Intra-Aortic Balloon Pumping (IABP) at Different Levels of Experimental Acute Left Ventricular Failure*

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Effectiveness of intra-aortic balloon pumping (IABP) as a circulatory assist device was experimentally evaluated in eight groups of dogs at various stages in the sequence: coronary occlusion—myocardial infarction—acute left ventricular failure—coronary shock. When instituted during the period of reversibility of myocardial ischemic changes produced by coronary ligation, IABP improved collateral coronary flow, limited the extent of myocardial necrosis and prevented the development of left ventricular failure. When instituted following the development of acute left ventricular failure, IABP “assisted” the left ventricle, as evidenced by improvement in left ventricular dimensions, pressures, flows, systemic and myocardial metabolic parameters. When instituted at the stage of shock, IABP had only slight and transient effects. Problems related to the use of IABP were studied. Lack of a 100 percent fail-safe device was viewed as the main deterrent, at the present time, to clinical application.

Intra-aortic balloon pumping (IABP), introduced by Moulopoulos, Topaz and Kolf in 1962 and popularized by Kantrowitz and associates in 1968, is an attractive method of left ventricular assistance, both from a theoretical and a practical point of view.

Theoretically, deflation of the balloon, synchronized with the opening of the aortic valve, represents a drop in aortic impedance which allows the left ventricle to eject more and faster, with a simultaneous reduction in intracavitary pressure; inflation of the balloon in diastole augments the diastolic aortic pressure (Fig 1) and improves coronary flow.

As for practical applicability, IABP appears ideally suited for patients who are acutely ill. The system is relatively simple, can be applied rapidly, and does not require a major surgical procedure or extracorporeal circulation.

On the other hand, numerous problems, not all of which are presently solved, are connected with its use (Table 1).

It was the purpose of this work to evaluate the effectiveness of IABP as a method of left ventricular assistance. Specifically, answers were sought to the following questions:

1) Following coronary occlusion, would IABP improve collateral coronary flow, reduce the extent of myocardial ischemia, and prevent the development of acute heart failure and shock?

2) Following the development of acute left ventricular failure, would IABP assist the left ventricle in maintaining aortic and regional flows, decrease left ventricular load and oxygen demand, and improve coronary flow and oxygen supply?

3) Following the development of shock, would IABP provide the auxiliary pump that is necessary to maintain tissue perfusion?
A Westinghouse intra-aortic balloon device was used, consisting of a standard cigar-shaped polyurethane balloon measuring 15 mm in maximum diameter, 15-20 cm in length, on a polyurethane catheter. The balloon was inserted through a femoral, or iliac, artery and advanced into the descending thoracic aorta, with the tip positioned to lie just below the origin of the left carotid artery. Actuation and synchronization were controlled by means of a Westinghouse ECG-triggering and pumping unit, using helium under 200 mm Hg pressure for inflation and a vacuum pump at -5 mm Hg for deflation.

IABP was applied in 103 mongrel dogs, averaging 20 kg in weight, arranged in eight groups. All dogs were pretreated with reserpine 0.1 mg/kg/day, given orally for three days prior to the experiment, and treated with a constant infusion of propranolol-chlorpromazine, 0.012 mg/kg/minute to a total of 2 mg/kg. This pharmacologic preparation was found in preliminary experiments to "isolate" the heart from its neurogenic-humoral controls, reduce the incidence of ventricular fibrillation or severe dysrhythmias following coronary occlusion, and keep the preparation stable for adequate periods of time.

Anesthesia was maintained with intravenous sodium pentobarbital (25 mg/kg). Respiration was assisted with a Bird respirator and endotracheal tube, using 40 percent oxygen. Euvolemia was maintained by replacing calculated blood losses with Ringer's lactate solution fourfold, "covering" the urinary output with 4 percent saline solution and obligatory losses with 5 percent dextrose/water (20 drops/min).

Acute left ventricular failure was reproduced by a method previously developed, consisting of ligation of the anterior descending ramus of the left coronary artery (ADR) at different levels, supplemented by ligation of collateral vessels at 5-15 minute intervals, in order to produce ischemia of the distal third (preparation type 1), distal half (preparation type 2), and distal two-thirds (preparation type 3) of the left ventricular free wall.

Left ventricular volumes were measured from biplane ventriculograms, using the ellipsoid reference figure and the "area-length" method of Dodge. The smallest volume was taken as ESV, the largest as EDV, StV was calculated as the difference between the two, the EF as the ratio StV/EDV.

Pressures were measured by means of 2 mm ID saline-filled polyethylene catheters inserted into the left ventricle through the apex, the left atrium through a segmental pulmonary vein, the ascending aorta through a carotid artery and connected to P23db equisensitive pressure transducers.

Arterial flows were measured by precalibrated electromagnetic flow probes and a Biotronix BL 810 flow meter. "Zero flow" obtained by the use of arterial occluders (In Vivo Metric) placed 1 cm distal to the flow probes. Left ventricular stroke volume was calculated by planimetry of the aortic flow curve corresponding to left ventricular systole. Coronary sinus flow was measured by gravity drainage of coronary sinus blood for 30 seconds through a siliconized catheter circumferentially suture-ligated at its ostium in siliconized tubes. Urinary output was measured by gravity drainage of both ureters into a timed urometer.

Arterial pH, pO₂, pCO₂, and base excess were measured by an Astrup AME II apparatus, using the Siggard-Andersen...
nomogram. Oxygen content in vol percent was calculated from the Hct and oxygen saturation, this being obtained by means of an American Optical refracting oximeter according to the formula:

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\frac{\text{Hct}}{3} \times 1.34 \times \text{O}_2 \text{ saturation} = \text{O}_2 \text{ content in vol percent}
\]

Lactate and pyruvate were measured from arterial, venous and coronary sinus blood by the methods of Barker and Summerson and Friedman and Haugen; and TBXL and MXL were calculated from Huckabee's formulas. MVO\textsubscript{2} was calculated as the product of arterial-coronary sinus oxygen content difference and coronary sinus flow.

Derived parameters—cardiac output (CO), total peripheral resistance (TPR), tension-time index—were calculated from known formulas.

Preparation type 1 produced a level of failure (LVF\textsubscript{1}) characterized by: 1) moderate increase in systolic and diastolic volumes with reduction of the ejection fraction; 2) moderate hypotension (15-25 percent reduction of mean aortic pressure); 3) moderate reduction of cardiac output (CO) with 15-25 percent reduction of aortic, carotid, renal, and coronary flows; 4) slight elevation of total peripheral resistance (5±5 percent); 5) slight elevation of AVO\textsubscript{2} (7.5±2.5 vol percent); and 6) slight elevation of TBXL (1.5±0.5 mM/L). Preliminary experiments\textsuperscript{4} demonstrated that LVF\textsubscript{1} was a stable level of failure for periods up to six hours.

Preparation type 2 produced a level of failure (LVF\textsubscript{2}) characterized by: 1) moderately severe increase in systolic and diastolic volumes with moderately severe reduction of the EF; 2) marked hypotension (25-40 percent reduction of MAP); 3) marked reduction of CO with 25-50 percent reduction of aortic, carotid, renal, and coronary flows; 4) elevation of left ventricular end-diastolic pressure (LVEDP) and mean left atrial pressure (MLAP) above 12 mm Hg (15±3 mm Hg); 5) increase of TRP (28±4 percent); 6) increase of AVO\textsubscript{2} (15±2 vol percent); and 7) elevation of TBXL (3±1 mM/L). Preliminary experiments\textsuperscript{4} demonstrated that LVF\textsubscript{2} was a stable level of failure for periods up to two hours.

Preparation type 3 produced a level of failure (LVF\textsubscript{3}) comparable to the "coronary shock" of Apress,\textsuperscript{9} characterized by: 1) severe increase in ESV and EDV with severe reduction of the EF; 2) severe hypotension (decrease above 50 percent of MAP); 3) severe reduction of CO (above 50 percent) with above 50 percent reduction of aortic, carotid, renal, and coronary flows; 4) complete failure of tissue perfusion, evidenced by severe oliguria-anuria, metabolic acidosis, and low p\textsubscript{O\textsubscript{2}}. Preliminary experiments\textsuperscript{3} demonstrated that LVF\textsubscript{3} was a stable level of failure only for periods up to 30 minutes.

RESULTS

Twenty-five balloons were used in the present groups of experiments. Fatigue-testing of the balloons was carried out by subjecting them to 48 hours of helium pumping under 250 mm Hg in a 37°C bath of saline solution. None ruptured, leaked or became deformed in vitro; however, one of the 25 leaked in the animal, producing acute gas embolism and sudden death.

Optimal balloon sizes were determined by hemodynamic monitoring and aortograms. The desired balloon size was considered to be the one that would produce the greatest hemodynamic effects (reduction of systolic left ventricular pressure, augmentation of aortic diastolic pressure, increase of aortic flow) without the balloon coming in contact with the aortic wall. Optimal sizes for dogs of 15, 20, and 25 kg weight proved to be balloons with volumes of 15, 20, and 25 ml, respectively, with diameters no larger than 15 mm.

The best position was found to be that with the balloon tip lying just below the origin of the left carotid artery.

The problem of mistriggering from dysrhythmias was circumvented by using pretreatment with reserpine, which depletes the myocardium of the endogenous catecholamine stores\textsuperscript{10} and thus prevents the liberation of catecholamines produced by myocardial ischemia, and treatment with propranolol, which has a definite anti-arrhythmic property.\textsuperscript{11} The incidence of ventricular fibrillation in animals so treated was reduced to 10 percent of that in untreated animals;\textsuperscript{a} the incidence of severe dysrhythmias was markedly reduced; heart rate was maintained in a range of 90-110 strokes/minute, most convenient for this
method of assisted circulation.

For synchronization of balloon deflation with the opening of the aortic valve, it was found that the best signal was the peak of the LVP dp/dt curve (Fig 2). Inflation of the balloon was timed with the dicrotic notch of the central aortic pressure waveform.

For the best rate of balloon inflation-deflation, pumping with helium under 200 mm Hg and using a vacuum pump at -5 mm Hg for deflation was found to be most effective.

The mode of gas transfer (from the inflated or deflated balloon position) appeared to be an important problem. In the system operating from the inflated position, non-triggering caused by arrhythmias left the balloon inflated for one or more beats, producing sudden and irregular increase in aortic impedance. In the other system, it was more difficult to synchronize deflation of the balloon with the opening of the aortic valve.

**Group 1.** Following ligation of the anterior descending ramus at its upper/middle third in the control group of six dogs, circumflex artery flow and coronary sinus flow remained unchanged until development of hypotension, when they gradually decreased.

In the group of six dogs in which IABP was instituted immediately after coronary ligation (Table 2), circumflex artery flow was increased 13-35 percent, coronary sinus flow 2-10 percent.

**Group 2.** Two hours after ligation of the circumflex artery in the control group of six dogs, "fuchsinophilic degeneration" was found to involve 75-80 percent of the myocardial cells of the posterior papillary muscle, as compared with the uninfarcted anterior papillary muscle (Fig 3). By contrast (Fig 4), only 10 percent were involved at the end of two hours in the animals treated with IABP.

**Group 3.** In six dogs, IABP was instituted immediately after completion of the pharmacologic-surgical preparation type 1, normally leading to LVF. The development of failure was prevented. When pumping was discontinued after one hour, the animals remained stable and were alive at the end of the sixth hour.

**Group 4.** In 12 dogs, IABP was instituted after completion of preparation type 2, normally leading to LVF. The development of failure was prevented. Upon discontinuation of pumping—after one hour in three dogs, after two hours in three dogs, three hours in three dogs, and four hours in three dogs—the animals developed left ventricular failure (LVF).

**Group 5.** In six dogs, IABP was instituted immediately after completion of preparation type 3, normally leading to LVF. The development of failure was retarded, but not prevented. These animals survived longer (average 120 min) than those of Group 8 (average 90 min), but all died: three in ventricular...
fibrillation, three in progressive failure, shock and asystole.

**Group 6.** In 15 dogs, IABP was instituted after recognition of LVF₁. The effects were evaluated in terms of changes in volumes, pressures, flows, systemic and myocardial metabolic parameters. As stroke volume (StV) was improved (19-25 percent), there was a return to pre-failure levels of left ventricular end-diastolic (EDV) and end-systolic (ESV) volumes. There was a lowering of systolic pressures (17-24 percent) with an average 6 percent increase of mean AoP. Aortic flow was improved an average 19-25 percent, carotid flow (CaF) 14-27 percent, renal flow (ReF) 9-25 percent and coronary sinus flow (CSF) 7-25 percent. Total peripheral resistance was decreased 3-5 percent. The improvement of total body excess lactate (TBXL) and myocardial excess lactate (MXL) reflected the betterment of tissue perfusion. When pumping was discontinued after one hour, the animals' condition remained stable. Survival at six hours was 100 percent.

**Group 7.** In 25 dogs in LVF₂, most significant were the improvement of the ejection fraction (EF) of the left ventricle, with decrease of ESV and EDV, and the return to normal of mean left atrial pressure (MLAP) (Fig 5 and 6). StV was increased an average of 16 percent, the EF 55 percent. Systolic pressures were further decreased an average 5 percent, while mean AoP was increased an average 10 percent. Aortic flow was improved an average 16 percent, CaF 14 percent, ReF 27 percent, CSF 8 percent. Total peripheral resistance was decreased 19 percent. Improvement of tissue perfusion was demonstrated by a 54 percent decrease in A-V oxygen difference, and a 58 percent decrease in TBXL. The improvement in respiratory function was reflected by improvement in arterial pO₂ and oxygen saturation. The improvement in myocardial metabolism was reflected by a 70 percent decrease in MXL.

The improvement in all parameters was, however, found to be pump-dependent for the periods of time the experiments were conducted, ie up to four hours, and discontinuance of pumping caused a rapid deterioration in the animals' conditions. While survival was improved in the animals being assisted, "weaning" of the assist device proved to be a serious problem. Of 25 control dogs (dogs in LVF₂ not assisted by IABP), five (20 percent) died in ventricular fibrillation or asystole between the second and the third hour, six (25 percent) died between the third and fourth hour, eight (32 percent) died between the fourth and fifth hour, six (25 percent) were still alive at six hours. Of the assisted dogs, none died during four hours of pumping; ten (40 percent) died within one hour after pumping was discontinued, 15 (60 percent) died within the second hour.

**Group 8.** In 15 dogs in LVF₃, IABP had only transitory hemodynamic effects. During the first 15 minutes of pumping, StV was increased an average

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**IABP in LVF₂**

![Diagram](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21507/)

**Figure 5.** IABP in LVF₂: mean left atrial pressure is 24 mm Hg. Immediate decline is evident upon institution of IABP.

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25 percent, EF 44 percent, mean AoP 3 percent; TPR was reduced an average 22 percent. These effects were, however, transient. Systolic pressures were not affected; LVEDP and MLAP were not improved; CVP gradually rose (300 percent of pre-failure). Carotid flow was reduced an average 38 percent, renal flow and urinary output remained unchanged, and coronary sinus flow was slightly decreased. The metabolic parameters reflected the progression of failure: arterial pH decreased to 7.25; arterial pO₂ remained unchanged; A-V oxygen difference and TBXL were not affected; MXL remained high. All parameters followed a downhill trend; all animals died within the same period as the 15 untreated dogs: 60 ± 30 minutes.

**FIGURE 6.** After discontinuance of pumping, MLAP immediately rises, and deterioration of the animal's condition is further evidenced by reduction of systemic pressure and coronary flow.

**FIGURE 7.** IABP in LVF₃: aortograms show retraction-expansion of the aortic wall produced by deflation-inflation of the balloon because of reduced intra-aortic blood volume.
"The decision to mechanically support a failing heart need not be an emergency." Proper timing of application is, in fact, a key for the success of this method of assisted circulation.

In Groups 1-4, the increase in coronary flow, both arterial and venous, does not necessarily imply an improvement of tissue perfusion. "Fuchsinophilia" is a non-specific histologic method for the demonstration of "early myocardial infarction." One might also argue that the great degree of variability of coronary collateral circulation in different dogs could have influenced the results of balloon pumping. However, considering the results of these four groups of experiments together, one is probably justified in concluding that IABP is indeed capable of reestablishing circulation of oxygenated blood to ischemic myocardial areas, thus limiting the extent of myocardial necrosis.

Jennings and associates demonstrated the sequelae of coronary occlusion to be especially uniform in the posterior papillary muscle of the left ventricle following ligation of the circumflex artery. They noted by electron microscopy that ischemic myocardial cells could regain contractility if circulation of oxygenated blood was restored within 45 minutes. This, therefore, appeared to be in the dog the time of reversibility before development of myocardial necrosis. From the presented results, it may be assumed that, when used within this period of reversibility, IABP can recuperate a large number of ischemic myocardial cells that would otherwise become necrotic.

The poor results in Group 5 suggest that extensive coronary occlusion, producing myocardial ischemia involving two-thirds of the free left ventricular wall, is of an extent that cannot be helped by IABP. The results of Groups 6, 7, and 8 indicate that the greater the extent of myocardial injury, the less can be expected of IABP. In LVF, IABP not only restored normal hemodynamics, but also, when discontinued after one hour, left the animal in a normal, stable condition. In LVF, left ventricular assistance was found to be pump-dependent; and discontinuance of pumping caused rapid deterioration in the animal's condition. In LVF, IABP could produce transient hemodynamic improvement, but no improvement in the metabolic parameters or in survival.

While the results of the present work are in agreement with those of some investigators as to the enhancement of coronary flow and cardiac output, decrease of total peripheral resistance and improvement of survival, they appear to be in disagreement with those of others who have advocated the use of IABP for the treatment of coronary shock. This may be due to the use of different experimental models and different criteria for the definition of shock.

The poor performance of IABP at the stage of...
shock can be easily explained. From current understanding of the physiopathologic mechanisms operating in coronary shock, it appears that IABP cannot by itself correct all the derangements of this condition. Respiratory failure, relative hypovolemia, microcirculatory changes, and myoelectrical instability cannot be influenced by IABP. On the other hand, balloon pumping appears to be limited in its performance by the "occlusive position" the balloon comes to occupy when the severe reduction of CO produces a severe reduction of central aortic volume. In this situation, inflation-deflation of the balloon (Fig 7) appears to produce an expansion-retraction of the aortic wall more than a movement of blood. Furthermore, analysis of the carotid flow curve shows that, due to disproportion between balloon and left ventricular stroke volumes, deflation of the balloon produces a negative pressure gradient in the central aorta leading to retrograde flow (Fig 8).

Although quantification of left ventricular failure has been recognized as necessary in the human, there is no method at the present time capable of indicating precisely the extent of coronary occlusion and myocardial ischemia, the severity of the patient's "status." However, a "trend" can be detected in the clinical course through accurate monitoring of myoelectrical activity and hemodynamic-metabolic parameters. It is the authors' belief that early diagnosis of a trend of deterioration, despite adequate pharmacologic treatment, indicates the proper time for the institution of IABP. Further deterioration, despite IABP, signifies myocardial injury of a severity greater than can be aided by this method and provides the indication for additional measures of assisted circulation. The question of duration of left ventricular assistance necessary for restoration of spontaneous adequate performance is at the present time unsolved. Trial of withdrawal remains the only criterion available.

Conclusions

1) The results of intra-aortic balloon pumping in 103 pharmacologically-prepared mongrel dogs, in which grade ischemic failure had been induced, have been presented.

2) Under the circumstances of these experiments, in other than massive coronary occlusion, IABP is capable of improving myocardial perfusion, reducing the extent of myocardial ischemic damage, and of preventing the development of left ventricular failure.

3) Assuming a clinical extrapolation of these data, IABP, once proved to be safe, should be used early in the course of progressive ischemic left ventricular dysfunction.

4) When employed after the development of acute left ventricular failure, IABP can improve stroke volume and the ejection fraction of the left ventricle, improve aortic, carotid, renal, and coronary flows, reduce LVEDP and MLAP, improve tissue perfusion, and increase survival.

5) IABP is not of benefit in terminal experimental ischemic left ventricular failure — "coronary shock."

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Seeming Optical Illusion: Ptosis of the Larynx in Patients with Emphysema

Cardinal pathologic changes characteristic of emphysema, a term certainly shorter and more expedient than chronic obstructive lung disease or other complex euphemistic terms, are: 1) increased vacuolation and deterioration of the alveolar parietes, with consequent formation of large air spaces; 2) dilatation, atrophy and complete disruption of walls of respiratory bronchioles; 3) fragmentation and disturbed pattern, decrease in the number and disappearance of adjacent elastic fibers; 4) obliteration and loss of perialveolar capillaries. The summation of these adverse changes is decrease in the specific terminals of the lung for gas exchange, the transformation of certain extent of the lung parenchyma into an irreversible confluence of the terminal air passages and into functionless, decreased surface area. Centrilobular emphysema with typical anatomic damage in the area of the respiratory bronchiole near the center of the secondary lobule is commonly found in the upper portions of the lung. Panlobular (panacinar) emphysema with destruction and destruction of alveoli connected to the respiratory bronchiole is likely to be prevalent in the lower parts of the lung. It is reasonable to postulate that regardless of the pattern of distribution of pathologic changes, pathogenetic agents are probably one or the combination of several of the following: 1) bronchospasms induced by atmospheric irritants, oxides of nitrogen, ozone, sulfuric acid, industrial pollutants, allergens, cigarette smoke; bronchospasms interfere with physiologic bronchocatharsis and may favor aerodynamic trauma on the alveolar septa by heightened intrapulmonary air pressure; 2) alveolar septa may be damaged by the toxic effect of oxidizing type or reducing type of air pollutants, as well as by microbial toxins; 3) excessive cough associated with bronchopulmonary irritation or infection may result in pulmonary pneumonic hypertension and consequent damage to the terminal elements of the lung; 4) enzymatic deficiency (deficiency in alpha one antitrypsin) may undermine the integrity of the lung parenchyma; 5) high levels of carbon dioxide may exert deleterious influence upon the alveolar walls, as suggested by the experimental work of Xalabarder (Inst. Antituberc. "Francisco Moragas," Barcelona, 14:25, 1961); 6) Cloetta (Arch. exper. Path. Pharmac. 66:409, 1911 and Virchow’s Arch. 152:339, 1193) first expressed the view that oblitative changes in the blood vessels of the lung contributed to degenerative alterations in its elastic fibers and alveoli. With major loss of the pulmonary elastic elements, the lung's hilusward recoil is greatly decreased, with consequent pronouced decrease in or complete disappearance of the intrapleural negative pressure. Because of this, the inspiratory muscles are bound to distend the thoracic cage. Also, there is a lowering of the level of the diaphragm. If it is agreed that 0.7 part of the vital capacity of the lung is contributed by the function of the diaphragm, it is obvious that impairment of diaphragmatic function shifts the task of maintaining pulmonary ventilation upon other inspiratory muscles. Measurements by Bjorkman (Nord. Med. Tdskr. 10:1964, 1935) while inducing artificial pneumothorax demonstrated that only one-fifth of the air injected resulted in pulmonary relaxation while four-fifths was contributing to expansion of the chest wall. Pronounced interference with the normal ventilatory mechanics of the chest, that is, the movement of air into and out of the lung, constitutes ventilatory insufficiency. This, in turn, brings to the fore increased functioning of the external intercostal and the intercartilaginous portion of the internal intercostal muscles. Their action tends to raise the ribs. The axis of rotation of the upper ribs is responsible for their ventral movement (elevation) which is larger than that of the lower ribs, with corresponding changes in the dorsoventral diameter of the thoracic inlet. Ventilatory movement of the first rib tends to raise the sternum. The scaleni and the sternocleidomastoid muscles have a significant role as auxiliary respiratory muscles. The former elevate the first two ribs; the sternocleidomastoids elevate the sternum. As a result of such exaggerated function of these auxiliary respiratory muscles and elevation of the thorax upward and forward, the larynx may appear in a position lower than normal. This finding should not be taken as an indication of ptosis of the larynx; rather it should be considered a result of higher than normal position of the operculum of the thoracic cage.

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