

Prevention of neurological damage during open-heart surgery

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Branthwaite, M. A. (1975). *Thorax*, 30, 258–261. **Prevention of neurological damage during open-heart surgery.** A previous study of neurological damage related to open-heart surgery suggested that the onset of cardiopulmonary bypass is a time of particular hazard, and there is evidence that both microemboli from the extracorporeal circulation and inadequate cerebral perfusion may be contributory factors.

Measures to eliminate or minimize these hazards have been introduced, and a clinical survey has been undertaken to evaluate their efficacy. There has been a very highly significant decrease in the incidence of neurological damage as judged by comparison with the results of a similar survey carried out before these measures were introduced.

In spite of limitations imposed by differences in workload, perfusion techniques, and methods of data collection, it is concluded that the prophylactic measures have been responsible for the reduction in the incidence of neurological damage.

A previous clinical survey, based upon the retrospective analysis of the case notes of 417 patients submitted to open-heart surgery during 1970, revealed an incidence of neurological damage of 19.2% (Branthwaite, 1972). Evidence derived by monitoring cerebral function with a heavily filtered electroencephalograph (Cerebral Function Monitor, Devices Ltd) suggested that the onset of perfusion is a time of particular hazard (Branthwaite, 1973). Microemboli from the extracorporeal apparatus, and inadequate cerebral perfusion caused by an abrupt change in the character and magnitude of the perfusing pressure, could contribute to these findings, and there is experimental and clinical evidence which suggests that both factors may be of importance (Stone *et al.*, 1967; Hasbrouck and Rigor, 1969; Javid *et al.*, 1969; Patterson and Kessler, 1969; Hill *et al.*, 1970; Osborn *et al.*, 1970; Tufo, Ostfeld, and Shekelle, 1970; Egeblad *et al.*, 1972; Sanderson, Wright, and Sims, 1972; Branthwaite, 1974). Since 1970, filters for microemboli have been incorporated into the extracorporeal circuit used in this hospital both on the arterial line (Barrier filter—Johnson and Johnson), and on the coronary suction lines (Bromlus filter—Lusterlite Products Ltd.), and the hazards of sudden hypotension, particularly at the onset of bypass, have been recognized. Vaso-pressor agents are given if hypotension occurs

abruptly, particularly if it is associated with either depression on the cerebral function monitor (Figure) or dilatation of the pupils. A second survey was undertaken during 1973 to evaluate the efficacy of these measures.

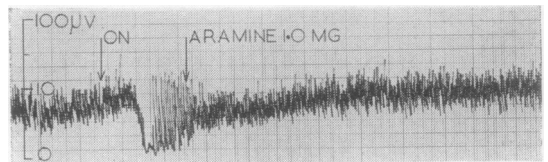


FIGURE Record obtained from the cerebral function monitor at the onset of perfusion in a 43-year-old woman with mild mitral stenosis and hypertension (160/100 mmHg). The arterial blood pressure fell to between 30 and 40 mmHg when the aorta was cross-clamped, in spite of a flow of 80 ml/kg per minute from the oxygenator. The resulting cerebral depression was reversed rapidly by the administration of metaraminol (Aramine), and the patient regained consciousness after the operation without any evidence of neurological deficit. Paper speed—6 mm/min.

MATERIAL AND METHODS

The case notes of all patients submitted to open-heart surgery during 1973 were inspected. The data were collected retrospectively to preserve

comparability with the methods used in 1970, and the same criteria were used to define neurological damage. These criteria were impairment of consciousness, voluntary movement or vision, separately or together, apparent within three days of operation. Impaired consciousness ranged from coma to confusion of a severity sufficient to interfere with response to simple commands, but confusion alone in patients who were hypoxic postoperatively (arterial oxygen tension less than 60 mmHg) was discounted.

The factors considered in relation to neurological damage in 1973 are listed (Table I). Information on one or more of these factors was unavailable in 23 patients, and these were excluded from the statistical analysis. The collection of data was complete for all patients who suffered neurological damage.

TABLE I
FACTORS CONSIDERED IN RELATION TO INCIDENCE OF NEUROLOGICAL DAMAGE

Age
Duration of perfusion
Preoperative neurological status
Nature of operation
History of previous intracardiac surgery
Type of oxygenator
Nature of priming solution

The association between the factors listed in Table I and the incidence of neurological damage was analysed by multiple regression analysis so that the influence of interrelationship between some of the variables could be eliminated.

RESULTS

Neurological damage was sustained by 40 of the 538 patients who survived the operative procedure (Table II). The incidence of 7.4% compares with a corresponding figure of 19.2% in 1970, and this difference is very highly significant ($P < 0.001$). Nineteen of the 40 patients died in the post-operative period, and the neurological lesion was

TABLE II
NATURE OF NEUROLOGICAL DAMAGE SUSTAINED IN 1973

Hemi- or mono-plegia or paresis	14	(36)
Coma ¹	16	(20)
Confusion	10	(22)
Quadriplegia	1	(1)
Epilepsy	1	(2)
Midbrain lesion	—	(2)

Figures in parentheses are the comparable values for 1970.

¹Three patients in 1970 and two in 1973 had localizing signs of a hemiplegia, as well as coma.

wholly or partly responsible in 10 cases. Twelve of the 21 patients with neurological damage who survived to leave hospital were left with residual disability of varying severity at the time of their discharge.

Probable aetiological events were suspected on clinical grounds in 22 of the patients who sustained neurological damage in 1973 (Table III) and nine of these patients were aged 5 or less. The incidence of neurological damage which could not be attributed to any specific feature of the operative period fell from 11.5% (48 of 417 cases) in 1970 to 3.4% (18 of 538 cases) in 1973 ($P < 0.001$).

TABLE III
SUSPECTED AETIOLOGY OF NEUROLOGICAL DAMAGE IN 22 PATIENTS

Hypotension, hypoxia or cardiac arrest	15
Suspected embolism of clot or air	2
Hypernatraemia ¹	2
'Vertebral steal' syndrome ²	1
Uraemia	1
Cerebral abscess	1

	22 Cases Aetiology suspected	18 Cases Aetiology unknown	Mean Diff. ± SE of Mean	Significance
Age (yr)	30.0	53.1	23.1 ± 7.0	< 0.01
Duration of bypass (min)	134.3	116.6	17.7 ± 25.1	> 0.1

¹Complicating inadequate removal of salt crystals from the membrane oxygenator.

²During resection of a thoracic aortic aneurysm.

Those factors which were found to be positively associated with an increased incidence of neurological damage in 1970 continued to feature in 1973 (Table IV). These were age, increasing duration of perfusion, and the presence of a previous neurological lesion. Use of an all-blood prime appeared as an additional contributory factor in 1973, but neither the nature of the operation nor a history of previous cardiac surgery were important. There was a quadratic relationship between age and neurological damage

TABLE IV
FACTORS ASSOCIATED WITH NEUROLOGICAL DAMAGE: RESULTS OF MULTIPLE REGRESSION ANALYSIS

	Variance Ratio	Significance
Duration of perfusion	27.3	< 0.01
Preoperative neurological abnormality	10.0	< 0.01
Use of an all-blood prime	9.4	< 0.01
Age ¹	4.7	< 0.05

¹There was a quadratic relationship between age and neurological damage.

in 1973, probably accounted for by the higher incidence of specific complications in the very young.

DISCUSSION

Analysis of the results obtained in 1970 emphasized the importance of perfusion in the aetiology of neurological damage. Since that time prophylactic measures have been introduced and there has been a very highly significant decrease in the incidence of neurological damage. Before it can be concluded that these events are causally related, it is necessary to explore the validity of the comparison between the results in 1970 and in 1973.

There have been a number of changes in the nature of the work and the techniques of perfusion between 1970 and 1973 (Table V). The type of operative procedure was unrelated to the occurrence of neurological damage in their series, and the choice of oxygenator was also unimportant, apart from an apparently favourable influence of perfusion with the Temptrol in 1970. The virtual exclusion of this oxygenator during 1973 would be expected to enhance, rather than diminish, the incidence of neurological damage, and it is regarded therefore as an unimportant difference between the two years.

Use of an all-blood prime was an unfavourable feature in 1973, although the nature of the priming solution did not influence the neurological outcome in 1970. More patients in 1970 were subjected to perfusion from apparatus which had been primed with whole blood, but the number involved (59) was relatively small. It is highly un-

likely therefore that any inapparent, adverse effect derived from the use of an all-blood prime in 1970 could be responsible for more than a very small proportion of the excessive incidence of neurological damage in that year.

Much greater uncertainty surrounds the accuracy of the data collection. The information was derived retrospectively from the case notes and these had been completed by different staff. It is quite possible that greater awareness of the problem of neurological damage in 1970 favoured more accurate and detailed assessment, and comments on neurological status appeared more commonly in the notes in 1970. It is particularly likely that minor degrees of dysfunction were not recorded in 1973, although the similarity in the recorded incidence of confusion in patients with neurological damage (27.5% in 1970; 25% in 1973) does not support this possibility. The other lesions all represent major postoperative complications and it is likely they would be recorded. Furthermore, even if patients with confusion as the sole index of neurological dysfunction are excluded from the comparison, there is still a very highly significant decrease in the incidence of neurological damage, from 13.9% in 1970 to 5.6% in 1973 ($P < 0.001$).

This analysis suggests that none of the differences in the work, perfusion technique or data collection was sufficient to account for the reduction in the incidence of neurological damage between 1970 and 1973. It must be concluded therefore that the introduction of prophylactic measures has been responsible for the improved results. Unexpected neurological damage has not been eliminated entirely and there can be no room for complacency until this ideal has been achieved.

I am grateful to the surgeons of the Brompton Hospital for permission to study patients under their care, and to Miss C. M. Devine who kindly provided statistical assistance.

The study has been supported by a grant from the Medical Research Council, and the results form part of a thesis submitted for the degree of MD (Cantab).

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TABLE V
COMPARISON OF THE WORK AND PERFUSION
TECHNIQUES IN 1970 AND 1973

	1970	1973
<i>Operative procedure</i>		
Mitral valve surgery	142	140
Aorta and aortic valve surgery	115	111
Multiple valve replacement	31	94
Left to right shunts	56	84
Right to left shunts	39	37
Relief of right heart obstruction	16	9
Ischaemic heart disease	10	44
Miscellaneous	8	19
<i>Oxygenator</i>		
Rotating disc	326	428
Rygg	56	82
Temptrol	30	4
Membrane	0	20
<i>Nature of priming solution</i>		
All blood	59	20
Full haemodilution	39	344
Mixture of blood and electrolyte solution	288	155

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