheter was advanced through the needle which was then withdrawn to permit

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exercise studies. The manometer zeros were adjusted to midchest level. Cardiac output was calculated from dye-dilution curves using indocyanine green (Cardio-Green) and the Waters densitometer.

Nine patients were exercised in the supine position on an Elema Schonander bicycle ergometer at a load of 150 to 300 kg-m/min for 5 minutes, and the aortic gradient, mean pulmonary artery pressure and cardiac output were measured at this time. In patient 10, the left ventricular pressure and aortic gradient could not be recorded during exercise because of a percutaneous direct left ventricular puncture with a needle only.

In case 8, the left ventricle could not be entered transseptally and in case 1 the mean pulmonary artery pressure was not recorded during exercise. Aortic cineangiography was performed to test the degree of reflux into the left ventricle in all cases.

Results

With one exception (case 11) all patients have shown marked clinical improvement, having reverted to a near asymptomatic state or a state of mild residual disability. Unlike the U.C.T. mitral valve which has resulted in a 60% incidence of cerebral emboli only two patients (one in this group) have had mild transient hemiparesis or monoparesis. None have been on anticoagulant therapy.

The hemodynamic data are given in table 2. A comparison of preoperative and postoperative findings at rest show that, postoperatively, cardiac indices were within the normal range in all except case 11, whereas preoperatively they were abnormally low in three cases and abnormally high in two.

Most patients had a satisfactory reduction in pulmonary artery and left ventricular diastolic pressure when this had been elevated before surgery.

Figure 1

The prosthetic aortic valve.

Figure 2

Simultaneously recorded postoperative aortic and left ventricular pressures from case 1 at rest and during exercise. This patient had the Ao 4 valve inserted. At rest the mean aortic gradient was 23 mm Hg. During exercise the mean gradient increases to 57 mm Hg.
At rest, the pulmonary artery pressure was normal in seven cases and mildly elevated to mean values of 23, 25, 26, and 35 mm Hg in the remaining four. Postoperatively all patients had residual aortic valve gradients (fig. 2). The mean aortic gradient varied between 8 and 38 with a mean of 24 mm Hg, and the effective valve area was calculated by the Gorlin formula 0.76 to 1.27, with a mean of 1.085 cm².

At rest, the mean effective valve area for the size 3 valve was 0.90, for the size 4, 1.08 and for the single size 5 valve, 1.27. Although the mean figures seem to show a slight difference in the effective orifice area in relation to the size of the valve used, there is considerable overlap in individual cases (fig. 7). Aortic cineangiography showed that the valve was completely competent in seven cases while in three small amounts of contrast medium which gathered beneath the valve and in the aortic sinuses during systole were pushed back into the left ventricle as the mobile portion of the valve descended at the onset of diastole. In one case definite but mild aortic insufficiency was present, but it was difficult to determine whether this was due to improper seating of the mobile portion in the ring, or whether it occurred between the valve ring and the aortic wall. The preoperative and postoperative data are diagramatically shown in figures 3 and 4.

During exercise with a workload of 150 to 300 kg-m/min for 5 minutes the aortic gradient increased to a mean of 35.5 mm Hg (range 8-65 mm Hg). Cardiac index increased to a mean of 4.51 L/min/m² (range 1.53-6.85) and the effective valve area decreased slightly to 0.98 cm² (range 0.78-1.18). The mean effective valve area for Ao 3 was 0.88; for Ao 4, 1.10 and for Ao 5, 1.15 cm². In the

Figure 3
This diagram shows the changes in cardiac index on the left and mean pulmonary artery pressure (PAP) on the right that occurred after aortic valve replacement. The preoperative valves are shown by the open circles (the solid circles indicate the patients with mitral and aortic replacements) and the postoperative values by the crosses. The values enclosed by the dark horizontal lines lie within the normal range for our laboratory.
### Table 2

**Hemodynamic Data Preoperatively and Postoperatively**

<table>
<thead>
<tr>
<th>Name</th>
<th>(Case no), age &amp; sex</th>
<th>State</th>
<th>BSA</th>
<th>HR</th>
<th>Rhythm</th>
<th>PAP</th>
<th>Mean</th>
<th>x = Wedge LAP</th>
<th>Mean</th>
<th>C.I.</th>
</tr>
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<tbody>
<tr>
<td>M.S.</td>
<td>Preop. (1)</td>
<td>Rest</td>
<td>1.43</td>
<td>66</td>
<td>S.R.</td>
<td>18/8</td>
<td>12</td>
<td>13/6</td>
<td>9</td>
<td>3.00</td>
</tr>
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<td>Postop. 37 F</td>
<td>Rest</td>
<td>1.40</td>
<td>75</td>
<td>S.R.</td>
<td>16/5</td>
<td>7</td>
<td>8.5/1.5</td>
<td>5</td>
<td>3.00</td>
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<tr>
<td></td>
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<td>S.R.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M.H.</td>
<td>Preop. (2)</td>
<td>Rest</td>
<td>1.82</td>
<td>96</td>
<td>S.R.</td>
<td>26/8</td>
<td>17</td>
<td>11/7</td>
<td>9</td>
<td>1.82</td>
</tr>
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<td>66</td>
<td>S.R.</td>
<td>16/5</td>
<td>9</td>
<td>8.5/1.5</td>
<td>3</td>
<td>2.80</td>
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<td>J.K.</td>
<td>Preop. (3)</td>
<td>Rest</td>
<td>1.50</td>
<td>110</td>
<td>S.R.</td>
<td>100/41</td>
<td>62</td>
<td>54/27</td>
<td>33</td>
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<td>1.57</td>
<td>87</td>
<td>S.R.</td>
<td>38/17</td>
<td>23</td>
<td>10/3</td>
<td>6</td>
<td>3.18</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Exercise, 300 kg-m for 5 min</td>
<td>1.57</td>
<td>143</td>
<td>S.R.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D.S.</td>
<td>Preop. (4)</td>
<td>Rest</td>
<td>1.46</td>
<td>75</td>
<td>S.R.</td>
<td>47/23</td>
<td>35</td>
<td>37/23</td>
<td>28</td>
<td>3.80</td>
</tr>
<tr>
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<td>Postop. 23 F</td>
<td>Rest</td>
<td>1.55</td>
<td>60</td>
<td>S.R.</td>
<td>17/6</td>
<td>11</td>
<td>6.5/2.5</td>
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<td>105</td>
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<td></td>
<td></td>
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<td>Preop. (5)</td>
<td>Rest</td>
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<td>S.R.</td>
<td>39/16</td>
<td>28</td>
<td>x 27/13</td>
<td>22</td>
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<td>63</td>
<td>S.R.</td>
<td>30/10</td>
<td>19</td>
<td>12/7</td>
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<tr>
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<td>Exercise, 300 kg-m for 5 min</td>
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<td>143</td>
<td>S.R.</td>
<td></td>
<td></td>
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<td>Rest</td>
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<td>56</td>
<td>S.R.</td>
<td>35/11</td>
<td>19</td>
<td>11/5</td>
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<td>S.R.</td>
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<td>Rest</td>
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<td>78</td>
<td>S.R.</td>
<td>31/12</td>
<td>19</td>
<td>x 17/9</td>
<td>13</td>
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<td>72</td>
<td>S.R.</td>
<td>19/8</td>
<td>13</td>
<td>x 9/4</td>
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<td>Preop. (8)</td>
<td>Rest</td>
<td>1.52</td>
<td>67</td>
<td>S.R.</td>
<td>30/9</td>
<td>19</td>
<td>x 14/8</td>
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<td>Rest</td>
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<td>58</td>
<td>S.R.</td>
<td>23/9</td>
<td>15</td>
<td>9/5</td>
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<td>92</td>
<td>S.R.</td>
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<td>J.vR.</td>
<td>Preop. (9)</td>
<td>Rest</td>
<td>1.44</td>
<td>138</td>
<td>Atrial fibrillation</td>
<td>70/34</td>
<td>50</td>
<td>33/11</td>
<td>18</td>
<td>1.65</td>
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<td>Rest</td>
<td>1.49</td>
<td>102</td>
<td>S.R.</td>
<td>32/18</td>
<td>25</td>
<td>17/7</td>
<td>12</td>
<td>2.80</td>
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<tr>
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<td></td>
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<td>50</td>
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<td>Rest</td>
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<td>132</td>
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<td>113/60</td>
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<td>56/42</td>
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<td>98</td>
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<td>35/15</td>
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<td>3.40</td>
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<td>155</td>
<td>S.R.</td>
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<tr>
<td>A.A.</td>
<td>Preop. (11)</td>
<td>Rest</td>
<td>1.92</td>
<td>93</td>
<td>S.R.</td>
<td>47/24</td>
<td>32</td>
<td>32/20</td>
<td>25</td>
<td>1.70</td>
</tr>
<tr>
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<td>Postop. 54 M</td>
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<td>93</td>
<td>S.R.</td>
<td>46/23</td>
<td>35</td>
<td>35/23</td>
<td>29</td>
<td>1.20</td>
</tr>
<tr>
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<td></td>
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<td>100</td>
<td>S.R.</td>
<td>70/42</td>
<td>58</td>
<td>60/30</td>
<td>45</td>
<td>1.53</td>
</tr>
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</table>

**Abbreviations:** BSA, body surface area in m²; HR, heart rate in beats per minute; PAP, pulmonary artery pressure in mm Hg; LAP, left atrial pressure in mm Hg; Wedge, wedged pulmonary artery pressure; CI, cardiac index; PVR, pulmonary vascular resistance in simple units obtained by dividing the transpulmonary gradient by flow; LVP, left ventricular pressure, mm Hg; AP, aortic pressure; SEP, systolic ejection period as duration of systole in seconds; AI, aortic incompetence roughly classified into 4 grades from aortograms; MI, mitral incompetence; MS, mitral stenosis; TS, tricuspid stenosis; kg-m, kilogram-meters per minute.
eight cases in which it was measured the mean pulmonary artery pressure rose to abnormally high levels: 40 to 70 mm Hg in seven cases, and to 27 in the remaining case.

The abnormal response to exercise could be ascribed to mitral valve obstruction in four cases. Patients 5 and 8 both had slight mitral valve stenosis and patients 9 and 10 both had mitral valve prostheses which were also mildly stenotic.8

Circulation, Volume XXXIII, April 1966
In the remaining three patients the cause was abnormal elevation of the left ventricular end-diastolic pressure during exercise. One of these (case 6) had moderate systemic hypertension and one (case 3) had complete atheromatous occlusion of the right coronary artery. In case 11, mild mitral and aortic insufficiency were present but did not seem severe enough to account for the left ventricular failure. The data at rest and during exercise are summarized in figures 5 and 6.

**Discussion**

It appears from these studies that the prosthetic aortic valve functions as a competent but moderately stenotic valve under conditions of rest and moderate exercise.

While not ideal, the residual obstruction should be well within the capacity of a normal left ventricular myocardium. However, it might well be that in patients with a damaged or failed myocardium, due to severe longstanding aortic valve disease, the residual obstruction could be a significant load.

It is clear that there is a marked discrepancy between the actual orifice area of the valve ring inserted and the effective valve area calculated from the Gorlin formula (fig. 7).

There are probably serious objections to the use of the Gorlin formula in calculating orifice areas with any accuracy. In particular, the mathematical errors involved in assuming a "constant" in the formula may provide an error of between 20 and 40%.9

The presence of turbulent and pulsatile flow which the hydraulic formula does not take into account probably also invalidates it as far as an exact hemodynamic measurement is

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**Figure 4**

The mean aortic gradient on the left and the degree of aortic incompetence on the right before and after aortic valve replacement. The preoperative and the postoperative values are indicated by the circles and crosses, respectively. The solid circles indicate the two patients with both aortic and mitral replacements. Postoperative gradients are present in all cases. Mild aortic incompetence was present in only one. Trivial amounts of reflux during normal closure of the ball valve were seen in four cases.

*Circulation, Volume XXXIII, April 1966*
The changes in mean aortic gradient and mean pulmonary artery pressure that occurred during exercise. Abnormal elevation of the latter occurred in seven of eight cases where this pressure was measured.

The changes in mean left atrial pressure and left ventricular diastolic pressure during exercise. In the eight patients where the latter were measured, four showed a normal response and in four the diastolic pressure rose to abnormally high levels.
concerned. Nevertheless the finding that the calculated effective orifice is only 35 to 40% of the actual orifice area suggests that some other cause of obstruction to left ventricular ejection is present in these cases. Morrow et al.\textsuperscript{10} have suggested that the residual gradient might be due to secondary hypertrophic subaortic stenosis. However in case 9, a thin polythene catheter, that passed from the left ventricle across the aortic prosthesis and was subsequently withdrawn, localized the gradient to the level of the valve, and in three cases left ventricular cineangiograms showed no evidence of secondary hypertrophic subaortic stenosis.

Another possibility is that during systole the mobile portion of the valve moves up into a narrower portion of the ascending aorta and that the area between the aorta and the ball might be less than the area of the valve ring. This seems unlikely as the aorta immediately above the valve ring is usually much wider than the ring itself.

To test this, measurements were made of the area between the aortic wall and the ball during systole from the cineangiograms. Measurements of the fixed part of the valve were used to obtain the correction factor.

The results are given in figure 8. In most instances the area between the ball and the aorta during systole exceeded the area of the

![Diagram](image)

**Figure 7**

The actual orifice of the prosthetic valve on the abscissa has been plotted against the effective orifice areas as calculated by the Gorlin formula in 10 cases. The Ao 3 valve with orifice area of 2.25 cm\(^2\) gives a mean calculated area in five cases of 0.90 cm\(^2\). The Ao 4 with orifice area of 2.83 gives a mean of 1.07 cm\(^2\) in four cases and the single Ao 5 valve with an orifice area of 3.46 gave an effective area of 1.27. Although there would appear to be a tendency for the larger valves to have slightly larger orifice areas the difference is very little, suggesting that some factor other than the actual orifice area is responsible for the residual obstruction.
AORTIC PROSTHETIC VALVE

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**Figure 8**

*The residual area between the mobile portion of the prosthesis and the wall of the aorta during systole called the "lateral wall area" on the abscissa has been plotted against the calculated effective orifice area on the ordinate. There is clearly no relationship between these areas, and even in the two instances in which the lateral wall area was less than the actual orifice area, it still exceeded the calculated effective orifice area for these two cases.*

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valve ring. In only two patients (cases 3 and 11) with very narrow aortas and very little dilatation at the level of the aortic sinuses was the residual area less than the actual area of the ring, but, even here, the residual area was twice that calculated by the Gorlin formula in these cases.

It is quite clear from a consideration of hydraulic principles that the mobile portion of the prosthetic valve must have both inertial and viscous forces operating on it during ventricular ejection and that some loss of pressure head across the valve is inevitable and implicit in the ball valve mechanism.

We believe that this is the major cause of the residual gradient observed in these cases and that it probably accounts for the discrepancy between the area of the actual valve ring and the effective orifice area as calculated by the Gorlin formula.

As regards the hemodynamic response to exercise, it is a little disturbing to find abnormal elevation of pulmonary artery pressures and left ventricular diastolic pressures in three out of five cases without associated mitral valve disease and in one case with this complication. The poorly contracting left ventricles with high end-diastolic pressures seen in four cases were similar to those encountered in some of our cases following mitral valve replacement. It is uncertain to what extent the absent right coronary artery and systemic hypertension were responsible in two (cases 3 and 6) of these cases. In case 9, with both mitral and aortic prostheses, the onset of atrial flutter at a rate of 220 per minute during exercise may have been responsible for the abnormal rise in left ventricular end-diastolic pressure. In case 11, no obvious cause for the left ventricular failure could be found.

Two patients have actually complained of chest pain on exertion suggestive of angina pectoris, but no major proximal coronary artery obstruction was present in either of
these postoperatively when studied by selective coronary angiography.

We have speculated about intimal sclerosis of the muscular coronary arteries as described by Barratt-Boyes, altered flow patterns past the coronary ostia caused by the mobile portion of the valve, irreversible myocardial damage, the result of long-standing aortic stenosis, and incompetence, coronary emboli, or myocardial damage sustained during cardiac bypass and coronary perfusion, as possible etiological factors.

The hemodynamic results are very similar to others previously reported following the insertion of the Starr-Edwards aortic prosthesis and even the hinged leaflet aortic valve.

The smaller gradients obtained in some of the reports are, we feel, due to the use of a peripheral arterial pressure rather than a central aortic pressure which in many cases may be grossly misleading due to the augmentation of the peripheral pressure pulse.

Conclusions and Summary

The insertion of the University of Cape Town prosthetic aortic valve into patients severely disabled by aortic valve disease has been associated with marked clinical improvement. Furthermore, there has been a gratifyingly small incidence of embolization even though anticoagulants have not been used.

This report concerns the hemodynamic studies performed at rest and during exercise on 11 patients who have survived a year or more following surgery.

The valve is moderately stenotic and without significant incompetence. Definite regurgitation occurred in one case. The effective orifice area as calculated by the Gorlin formula varies between 0.75 and 1.27 cm² with a tendency for the smaller sized valves to be associated with the smaller calculated effective areas. The discrepancy between the actual orifice area and the calculated effective area appears to be due to the additional obstruction offered by the mobile portion of the valve to left ventricular ejection and is unavoidable in a ball valve mechanism of this type.

The abnormal rise in pulmonary artery pressure after exercise was due to associated mitral valve disease in four patients and was accompanied by an abnormal rise in left ventricular end-diastolic pressure in one of these cases. In three patients without mitral valve disease, the abnormal exercise response was associated with an undue rise in left ventricular diastolic pressures. In only one patient was the exercise response near normal.

The poorly functioning left ventricle was similar to that found in some of our cases following mitral valve replacement and possible etiological factors are discussed.

Acknowledgments

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References


Graves on Education of the Physician (1848)

There is no system capable of communicating information to the indolent; every man must depend chiefly on his own assiduity, and all the teacher can do is to facilitate the means of acquiring knowledge, and afford an example of punctuality and attention. I would seriously recommend every one who undertakes the management of cases, to set out with a fixed determination to persevere throughout the whole session. Few things give me more concern than to find young men, who have commenced with ardour, becoming by degrees less and less industrious, until their hospital attendance degenerates into an irksome task, imperfectly performed, and at last wholly neglected. One of the most valuable things which the student can acquire, is a habit of daily diligence. The knowledge requisite for the efficient discharge of our professional duties is not to be acquired by sudden starts of intense application, or by the overwrought strivings of desultory exertion; it demands a daily and hourly attention, a steady, constant, and accurate course of observation, continued uninterruptedly for years.—ROBERT J. GRAVES: Clinical Lectures on the Practice of Medicine, ed. 2, vol. 1. Dublin, Fannin and Co., 1848, p. 14.
The Hemodynamics of the University of Cape Town Aortic Prosthetic Valve

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