Letters to the Editor

Major Embolic Complications of Open Heart Surgery and DDA

To the Editor:
I read with interest the article by Furlan & Breuer on "Central Nervous System Complications of Open Heart Surgery". DDA is a variant of atherosclerosis which includes patients with "brittle aortas" wherein showers of cholesterol emboli repeatedly occur spontaneously or from trauma. I believe it is possible that a relatively high percentage of patients who suffered major embolic complications from open heart surgery had DDA. It would be of interest to know whether there was any evidence for a preponderance of this disorder pre-operatively in patients who had post-operative CNS complications. Pre-surgical erythrocyte sedimentation rates, cranial CT scans showing small lacunar-type infarcts especially in the caudate nuclei and a history of ischemic disturbances including the gastrointestinal system might be considered presumptive evidence of pre-existing low-grade DDA. I agree with the author's search for defects in cerebral auto-regulation in such patients but perhaps also one should search for sensitive indicators of low-grade chronic cholesterol embolization pre-operatively in such patients. Moreover anticoagulation might be withheld or reduced in such patients because there is evidence that such patients have a greater risk of complications from anticoagulation.

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The previous letter was submitted to the authors and following is their reply.

To the Editor:

We agree with Dr. Coppeto that atheromatous embolization from the aorta is a potential risk during coronary artery bypass graft (CABG) surgery, but doubt that this risk can be quantitated pre-operatively. During CABG surgery atherosclerotic plaques are frequently seen in the aorta, although the aorta is considered "unclamplible" in only one patient per thousand (Golding LR: personal communication). Our surgeons avoid cannulating or clamping obviously diseased segments of aorta, yet an audible crunch is sometimes heard as the aortic cross-clamp is applied.

Thurbeck et al. speculated that turbulence proximal to an aortic cross-clamp would tend to "churn up" atheromatous material and predispose to embolism. How often this occurs during CABG surgery, and to the extent, remains unknown. Multiple atheromatous brain emboli are rarely found in large series of open heart surgery cases coming to autopsy. 1, 2 Subtle neuro-psychiatric disturbances after open heart surgery have been ascribed to microembolism (ie, <20 microns) of air, fat, particulate matter or platelet/thrombus clumps generated during cardiopulmonary bypass, but have not been linked with atheromatous emboli. 3 Atheromatous emboli tend to be larger (ie, >50 microns) and produce multiple cerebral infarcts, often in a watershed distribution. 4, 5 We, and others, have documented multiple atheromatous emboli ("embolic shower") from the aorta as one cause of coma complicating open heart surgery. It is much more difficult to demonstrate the source of a single atheromatous embolus since these patients often have widespread atherosclerotic disease.

Sincerely,
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References

Monosialoganglioside Therapy in Stroke

To the Editor:

Since the neurons of the Central Nervous System (CNS) are not able to regenerate after injury, it is a widely accepted opinion that until now the therapy of acute cerebral stroke should aim to maintain the most favourable environment for the cells surviving in the area surrounding the core of the lesioned tissue. 1 However, an increasing number of experimental data demonstrates that the mature CNS has a potential capacity for a structural reorganization and for a functional recovery following focal brain damage. 2-3 These processes seem to be promoted by brain gangliosides, natural components of cellular membranes, which play a role in various neuronal regenerative events. 4,5

On this line, the possibility has been emphasized that chronic administration of brain gangliosides may be useful for the treatment of brain injury and degenerative disorders also in humans. 6, 7 We present here the effects of monosialotetrahexosylganglioside-sodium salt (GM1), a component of the ganglioside series, on the clinical course of patients affected by cerebral stroke of haemorrhagic or ischaemic nature.

Thirty-eight patients (24 men and 14 women) participated in our study. The subjects, or their relatives, gave informed consent to the double-blind placebo trial and were allocated at random into two groups of 19 patients each. The study started 12-15 days after the onset of neurological deficits, when anti-edema therapy had already been stopped. The first group (mean age ± S.D. = 60.6 ± 6.3 years) was treated with GM1 injected i.m. at the dose of 20 mg twice a day, for six weeks. The compound GM1 we employed is extracted from bovine brain by Fidia Research Laboratories (Abano Terme, Italy). In this group 4 patients did not complete the trial: I subject did not keep the treatment schedule, the other 3 worsened and were treated again with corticosteroid therapy. The second group (mean age ± S.D. = 61.4 ± 8.4 years) received placebo, a saline solution injected i.m. in the same way as the active drug, twice a day for six weeks. In this group 15 subjects completed the trial, 3 died and 1 worsened and therefore underwent anti-edema treatment.

At the beginning and at the end of the study, all patients underwent physical, neurological and laboratory examinations (including EEG and brain CT scan) and were also tested for seric antiganglioside antibodies. Neurological deficits were evaluated every week and were scored from 0 (death) to 100 (normal), according to indications of Mathew et al., modified by Fritz and Werner. 8 During the trial the patients underwent an individualized physiotherapy.
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J R Coppeto

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