An experimental study of intra aortic balloon pumping within the intact human aorta

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Abstract

Objective: Intra-aortic balloon pumping is a therapeutic technique which carries a significant morbidity related to the interaction between the balloon catheter and the aorta. The aim of this study was to visualise directly the dynamic action of the balloon catheter within the cadaveric human aorta in an artificial circulation. Methods: An artificial circulation was constructed using of PVC tubing, a filter and a roller pump. A series of five intact cadaveric human aortas were then individually studied by placing each in series within the circuit. A balloon catheter was advanced via the left common iliac artery into the descending aorta under direct angioscopic vision. Balloon pumping was then commenced. The circuit was perfused with normal saline at a flow rate of 3 l/min. Pump actions of 1:1 and 1:2 were simulated. Each aorta at the end of the experiment was subjected to histological examination. Result: The balloon only appeared to make direct contact with the wall of the aorta during deflation when it was swept to one side by the circulating fluid. During maximal inflation the only points of contact were the tip of the catheter and the entry site. Side branches of the aorta were not occluded by the balloon. There was considerable atheromatous debris visualised within the lumen of the aorta. Atheromatous plaques were seen to fissure and disrupt by a pressure wave action and not by direct contact with the balloon. Conclusion: The balloon catheter moves relative to the wall of the aorta during inflation and deflation. Contact between the balloon and the aorta only occurs during deflation. Side branches of the aorta are not occluded by the catheter. Plaque disruption and embolus formation appear to result from pressure wave action rather than direct contact with the balloon. This may have implications for future balloon design. Further investigation of this poorly understood interaction between the balloon and the aortic wall is required. © 1997 Elsevier Science B.V.

Keywords: Intra-aortic balloon counterpulsation; Artificial circulation; Thoracic aorta

1. Introduction

The morbidity associated with intra-aortic balloon augmentation frequently leads to early removal of the balloon catheter in critically ill patients. Complication rates during balloon pumping have been reported at between 20 to 30% of cases [1,5,8,9]. Much of this morbidity is due to the interaction between the balloon catheter and the aortic environment although complications secondary to the physical process of insertion of the balloon have also been reported [2].

This study investigates the mechanisms responsible for the development of complications during intra-aortic balloon counterpulsation by means of studying the dynamic action of the balloon within the intact cadaveric aorta in an artificial circulation. The study addresses the following questions; what is the movement of the balloon within the circulation? How does atheromatous embolisation occur? Does the balloon directly occlude branches of the aorta?
2. Materials and methods

Five intact human aortas were used for the experiments. These were obtained from cadavers undergoing routine hospital post-mortem examination. Consent was obtained from the relatives of the deceased in each case for the use of the tissue. Details of the individual patients are shown in Table 1. Each aorta was rinsed with normal saline to remove post-mortem debris from its lumen. All side branches were ligated with the exception of the left subclavian and the common iliac vessels.

An artificial circulation was used. The circuit consisted of a standard 3/8 inch PVC perfusion tubing loop and a roller pump (Cobe Laboratories). A submicron filter (0.2 micron Pall Laboratories) was incorporated into each circuit in order to prevent recirculation of debris and embolic material. For the purpose of each experiment the loop was divided and an intact cadaveric aorta was incorporated into the circuit with the inflow at the aortic valve and the outflow at the right common iliac artery, Fig. 1. The entire length of aorta was laid on the surface of an ice bath. Deformation of the ice allowed the vessel to assume its natural shape retaining the curvature of the aortic arch. The circuit was perfused with normal saline at 3 l/min using non-pulsatile flow.

Direct angioscopic images of the interior of the aorta were obtained using a rigid video endoscope (Olympus Keymed, camera OTV 54, lightsource CLV U20, Sony monitor) inserted via the left subclavian artery, secured with a snugger. The angioscopic appearances of each aorta were noted prior to insertion of the balloon pump and the site of any atheromatous plaques recorded. An intra-aortic balloon (Datascope 9.5 Fr/40 cc) was then advanced under angioscopic control into the aorta via the left common iliac artery until its tip was just distal to the left subclavian artery. The position of the balloon catheter was confirmed by palpation through the wall of the vessel. The balloon catheter was secured with a snugger around the iliac artery forming a watertight seal. The balloon catheter was attached to a Datascope System-90 pump.

In this way the movement of the balloon within the circulation could then be directly observed. Video recordings were taken of the balloon pump action at simulated 1:1 and 1:2 pump actions within each aorta. The balloon pump was triggered using a Datascope ECG signal generator with an intrinsic rate of 60 beats per min. Attention was directed to the effects of the balloon pump action on the previously noted atheromatous areas within the aorta. Throughout each experiment the pressure within the proximal aorta was recorded continuously. At the end of each experiment each aorta was then fixed in formalin and subjected to pathological examination. Sections were taken from areas of specific interest and subjected to light microscopy after staining with haematoxylin and eosin.

3. Results

Each aorta showed evidence of atheromatous change as visualised endoscopically prior to insertion of the balloon pump. See Table 2. This was seen to be maximal on the dorsal surface of the aorta, while the ventral surface was virtually spared. It was in practice, difficult to quantify objectively the atheromatous lesions in each aorta. However aortae numbers 1, 4 and 5 were grossly affected. In two of these aortae (number 1 and 5) a large number of degenerative lesions were seen mid way between the origins of successive pairs of intercostal ostia. The appearances were of ‘yellow elevated patches.’ Some of these plaques showed a roughened surface with rigid calcified spiculae (Fig. 2 and Fig. 3).
Two of the five aortae (Aorta 1 and 2) were perfused for 1 h only as a watertight seal could not be maintained leading to subsequent leakage of the perfusate.

Intra-aortic pressure: pressure recordings during non pulsatile flow at 3 l/min, before and during balloon augmentation are given in Table 3. The average value of the mean arterial pressure recorded prior to intra-aortic balloon action was 45.4 ± 8.32 mmHg. The average value of the mean arterial pressure recorded during intra-aortic balloon action on 1:1 was 88.6 ± 9.18 mmHg and the peak augmentation pressure was 146.8 ± 22.42 mmHg. Fig. 4 and Fig. 5 show the pressure tracings before and during balloon action.

Throughout the experiment a number of fine intimal flaps were raised during instrumentaton, and visualised endoscopically as 1–2 mm flaps which oscillated during perfusion. The entire length of the descending aorta was easily visualised (Fig. 6 and Fig. 7). The 9.5 Fr/40 cc balloon with a diameter of 1.5 cm on inflation did not appear to touch the aortic wall on maximal inflation. Side branches of the aorta were not occluded by the balloon catheter during augmentation. In all cases the lower extent of the balloon appeared to be situated below the coeliac artery when the tip was immediately distal to the subclavian artery.

The movement of the balloon within the aortic lumen appeared to be complex. The central axis of the balloon occupied a series of different positions during pumping.

During deflation the balloon was seen to be swept to one side of the aorta by the circulation. (This movement appeared to be enhanced during the prolonged phase of deflation associated with a 1:2 simulated rhythm). The axis of the balloon re-obtained its central position at the end of inflation.

The catheter also moved relative to the long axis of the aorta. During inflation it advanced proximally towards the aortic valve. This position was held until deflation when it dropped back towards the bifurcation as it was moving laterally towards the wall of the aorta.

Endoscopic inspection of the aortic wall in aorta number 5 demonstrated a calcified atheromatous plaque that was disturbed by the pressure wave generated from intra-aortic balloon movement rather than from direct trauma. Fissuring of this previously smooth intimal surface of the plaque situated 3 cm distal to the left subclavian artery on the dorsal aspect of the aorta was seen during Balloon action (Fig. 2 and Fig. 3). After fissuring atheromatous material was seen embolising out of this plaque into the circulation.

A second lesion situated at the ventral surface between superior mesenteric artery and renal vessels of the aorta number 4 appeared to become mobile during balloon action but no fissuring or embolic event observed endoscopically.

At the end of the experiment each aorta was placed in 10% formalin. The interior of the aorta was ins-
Fig. 2. Angioscopic image of the tip of the balloon catheter within the lumen of the aorta adjacent to a raised atheromatous plaque. The plaque has fissured.

spected macroscopically. This revealed a distribution of atheroma as previously noted. The atheromatous disease was classified as severe by the pathologist, in aortae numbers 1, 4 and 5. Calcification and irregularity of the intima was noted in aortae numbers 4 and 5 and it was mainly distributed at the abdominal region. Plaques appeared to be disturbed in two of the aortae (number 4 and 5) and sections were taken from these areas. Plaque fissuring was confirmed on light microscopy after staining with haematoxylin and eosin.

4. Discussion

Intra aortic balloon pumping is a recognised therapeutic means of supporting the failing heart [14,17]. There is a significant morbidity associated with its use [12] and this morbidity frequently results in early removal of the balloon pump leading to subsequent haemodynamic deterioration. Although some of the complications are directly related to the insertion of the pump others are related to the interaction between the balloon and the aortic environment during balloon augmentation [15,16].

Fig. 3. Line drawing depicting the endoscopic view of the aorta in Fig. 2.
Table 3
Pressures within the proximal aorta during non pulsatile flow before and during balloon augmentation (mmHg)

<table>
<thead>
<tr>
<th></th>
<th>Aorta 1</th>
<th>Aorta 2</th>
<th>Aorta 3</th>
<th>Aorta 4</th>
<th>Aorta 5</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No balloon action</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>65</td>
<td>48</td>
<td>52</td>
<td>62</td>
<td>41</td>
</tr>
<tr>
<td>Diastolic</td>
<td>41</td>
<td>30</td>
<td>38</td>
<td>45</td>
<td>30</td>
</tr>
<tr>
<td>Mean</td>
<td>53</td>
<td>40</td>
<td>47</td>
<td>53</td>
<td>34</td>
</tr>
<tr>
<td><strong>Balloon action</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>117</td>
<td>108</td>
<td>93</td>
<td>130</td>
<td>100</td>
</tr>
<tr>
<td>Diastolic</td>
<td>47</td>
<td>62</td>
<td>73</td>
<td>57</td>
<td>41</td>
</tr>
<tr>
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<td>Augmented</td>
<td>158</td>
<td>152</td>
<td>137</td>
<td>173</td>
<td>114</td>
</tr>
</tbody>
</table>

During these experiments the balloon described a complex motion within the aorta. The axis of the balloon catheter appeared to move relative to the longitudinal axis of the aorta during inflation and deflation. At maximal inflation the balloon lay centrally within the vessel. There was no contact at this stage between the balloon and the arterial wall other than at the tip and at its site of insertion. Side branches of the aorta were not occluded even in the aorta procured from the smallest patient (Aorta number 3). During deflation however the balloon was swept to one side by the passing circulation rather like a sail in the wind. There was direct contact between the vessel and balloon at this phase in the cycle. Moreover the deflationary movement of the balloon was always held in the same position.

An additional part of this movement relative to the aorta was a slight antegrade/retrograde movement of the whole balloon during deflation/inflation. The movement of the central axis of the balloon catheter could therefore be described as circular. During inflation the balloon finds a central position within the lumen and advances by 5 mm-1 cm toward the aortic valve. During deflation the balloon falls back towards the aortic bifurcation by a similar distance and is swept sideways striking the dorsolateral wall of the vessel. This position is held longer during 1:2 cycle augmentation than in 1:1 owing to the prolonged period of deflation. We hypothesise that this may be a mechanism of balloon perforation if the catheter is constantly striking and then rubbing alongside an irregular atheromatous area.

Topography of atheromatous aortic lesions has been studied by different investigators. Cornhill et al. [4] following autopsy from 109 male subjects concluded that in the thoracic aorta the highest distribution of atheromatous disease was on the dorsal surface while the ventral surface was virtually spared; in the abdominal aorta the regions of highest distribution were associated with the inflow tracts of the coeliac, superior mesenteric and renal vessels. Distribution of atheromatous lesions along the dorsal aspect of the vessel, also noted in our sample. This is the area that the balloon catheter observed to be in contact during deflation.

Direct contact between the catheter and arterial wall may also be a mechanism of intimal insult and fissuring of plaque and atheromatous embolisation. No such event was observed during this experiment however but indirect plaque disruption did occur. This was due to the shock wave effect of inflation on a plaque adjacent to the balloon but which was not in contact with it. An intra aortic balloon typically cycles at 144 000 beats every 24 h [14], the increments of repetitive pulse pressure applied on the aortic wall during each cycle may be significant enough to produce intimal pulse especially in calcified inelastic atheromatous aortae. The
Fig. 5. Sample pressure tracing during balloon augmentation in aorta number 1.

maximal perfusion time in this series of experiments was 6 h. The disruption of the plaque during counterpulsation observed during this experiment could have been due to post-mortem changes in the plaque which altered its physical characteristics and ability to withstand the altered haemodynamic forces of counterpulsation. Alternatively this may represent a real mechanism of systemic embolisation during counterpulsation.

Previous workers have studied the incidence of systemic arterial embolisation clinically and have found that although it is well documented as a complication of balloon pump insertion or removal, it is comparatively rare during actual counter pulsation. According to various investigators more than 60% of vascular complications occur during balloon insertion [3,13]. Isner et al. [7] studied at necropsy 45 patients who died after insertion of an intra-aortic balloon assist device.

Fig. 6. Angioscopic image of the descending aorta. The intra aortic balloon catheter can be seen lying within the lumen alongside the wall of the aorta during deflation.
Fig. 7. Line drawing depicting the endoscopic image seen in Fig. 4.

Of the total 20 complications, 12 that occurred resulted from insertion of the balloon and six were due to thromboembolism. Despite the clinical picture of embolic events in those six patients the authors failed to identify plaque disruption. Complications related to the balloon itself, such as visceral ischaemia and peripheral emboli were low in a series of 103 consecutive percutaneous intraaortic balloon pump insertions published by R. Martin and associates [10]. However embolic events including spinal cord ischaemia, coronary artery occlusion, renal artery occlusion and bowel ischaemia have been reported by other researchers [11,12]. Hazelrigg et al. [6] reviewed retrospectively 100 consecutive patients treated for cardiogenic shock with the intraaortic balloon pump placed through the ascending aorta thereby eliminating possible complications that would have occurred if a femoral approach had been utilised. They diagnosed cerebral vascular accidents in 2.5% of the patients and transient ischaemic attack in 1.2%. During the same period they evaluated 49 patients that had a femoral balloon placed. 16.3% had ischaemic problems in the lower extremities, 12.2% strokes and 4.1% visual field defects possibly from embolic events.

This study was an attempt to investigate the interaction between a balloon catheter and the intact isolated human cadaveric aorta within an artificial circulation. The experiments were limited by the use of non-pulsatile perfusion and by the use of isolated aortas which were not supported by surrounding tissues and organs. Although non-pulsatile perfusion was utilised pressure recordings confirmed that augmentation of the arterial wave form did occur and this has been illustrated in the text. The aorta was suspended in an ice bath and therefore the physical characteristics of the aorta secondary to the surrounding tissues and organs within the thorax in-vivo were lost. Within these constraints the experiments did produce some original results which may lead to greater understanding of the mechanisms leading to complications in the patient undergoing balloon counterpulsation.

We have demonstrated that a balloon catheter describes a complex movement relative to the aorta during inflation and deflation. This movement includes the repetitive contact between the balloon and one area of the aortic wall. Alterations in balloon morphology and in the timing cycle of inflation and deflation may influence the complication rate of balloon augmentation in the critically ill patient. We believe that further investigation of the interaction between the balloon catheter and the aorta is indicated. Further studies should include the use of pulsatile circulation, differing balloon catheter sizes and shapes and greater numbers of aortas.

References


