Cardiac performance enhancement from dobutamine in patients refractory to hypervolemic therapy for cerebral vasospasm

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The use of the beta-agonist dobutamine in combination with hypervolemic preload enhancement of cardiac performance was analyzed in 23 patients who failed to respond to traditional preload enhancement following aneurysmal subarachnoid hemorrhage. The patients ranged in age from 13 to 82 years, and three had a history of cardiac disease. Each patient underwent placement of a flow-directed balloon-tipped catheter and the following measurements were obtained during hyperdynamic therapy: pulmonary artery wedge pressure, central venous pressure, cardiac index, stroke volume index, total peripheral resistance, and left ventricular stroke work index (LVSWI). Mean baseline cardiac function was found to be within normal limits (LVSWI = 47.6 ± 4.2 gm/min/sq m and cardiac index = 3.30 ± 0.22 liter/min/sq m). After baseline measurements were recorded, 5% albumin was infused at 300 cc/hr and dobutamine was initiated at a rate of 5 to 10 µg/kg/hr. This hyperdynamic therapy with dobutamine in the presence of volume loading resulted in a 52% increase in cardiac index, a 15% increase in LVSWI, and a 21% decrease in total peripheral resistance. The clinical reversal of ischemic symptoms due to subarachnoid hemorrhage was evident in 18 (78%) of the 23 patients.

KEY WORDS • cerebral vasospasm • cardiac index • pulmonary artery • wedge pressure • subarachnoid hemorrhage • dobutamine

Volume expansion and induced hypertension continue to be the mainstay of therapy for the prevention and treatment of cerebral vasospasm induced by subarachnoid hemorrhage (SAH). In order to minimize the cardiac, hematological, and pulmonary sequelae of volume expansion and to maximize cardiac performance during therapy for cerebral vasospasm, we have employed flow-directed balloon-tipped catheters with cardiac output and hemodynamic monitoring.

We have previously described the effect of hypervolemic preload enhancement on cardiac performance following aneurysmal SAH, concluding that in previously healthy individuals a pulmonary artery wedge pressure (PAWP) of 14 mm Hg was associated with maximum cardiac performance. The current study addresses the treatment of patients with vasospasm who fail to respond to hypervolemic preload enhancement. In initiating this study, we sought to answer a number of questions, including the following: 1) can we markedly increase cardiac output without initiating cerebral, cardiac, and/or pulmonary sequelae? 2) can patients who fail to respond to hypervolemic preload enhancement potentially respond to further enhancement of their cardiac function with dobutamine? and 3) what is the result of dobutamine treatment on the cardiac parameters and neurological outcome of patients who fail to respond to hypervolemic preload enhancement alone?

Clinical Material and Methods

Patient Population

During the period from July, 1988, to August, 1991, 208 patients with a diagnosis of intracranial aneurysm underwent surgical intervention by the senior author (S.L.G.). Of these patients, 160 presented with SAH on admission, and hypervolemic therapy was begun in the intensive care unit at Los Angeles County - University of Southern California Medical Center or Huntington Memorial Hospital, as described below.

Fifty-five patients (34%) manifested neurological compromise as a result of vasospasm during hospitalization while on hypervolemic therapy. These patients
had Swan-Ganz catheters placed and their cardiac parameters maximized by the infusion of colloid based on the calculation of Starling curves. The 23 patients who remained refractory to hypervolemic therapy following Swan-Ganz catheterization and then were given dobutamine therapy are described in this study (Table 1).

Twenty patients had no history of cardiac or pulmonary disease or administration of drugs that might affect cardiac performance. Three patients had a history of cardiac disease. One patient (Case 18) suffered from a prior myocardial infarction and had associated left ventricular hypertrophy, one (Case 19) had congestive heart failure, and one (Case 21) suffered an acute inferior myocardial infarction just prior to transfer to the neurological service.

Patients ranged in age from 13 to 82 years (mean ± standard deviation 43.8 ± 16.72 years), and included eight males and 15 females. Based on the Hunt and Hess classification,14 three patients were assessed to be in Grade I, seven in Grade II, 11 in Grade III, and two in Grade IV. Aneurysms were located in the internal carotid artery in five patients, in the middle cerebral artery in three, the anterior communicating artery in eight, the basilar artery in one, the cavernous carotid artery in one, and the posterior communicating artery in two. Three patients had multiple aneurysms.

Although every attempt is made to operate within 72 hours on the majority of the patients in the better grades (I to III), a large number of patients are transferred from other facilities, and therefore clip ligation of the ruptured aneurysm is delayed. Despite this, the majority of patients with ischemic complications whom we manage are in the postoperative period. The new onset of lethargy with or without a focal neurological deficit was presumed evidence of the onset of cerebral vasospasm until proven otherwise.

Patients presenting in Grades I, II, or III underwent operative intervention within 7 days (mean 4 days), whereas patients presenting in Grade IV underwent operative intervention 14 days or more following the initial SAH. The onset of delayed ischemic neurological deficit ranged from Day 5 to 15 (mean 9.6 days) in those patients in whom the date of initial SAH was documented upon admission. Fifteen patients underwent hyperdynamic treatment following surgery, while eight were treated prior to surgery. Patients in this study were hospitalized for a mean of 26 ± 18 days. There were no complications secondary to either Swan-Ganz catheter placement or volume expansion in this series.

Thus, patients can be identified as either those who failed to respond to hypervolemia and required Swan-Ganz catheter placement (Group I) or those who remained refractory to catheterization and required the initiation of dobutamine (Group II).

**Protocol for Hypervolemia**

All 160 patients with a diagnosis of ruptured cerebral aneurysm were admitted to the neurological intensive care unit, received a bolus of 500 to 1000 cc of 5% albumin upon admission, and were hydrated at 150 cc/hr with lactated Ringer's solution. The beginning of vasospasm was defined by the new onset of lethargy with or without a focal neurological deficit, and supportive evidence consisted of either abnormally elevated velocity studies15-20 or narrowing of involved vessels on cerebral angiography. Computerized tomography (CT) scans were obtained on all patients to rule out epidural, subdural, or intracranial hematoma or hydro-

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**Table 1**

Clinical data for patients receiving hyperdynamic-hypervolemic therapy.*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Sex</th>
<th>Day of Span‡</th>
<th>Aneurysm</th>
<th>Neurological Grade</th>
<th>Response to Tx‡</th>
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<td>F</td>
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<td>III</td>
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<td>+</td>
</tr>
<tr>
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<td>24</td>
<td>F</td>
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<td>III</td>
<td>I</td>
<td></td>
</tr>
<tr>
<td>5</td>
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</tr>
<tr>
<td>6</td>
<td>31</td>
<td>F</td>
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<td>ICA</td>
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<td>7</td>
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<td>+</td>
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<tr>
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<td>F</td>
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<td>+</td>
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<td>42</td>
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<td>+</td>
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<tr>
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<td>I</td>
<td>+</td>
</tr>
<tr>
<td>17</td>
<td>58</td>
<td>F</td>
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<td>+</td>
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<tr>
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<td>61</td>
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<td>+</td>
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<tr>
<td>20</td>
<td>64</td>
<td>F</td>
<td>6</td>
<td>ACoA</td>
<td>II</td>
<td>II</td>
<td>+ intraop rupture</td>
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<tr>
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<td>V</td>
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<td></td>
</tr>
<tr>
<td>23</td>
<td>82</td>
<td>F</td>
<td>4</td>
<td>basilar</td>
<td>IV</td>
<td>II</td>
<td>+</td>
</tr>
</tbody>
</table>

* Tx = treatment; ACoA = anterior communicating artery; ICA = internal carotid artery; cavernous = cavernous carotid artery; MCA = middle cerebral artery; PCoA = posterior communicating artery; basilar = basilar artery. Neurological grading according to the Hunt and Hess scale.11

† Time of vasospasm after subarachnoid hemorrhage

‡ Symbols: + = reversal of ischemic symptoms from vasospasm noted clinically; - = no reversal noted.
cephalus as the cause of the decline in neurological status.

Fifty-five patients with evidence of spasm refractory to hypervolemia (Group I), as manifested by continued or progressive compromise, immediately underwent systemic and pulmonary arterial catheterization, and a No. 7 French 110-cm flow-directed thermodilution pulmonary artery catheter was positioned using a subclavian approach.

Baseline cardiac index (CI), stroke volume index (SVI), left ventricular stroke work index (LVSWI), and right ventricular stroke work index (RVSWI) were calculated as follows:

\[
CI = \text{cardiac output + body surface area}
\]

\[
SVI = \text{CI} \times \text{heart rate}
\]

\[
LVSWI = SVI \times (\text{MABP} - \text{WP}) \times 0.0136
\]

\[
RVSWI = SVI \times (\text{MPAP} - \text{CVP}) \times 0.0136,
\]

where MABP is the mean arterial blood pressure, WP the wedge pressure, MPAP the mean pulmonary artery pressure, and CVP the central venous pressure.

Following baseline measurements, fluid enhancement was instituted with hydroxyethyl starch or 5% albumin delivered at 300 cc/hr until the PAWP ranged from 12 to 16 mm Hg. Patients without cardiac or pulmonary compromise were then hydrated to maintain their PAWP at approximately 14 mm Hg. If the PAWP was greater than 16 mm Hg, mannitol (0.25 to 0.50 gm/kg) or furosemide (Lasix, 40 mg) was administered. If the PAWP was less than 12 mm Hg, the patient was given boluses of 5% albumin until the PAWP returned to 14 mm Hg.

For the three patients with cardiac compromise, the PAWP was recorded every 15 minutes until elevations to 20 mm Hg were documented. Cardiac performance curves were then generated for each patient, and their PAWP was subsequently maintained at a level where the CI and LVSWI were maximized.

Protocol for Dobutamine Treatment

Twenty-three patients who failed to respond to Swan-Ganz catheterization and volume enhancement (defined as having no improvement or a decline in neurological status over a 6-hour period, Group II) were given 5 to 10 \(\mu\)g/kg/min of dobutamine by intravenous infusion. Infusion rates of dobutamine were predicated on the maintenance of cardiac performance above that of the patient's physiological baseline level as established by the calculation of Starling curves upon admission to the intensive care unit (mean 3.30 ± 0.22 liter/min/sq m). Despite the effect of dobutamine in reducing PAWP, fluid resuscitation was continued as before with hydroxyethyl starch or 5% albumin at 300 cc/hr if the PAWP was less than 12 mm Hg. If the PAWP was greater than 16 mm Hg, 0.25 to 0.50 gm/kg of mannitol or 40 mg Lasix was administered.

Dobutamine treatment was continued based on clinical neurological response. In patients responding to dobutamine treatment, infusions were tapered over 3 to 4 days following initial response. Infusions were reinstated if ischemic neurological compromise occurred. In patients failing to respond to dobutamine treatment, infusions were tapered following a 10-day course.

Statistical Analysis

In interpreting the relationship between changes in the SVI, LVSWI, and CI as PAWP was increased during volume expansion, paired samples were analyzed using Student's t-test, with results being significant at p less than 0.05 in a two-tailed test.

Results

Cardiac Parameters

Fifty-five (34%) of 160 patients had clinical manifestations of neurological compromise resulting from vasospasm during their hospitalization. Of these patients, 22 responded to hypervolemia alone, with reversal or stabilization of their deficit, and did not require placement of a Swan-Ganz catheter and/or infusion of dobutamine. Baseline measurements of the CI, SVI, LVSWI, and RVSWI in the 55 patients with clinical manifestations of neurological compromise were calculated following Swan-Ganz catheterization in conjunction with volume enhancement and following volume enhancement and the institution of dobutamine (Table 2). Mean parameters (± standard deviation) of cardiac function following volume loading and Swan-Ganz catheterization in patients without cardiac compromise were an LVSWI of 47.6 ± 4.2 gm/min/sq m and a CI of 3.30 ± 0.22 liter/min/sq m. Hyperdynamic therapy with dobutamine resulted in a 52% increase in mean CI (5.0 ± 0.26 liter/min/sq m) and a 15% increase in mean LVSWI (54.51 ± 6.3 gm/min/sq m). Total peripheral resistance decreased by 21%, MABP increased by 11%, and heart rate increased by 20%.

Clinical Response to Hyperdynamic Therapy

The reversal of ischemic symptoms from vasospasm was noted clinically in 18 (78%) of 23 patients undergoing hyperdynamic treatment ranging from 4 to 10 days after failure to respond to hypervolemia. Of the 18 patients with clinical reversal, nine stabilized following initiation of hyperdynamic therapy: six improved

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Hypervolemia Alone</th>
<th>Hypervolemia With Dobutamine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>82.7 ± 4.3</td>
<td>99.6 ± 61</td>
</tr>
<tr>
<td>Cardiac index (liter/min/sq m)</td>
<td>3.30 ± 0.22</td>
<td>5.0 ± 0.26</td>
</tr>
<tr>
<td>LVSWI (gm/min/sq m)</td>
<td>47.6 ± 4.2</td>
<td>54.51 ± 6.3</td>
</tr>
<tr>
<td>Total peripheral resistance (dynes/sec/cm⁵)</td>
<td>1125.5 ± 115</td>
<td>894.0 ± 126</td>
</tr>
</tbody>
</table>

* Means values are expressed as ± standard deviation for 22 cases with hypervolemia alone and 23 with hypervolemia in conjunction with dobutamine. All values are significantly different from controls (p ≤ 0.01). Dobutamine was infused at a rate of 5 to 10 \(\mu\)g/kg/min.
Use of dobutamine in cerebral vasospasm

one grade and three improved two grades on the Hunt and Hess scale. Currently, no significant correlations could be attempted with regard to morbidity, given the absence of a control group and a patient population of this size.

Maximum therapy was attempted in all patients. Clinical evidence of the reversal of neurological symptoms at a critical level of CI was patient-specific. Once a critical index was identified, the dobutamine drip was titrated to maintain or exceed this level at all times. There was no significant difference in the mean CI between the 18 patients who responded to dobutamine and the five who did not. Two patients who failed to respond were noted to have cerebral infarcts on serial CT scans and, thus, there was no potential for response in these patients. It should be mentioned that this protocol is one that is evaluated on an hourly basis, and the determination to continue, increase, and/or decrease the level of dobutamine is based upon both clinical and radiographic examinations. In the current study, no attempt was made to equate patient response with the achievement of specific cardiac parameters. However, a suggested goal is to maintain the CI above 5.0 liter/min/sq m and specifically to achieve a CI of 6.0 liter/min/sq m, given that the majority of patients who responded to dobutamine maintained a CI above 5.0 liter/min/sq m.

It is interesting to note that, whereas in all patients undergoing pretreatment transcranial Doppler ultrasound (TCD) studies there were increased velocities indicative of spasm (peak velocity 200 cm/sec and mean velocity 120 cm/sec in the middle cerebral artery), in no patient was a fall in TCD velocity helpful in determining an endpoint to therapy. In fact, all patients had paradoxical elevations in TCD velocities during dobutamine therapy despite a clinical response. A prospective study comparing dobutamine and TCD velocities is currently underway.

Discussion

The purpose of the current study was to devise a therapeutic option that could provide for the reversal of clinical vasospasm refractory to hypervolemic treatment. The choice of a beta-agonist was based upon the concept that enhancing cardiac output and pulsatility of blood flow would result in increased perfusion to a dysautoregulating brain without the need to resort to severe degrees of systolic hypertension. In addition, we decided upon the agent that was associated with the fewest potential cardiac or systemic side effects.

Relationship of Cerebral Blood Flow to Cardiac Output

Using an animal model, Davis and Sundt were able to maintain stable arterial blood pressures while decreasing cardiac output. They demonstrated a significant decrease in cerebral blood flow (CBF) despite the maintenance of stable MABP's, thus suggesting a critical link between cardiac function and CBF in ischemic areas of brain. Keller, et al., supported this concept using a stroke model, demonstrating that increased cardiac output could improve microcirculatory flow without changes in MABP or blood viscosity. Tranmer, et al., and others, working with primate models of ischemia, have documented a profound loss of regulatory control in ischemic brain in response to alterations in cardiac output. They evaluated the effect of volume expansion with colloid and exsanguination to baseline cardiac output on local CBF, and found that local CBF in regions of ischemic brain varied directly with cardiac output, whereas flow in nonischemic brain was not affected by changes in cardiac output (p < 0.001). This suggests that variations in blood volume following expansion with colloid or exsanguination may cause significant changes in the intensity of ischemia.

Dobutamine Therapy

From the above discussion and numerous clinical observations, it is likely that a complex relationship exists between cardiac output and cerebral perfusion over and above the simple expedient of cerebral perfusion pressure. In addition, the pulsatility of blood flow has been reported to contribute significantly to cerebral perfusion in dysautoregulating brain. The beta-agonist dobutamine has been shown to increase the pulsatility of blood flow in addition to markedly increasing cardiac output. It has been reported that dobutamine caused a significant reduction in the pre-ejection period (when all valves are shut and the tension develops rapidly until ventricular pressure exceeds arterial pressure and the semilunar valves open), in the duration of the electromechanical systole (ventricular contraction), and in the ratio of the pre-ejection period to the left ventricular ejection time. Thus, the increase in contractility in conjunction with afterload reduction allows for an increase in pulse pressure.

The use of dobutamine in the postoperative care of patients following cardiothoracic surgery or in the presence of cardiac failure has been widespread for over a decade. Dobutamine is a synthetic sympathomimetic amine that functions as a potent inotrope by stimulating beta-1 receptors in the myocardium. Stimulation of peripheral beta-2 receptors (which overwhims its minimal alpha effects) and reflexive responses to the increases in cardiac output result in vasodilatation and afterload reduction. The net hemodynamic effects of dobutamine are similar to those in combination therapy with dopamine and a vasodilator. Thus, a reduction in the patient's peripheral vascular resistance following the initiation of dobutamine treatment is expected and the addition of agents such as alpha-agonists are not indicated. We believe that cardiac output and pulsatility are important determinants of flow to the dysautoregulating brain.

The use of pressors including dopamine and isoproterenol in the treatment of vasospasm following SAH is well documented in the literature. Although the assumption was made that the mechanism of symptomatic vasospasm reversal in these latter studies was based upon relaxation of spastic arterial smooth muscle cells, in fact, the more likely operant factors were increases in cardiac output secondary to the car-
Cardiac stimulant effect of these agents and the intravenous volume administered to maintain steady-state levels with an attendant increase in cerebral perfusion pressure. Despite the in vitro response of cerebral vessels to sympathomimetic amines including isoproterenol and salbutamol, no clinical studies to date have supported any clinically relevant response resulting from their use based upon direct interactions with beta-adrenergic receptors on cerebral vessels and resulting vasodilatation.21

In the current protocol, dobutamine treatment resulted in a mean 20% increase in heart rate. The mild increase in heart rate (as opposed to the marked tachycardia associated with isoproterenol therapy) is caused by the increased coronary blood flow that accompanies dobutamine's positive inotropic effect. The beneficial effects of dobutamine on hemodynamics and the lack of induction of endogenous norepinephrine following its use will minimize any potential negative effects on myocardial oxygen demand as compared to dopamine treatment.13 Thus, cardiac function can be maximized while avoiding ischemic cardiac injury in patients with either normal or compromised cardiac function. Finally, the potential arrhythmogenicity of isoproterenol at high infusion rates is avoided with the use of dobutamine (Table 3).3

The current study evaluates the clinical manifestations of cerebral vasospasm and the potential reversal of vasospasm. Future efforts are being directed at evaluating responses in CBF to both ischemic and nonischemic regions following the initiation of therapy as determined by positron emission tomography. In addition, changes in velocity in response to treatment as determined by TCD studies are also being pursued. We believe that with careful observation of cardiac parameters in addition to proper fluid management, the complications of fluid overload, congestive heart failure, and cardiac ischemia can be avoided.

Potential Complications Associated With Hypervolemia

In a review of 47 patients treated with prophylactic hypervolemia, Medlock, et al.,17 reported an unacceptably high complication rate associated with hypervolemia. Sixteen patients developed pulmonary edema and one died. The results of this study emphasize the importance of critical and frequent monitoring during therapy that has a profound impact on cardiac performance. The mere fact of having a Swan-Ganz catheter in place is not a guarantee against volume overload. Continuous hourly vigilance with appropriate modification of infusion rates may be necessary to avoid such complications.

A recent study by Shimoda, et al.,30 also suggested that hypervolemia therapy is contraindicated in patients found to have evidence on CT of edema and/or infarction at the time when a delayed ischemic deficit is manifested. They reported on a series of 112 patients with delayed ischemic deficits after SAH. Of 94 patients treated with hypervolemia, 18 (19%) had exacerbation of cerebral edema and eight (9%) developed a hemorrhagic infarction. This further underscores the importance of timely intervention (before infarction occurs), of decision-making with regard to how long to continue such powerful modalities, and of careful patient selection. Nothing significant will likely be gained by treating a patient with a fixed neurological deficit and a pre-existing infarct identified on imaging studies. Similarly, in patients who fail to respond to maximum therapy, continuing treatment longer than 6 to 12 hours will probably result in exacerbating cerebral complications.

We have not used dextran for several years because of its anticoagulant properties. The hemorrhagic sequelae found by Shimoda, et al.,30 may be related to this factor. We have not noted intracerebral hemorrhage as a sequel to hyperdynamic therapy. The cardiac parameters reported in the Shimoda series during “conventional hypervolemic therapy” (8 to 12 mm Hg) would not be consistent with maximization of cardiac performance. In addition, we have abandoned the use of CVP monitoring in our patients.15

It should be emphasized that the management of these patients is an ever-changing process based upon both the clinical and radiographic examinations. Any change in either will necessitate rapid manipulations of the hyperdynamic protocol by the clinician to optimize outcome.

Conclusions

In this study, we reported a 52% increase in CI in hyperdynamic therapy with dobutamine in the presence of volume loading, with only minimal increases (11%) in blood pressure. Hyperdynamic therapy with dobutamine in the presence of volume loading resulted in marked increases in cardiac function in addition to reflexive decreases in total peripheral resistance. Hyperdynamic-hypervolemic therapy clinically reversed SAH-induced vasospasm in 78% of patients failing to respond to volume expansion alone.

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Use of dobutamine in cerebral vasospasm

References