Intra-Aortic Balloon Counterpulsation for the Treatment of Ischemic Stroke

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Abstract

Colloid volume expansion has been shown to increase cerebral blood flow to ischemic brain in an animal stroke model and improve recovery in patients. It is, however, potentially hazardous to use in older patients because of frequently associated cardiovascular disease.

Intra-Aortic Balloon Counterpulsation might reduce the risks of using volume expansion therapy in the elderly patient.

This study was designed to see if Intra-Aortic Balloon Counterpulsation (without volume expansion), in an animal with a normal heart, would increase cerebral blood flow and EEG activity in the ischemic brain.

Unilateral cerebral ischemia was produced in baboons (n = 9) after right middle cerebral artery occlusion. A 12 ml intra-aortic balloon catheter was introduced into the descending aorta via the femoral artery prior to middle cerebral artery occlusion. The balloon was positioned distal to the origin of the left subclavian artery and following middle cerebral artery occlusion was inflated with each R wave on the ECG. Cardiac output, cerebral blood flow (by Hydrogen wash-out), computer-mapped EEG, and hemodynamic data were collected prior to middle cerebral artery occlusion and following occlusion both before and during counterpulsation.

Intra-Aortic Balloon Counterpulsation produced a significant increase in pulse pressure from 54.7 ± 21 to 70.6 ± 33 mmHg (p = .043). No significant change was seen in cardiac output, mean arterial pressure, or cerebral blood flow. Although the computer-mapped EEG improved and the right (ischemic) hemisphere cerebral blood flow did increase slightly from 16.9 ± 6.5 to 18.3 ± 8.3 ml/100 gm/min, the cerebral blood flow changes were not significant (p = .295).

It is possible that the desired increase in cerebral blood flow was not achieved partly because the animals were only 3–4 years old and were difficult to stroke.

We believe that there is merit to a follow-up study in older primates with colloid volume expansion where Intra-Aortic Balloon Counterpulsation is used to protect the heart from the deleterious effects of volume expansion and where the cardiac effects of volume expansion and counterpulsation are quantified.

Perhaps volume expansion with Intra-Aortic Balloon Counterpulsation will be safer and more effective than either treatment modality alone.

(All data reported as mean ± standard deviation)

Introduction

Stroke is the third leading cause of death in this country. Over three hundred thousand Americans are incapacitated by a stroke every year. The medical treatment of stroke has undergone evolution over time but is still ineffective in reducing death or severe dysfunction following an acute episode of cerebral ischemia. Allcock1 reports that only 29% of patients with a middle cerebral artery (MCA) occlusion had a good outcome after treatment, while Lascelles and Burrows2 reported only an 18% complete or almost complete recovery. Other studies have reported that 803 to 88%4 of patients had moderate to severe dis-
abilities or could not return to their previous lifestyles. The reported mortality from MCA occlusion varies between 5-21%. 

Neither emergency surgery nor anticoagulation have been successful in the treatment of MCA occlusion. 

Intravascular volume expansion with a colloid has been fairly successful in increasing cardiac output (CO) and cerebral blood flow (CBF) in experimental models of MCA occlusion and the clinical application has demonstrated some success.

Older patients are often the victims of stroke and the use of colloid volume expansion in this group has been less beneficial than in younger patients. In addition, the risk of volume expansion, increasing CO, preload, and afterload is increased in the older age group because of associated cardiovascular disease.

In a study from Northwestern 132 patients with first thrombotic or embolic stroke were studied during a comprehensive rehabilitation program. Sixty-one (46%) of these patients had coronary artery disease and, in addition, 16 of these had congestive heart failure (CHF). These (CHF) patients experienced three times the number of cardiac complications as the other patients. Coronary artery disease with and without CHF significantly lessened improvement during rehabilitation. These patients would clearly be at increased risk with colloid volume expansion therapy.

There is some disagreement as to why volume expansion is beneficial. Some argue that it is due to the increase in cardiac output while others contend that the mechanism is that of hemodilution and a decrease in viscosity. It is probably a combination of effects but the clinical study 4 that demonstrated little improvement in older patients, who also had smaller increases in cardiac output, suggests that the improvement in the CBF is largely dependent upon an increase in stroke volume which means an increase in myocardial work.

The Intra-Aortic Balloon Pump (IABP) has been shown to be helpful in the treatment of myocardial ischemia and myocardial infarction. IABP therapy increases myocardial blood flow during diastole and decreases myocardial oxygen consumption by decreasing afterload.

The use of IABP was not shown to improve CBF significantly in a canine stroke model although there was improvement in the CBF of some of the six animals in the experimental group. The use of the IABP in conjunction with volume expansion may offset some of the problems associated with volume expansion alone in older patients.

The purpose of this study was to evaluate IABP alone to see if there was an increase in CBF during counterpulsation in a primate model.

Materials and Methods

Nine young adult baboons (age 3-4 years) weighing 7-12 kgs. were anesthetized initially with ketamine 10 mg/kg and then Fentanyl 0.1 mg/kg. The animals were then intubated and ventilated with 100% oxygen. Twelve EEG leads and four CBF electrodes were implanted into the skull as previously described. ECG electrodes were attached to the limbs and blood pressure was measured in the ascending aorta by a catheter placed in the brachial artery.

Cardiac output was measured by green dye dilution using a cardiac output computer and a catheter in the brachial artery.

EEG power spectral mapping-Computer Mapped EEG (CME) was analyzed by dedicated hardware and software as we have previously reported. CBF measurements were determined from hydrogen clearance curves using the T 1/2 method and the initial slope index. The surgical technique is described elsewhere as is the measurement technique and instrumentation. The 12ml Intra-Aortic Balloon catheter was placed into the distal aorta via the femoral artery. The catheter was advanced so that the tip was located distal to the origin of the left subclavian artery. The balloon catheter was connected to an Intra-Aortic Balloon Pump console.

After baseline data were collected the right MCA was exposed and clipped via the transorbital approach as detailed by Hudgins. After a stabilization period of thirty-sixty minutes EEG, CBF, CO, and blood pressure measurements were recorded. After these data were recorded the IABP was started at a rate of 1:1. Measurements of the study parameters were again made after a sixty minute period of counterpulsation.

Statistical analysis of the data was performed on a computer using the SPSS statistical package. The test of significance was the two tailed t-test. All data are reported as mean ± S.D.

Results

IABP counterpulsation increased the pulse pressure from 54.7 ± 21 to 70.6 ± 33 mmHg (p = .043). There was no significant change in CO, mean arterial pressure or CBF in the baboons with IABP. The EEG power

a Model DTCC0-07, E for M-Honeywell Inc., Pleasantville, N.Y., 10570
b CME 100-07, Japan Systems Co. LTD., Tokyo, Japan
c Datascope Corp., Paramus, NJ 07652
d Datascope System 83, Datascope Corp., Paramus, NJ 07652
e SPSS, SPSS Inc., 444 N. Michigan Ave., Chicago, IL 60611
spectra analyzed by CME did improve and the right (ischemic) hemisphere CBF did increase slightly from $16.9 \pm 6.5$ to $18.3 \pm 8.3 \text{ml/100gm/min}$ although the increase was not significant ($p = .295$).

**Discussion**

This study demonstrates that in this model (young-3-4 year old baboons) and with this sample size ($n = 9$) there was no increase in CBF in the ischemic brain with IABP counterpulsation following right MCA occlusion.

Some of these animals were difficult to stroke and this may have been a function of their relatively young age.

This study suggests that IABP may not, in and of itself, increase CBF in the ischemic brain in the presence of a normal heart and a normovolemic status.

If IABP has a place in the treatment of ischemic stroke, it may be as a protection against increased myocardial work in colloid volume expansion therapy. *Neurosurgery.* 18:397–401, 1986.


