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ORIGINAL ARTICLE

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Cerebral cast angiography as an aid to medicolegal autopsies in cases of death after adult cardiac surgery

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Abstract Due to an increase in age of the patient population in cardiac surgery, cerebral complications are increasing in frequency, also as a cause of death. In order to reveal cerebral pathology associated with a fatal outcome after cardiac surgery, we re-evaluated the cast angiographs and medico-legal autopsy documents of 144 adult cardiac surgery subjects over a 7-year period. Special attention was paid to the ability of post-mortem cast angiography to aid in diagnosing cerebral pathology. The autopsy detected new ischemic cerebral lesions in 29 (20%) cases, of which 22 (15.3%) were recent infarcts, and 7 were cases of anoxic brain damage. Of the recent cerebral infarcts, 12 were associated with cerebral artery thrombosis, 4 showed multiple lesions, and the remaining 6 were small single infarcts. In addition, one subject had an intracerebral hemorrhage and 72 (50%) cerebral edema. By cast angiography, the leakage of contrast medium in the case of intracerebral hemorrhage and stenoses of intracranial and cervical arteries could be well demonstrated and also revealed 17 (77%) of the 22 recent cerebral infarcts. It was found to be suitable for detecting recent brain infarcts associated with main cerebral artery thrombosis, with a sensitivity of 92% (11 out of 12 cases), but was less sensitive in showing small recent infarcts with a sensitivity of 60% (6 out of 10 cases) and inferior for the older ones where none of the 6 cases were detected. Filling defects caused by cerebral edema were difficult to differentiate from technical errors and were encountered in 7 (4.8%) cases. A significant predictor for the 29 recent ischemic brain lesions was perioperative hypotension. The immediate cause of death was most often of cardiac (83%) and cerebral (14%) origin. In 14 cases, cerebral damage was considered to be an additional cause of death. The use of cerebral post-mortem cast angiography should be recommended, especially for its excellent ability to visualize intravascular pathology such as arterial stenoses and thromboses, with a 92% sensitivity in showing new main cerebral artery thromboses, before likely distortion of the vascular anatomy by dissection.

Key words Post-mortem · Cast angiography · Cardiac surgery · Cerebral complications

Introduction

Mortality associated with cerebral complications after cardiac surgery has risen in recent years, whereby the proportion of neurological deaths rose from 7.2% at the start of the 1970s to 19.6% in the 1980s [1]. Most detailed neuropathological autopsy studies of cardiac surgery patients were undertaken in the 1960s [2]. The autopsy studies carried on in the 1980s and 1990s were mostly aimed at discovering some special neuropathology findings such as atheroembolization, boundary zone lesions, antifoam agents, or small capillary-arteriolar dilatations [3, 4, 5, 6, 7, 8, 9].

Post-mortem angiography has been reported to be useful in routine medico-legal autopsy practice after neurosurgery, in assessing coronary by-pass grafts, and after vascular and abdominal surgery [10, 11, 12, 13]. Our aim was to test its utility in diagnosing cerebral pathology after fatal outcome of adult cardiac surgery, and at the same time to discover what kind of cerebral lesions are encountered in those deaths due to cardiac surgery.

Materials and methods

In the vicinity of the Department of Forensic Medicine of Helsinki University there are three hospitals (Helsinki University Central Hospital, the Mehiläinen Hospital, and the Deaconess Hospital) performing adult cardiac surgery. Those deaths occurring in connection with cardiac surgery routinely undergo a medico-legal autopsy. These hospitals performed 9,223 cardiac operations during the study period from 1989 to 1995. The mortality rate for cardiac surgery in

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Table 1 Annual rates for cardiac operations, and autopsies and cerebral cast angiographies performed

Year	Cardiac operations *(n)*	Autopsies after cardiac operations *(n)*	Cast angiographs (*n*% autopsies)	Cerebral complications *(n)* of those with cast angiographs (%)
1989	1157	39	19 (49)	5 (26)
1990	1080	33	19 (58)	5 (26)
1991	1049	36	24 (67)	3 (13)
1992	1254	38	15 (39)	4 (27)
1993	1507	52	16 (31)	5 (31)
1994	1574	48	25 (52)	6 (24)
1995	1602	39	26 (67)	2 (8)
Total	9223	285	144 (51)	30 (21)

these hospitals averaged 3% and remained stable (2.4–3.5%) during the study period (Table 1). This study was based on those 144 patients who died within 30 days of adult cardiac surgery or during the same hospitalization period and where cerebral post-mortem cast angiography was done in conjunction with the medico-legal autopsy. The mean age of the patients was 64.7 years. The operation was an emergency in 17, and urgent in 37. Of the patients, 21 (15%) died immediately in the operating room. For the type of cardiac operation see Table 2.

Cerebral cast angiography is a technique routinely used in our department of forensic medicine, although the inadequate number of the staff sometimes limits its use. The annual cerebral cast angiography rates compared with yearly cardiac operation and autopsy rates are shown in Table 1. The exact technique of post-mortem cast angiography is described in detail elsewhere [10, 13]. Briefly, after the aorta was cannulated and clamped distal to the left subclavian artery, silicon vulcanized at room-temperature and made radiopaque with lead oxide as a contrast medium, was infused at a pressure of 150 mmHg for 30 min. X-ray images were then taken, using anteroposterior and lateral projections for visualizing cerebral and cervical vessels. The X-ray images were then taken with the brain separated from the cranium. There were anteroposterior and lateral projections from 91 subjects, and 128 X-ray images of the separated brains were available.

After the cast angiography, the autopsy was carried out according to the normal medico-legal autopsy practice. The areas of the brain appearing grossly pathological or showing incomplete filling on the cast angiography were taken for histology, fixed in formalin, and stained with Weikert van Gieson and Giemsa stains.

The cast angiographs were first studied without the aid of the autopsy documents, in order to estimate the ability of cast angiography alone to reveal the cerebral pathology, and thereafter restudied with use of the autopsy documents. The technical adequacy of the cast angiography was evaluated by recording the air bubbles, massive amounts of air, and the overall filling; thereafter recording the stenoses and incomplete filling of the cerebral and neck arteries and the cerebral filling defects. The posterior cerebral circulation was evaluated by recording whether the posterior cerebral arteries arose from the anterior or the posterior circulation and recording the development of posterior communicating arteries. Hypoplasia of the posterior communicating arteries was the diagnosis when their size corresponded to the size of the most peripheral arteries on the angiography, or they did not exist at all.

With the autopsy documents at hand the contrast filling defects of brain tissue were categorized in each case according to the main

Table 2 Type of cardiac surgery and immediate cause of death in 144 surgical deaths (*CABG* coronary artery bypass grafting, *AVR* aortic valve replacement, *MVR* mitral valve replacement, *VSR* ventricular septal rupture)

Operation	*n*	Cause of death				
		Cardiac	Cerebral	Pulmonary	Hemorrhage	Mediastinitis
Primary CABG[a]	104	87	10	6	1	1
Redo CABG	11	9	2	0	0	0
AVR + CABG	9	7	2	0	0	0
MVR + CABG	7	7	0	0	0	0
AVR + MVR +/- CABG	3	2	1	0	0	0
VSR seclusion	3	2	0	0	1	0
Composite graft[a]	3	2	2	0	0	0
Others	4	3	0	0	0	1
Total	144	119	17	6	2	2

[a] Cardiac and cerebral causes conjoining in one case

Table 3 Variables associated with cerebral ischemic lesions in 144 autopsy subjects (*PCo* posterior communicating artery, *PCA* posterior cerebral artery, *ICA* internal carotid artery, *MCA* middle cerebral artery, *LVAD* left ventricular assist device)

Variable	Ischemic lesions (present/not present)	*P*
Absence or hypoplasia of PCo[a]	6/27	1
PCA from MCA/ICA[a]	2/18	0.360
Vertebral/basilar artery stenosis > 50%	2/7	0.687
ICA/MCA stenosis > 50%[a]	2/18	0.112
Ascending aortic atheroma	9/50	0.392
Emergency operation	2/15	0.527
Perioperative hypotension	10/71	0.0193*
Intra-cavity thrombus	0/3	1
Intra-aortic counterpulsation/LVAD	11/68	0.090
Perioperative myocardial infarction	20/76	0.658
Survival time after operation (hours ± SD)	189 ± 174/86 ± 148	0.007*
Recovery from anesthesia after 2 days[c]	10/14	0.196

[a] Of those having radiographs of separated brains
[b] Of those surviving the first postoperative day
[c] Of those surviving the second postoperative day
* Significant variable

pathology at autopsy, such as cerebral infarction, intracerebral hemorrhage, hypoperfusion, or edematous lesion, or as technical failures when present. A cerebral infarct was defined as a focal cerebral lesion and confirmed as an infarct by histology. Intracerebral hemorrhage was defined as a localized macroscopic hemorrhage. Cerebral edema was the category when the cerebral sulci were obstructed, when there was a "string sign" in the cerebellum, or when the histology revealed widened perivascular spaces. The cases with no focal lesions, but reddish discoloration of the cerebral matter and histology showing degeneration of cortical ganglion cells and capillary walls, were considered to be anoxic brain damage.

Some clinically interesting variables (Table 3) were tested for their association with cerebral pathology with the Fisher exact test for nominal data and Student's *t*-test for continuous data. In any paired system, if both brain lobes or both vessels had pathology findings, they were considered together for each individual case. The status of the posterior communicating arteries and the origin of the posterior cerebral arteries from the anterior circulation, were studied only when the angiograms of the brain separated from the cranium were available, and accordingly, extracranial carotid arteries only when X-ray images of cervical vessels were available. In studying the association of recovery from anesthesia with fatal ischemic brain lesions, recovery from anesthesia 2 days after the operation was considered to be delayed. In this situation the reference group included only those surviving until this point in time.

Results

Cast angiography findings

Areas of poor contrast filling were encountered in the angiograms from 54 subjects (37.5%). In 7 cases (4.8%), the angiograms had failed technically either because of improper filling or excessive air, yielding angiograms for 47 subjects for further study. In 15 the filling defects were associated with incomplete filling of one or more of the main cerebral arteries and in 1 case of the common and the internal carotid arteries. The cerebral artery most often involved was the middle cerebral artery (14 cases) followed by the posterior and the anterior cerebral arteries, each in 5 cases, and the basilar artery in 2 cases (Table 4). In 21 of the remaining 31 cases without incomplete filling of the cerebral or cervical arteries, the defects were bilateral or were situated both in the anterior and posterior parts of the brain while they were more localized in 10 cases.

In addition to the lack of poor filling, 38 subjects had significant (> 50%) stenoses of one or more cerebral or cervical arteries and 41 less severe (< 50%) stenoses (Table 4). Vertebral arterial hypoplasia existed in 10 cases. Poorly developed or undeveloped posterior communicating arteries were found in 36 (28%) of those 128 where angioradiographs of the separated brain were available.

Autopsy findings

Ischemic cerebral lesions were evident in 34 of the autopsy cases, with 22 recent cerebral infarcts, 6 older ones and 7 with anoxic brain damage, in 1 case conjoining with an old infarct. Of the 22 subjects with recent cerebral infarcts, 12 were associated with the main cerebral artery thromboses, 4 showed multiple lesions, and the other 6 were small and single. The posterior parts of the brain were involved only or in addition to the anterior ones in 9 (41%) subjects. Only one subject had an intracerebral hemorrhage.

Brain edema was found in 72 (50%) of the subjects, of which 12 had associated recent cerebral infarcts, 6 anoxic brain damage, and 1 intracerebral hemorrhage, with brain edema as the only cerebral pathology in 53.

Post-mortem clots were found in cerebral arteries of two cases, in one with an ante-mortem thrombosis and a cerebral infarct of the opposite side, and in the other with cerebral edema.

Cast angiography findings compared to autopsy

After comparison with the autopsy documents, the angiographic filling defects were judged according to their main pathology as representing cerebral infarcts in 17 cases, poor filling caused by increased intracranial pressure in 26 cases whereas cerebral edema was present at autopsy, post-mortem clot in the middle cerebral artery in 1 case, middle cerebral artery thrombosis without infarction in 1 case, cerebral hypoperfusion caused by common and internal carotid artery occlusion in 1 case, and intracerebral hemorrhage in 1 case (Table 5). In five cases the intravascular thrombus had propagated from the internal carotid artery to the middle cerebral artery, and in four of these also to the anterior cerebral artery. In addition, there were seven other middle cerebral artery thromboses, two of them without an accompanying infarct and two with an accompanying internal carotid artery stenosis. One case had a post-mortem thrombosis of the middle cerebral artery conjoining with the ante-mortem thrombosis of the middle cerebral artery of the opposite side. The basilar and

Table 4 Pathology of the main cerebral arteries on cast angiography of 144 subjects compared with autopsy findings

Artery	Incomplete filling on cast angiography	Autopsy finding of thrombosis			Stenosis > 50% or hypoplasia on cast angiography	Stenosis < 50% on cast angiography
		Ante-mortem	Post-mortem	Others		
Internal carotid	7	7	0	0	14	8
Middle cerebral	14	11	2[a]	2	3	9
Anterior cerebral	5	4	0	1	0	2
Posterior cerebral	5	4	0	1	2	9
Basilar	2	2	0	0	3	18
Vertebral	3	2	0	0	16	19

[a] In one conjoining with ante-mortem thrombosis on the opposite side

Table 5 Cerebral cast angiography filling defects compared to the main autopsy findings in 144 subjects

Autopsy findings *(n)*	Cast angiography filling defect (n = 47)		
	Main cerebral artery	Normal filling of cerebral arteries	Total
Recent cerebral infarct (22)	11	6	17
Cerebral edema (53)	1	25	26
Intracerebral hemorrhage (1)	1	0	1
Hypoperfusion without edema (2)	1	1	2
Postmortem thrombosis (1)	1	0	1
Total	15	32	47

posterior cerebral artery thromboses (four cases) were always associated with a cerebral infarct. Two patients had carotid artery occlusions without infarction.

Cast angiography gave good visualization of recent infarcts with a main cerebral artery thrombosis (92%) missing in only 1 poorly performed case out of 12 (Figs. 1, 2, 3), but it was less suited for other recent infarcts, of which only 6 out of 10 showed consistent changes. The angiographic defects tended to be localized in cerebral infarcts (only two presenting with multiple bilateral defects) than in those with technical errors or brain edema; in the for-

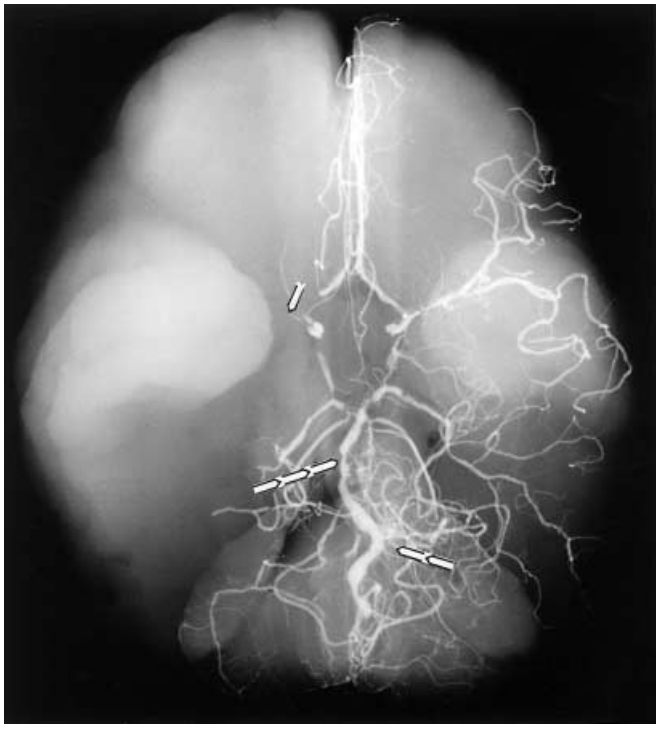

Fig. 2 Post-mortem cast angiography of a 70-year-old subject with thrombosis of right middle cerebral artery (⇐) after aortic valve replacement and coronary artery bypass grafting showing massive filling defect of the right fronto-parieto-temporal area of the brain with hypoplasia of the left vertebral artery (⇐⇐) and atherosclerotic changes in the basilar artery (⇐⇐⇐)

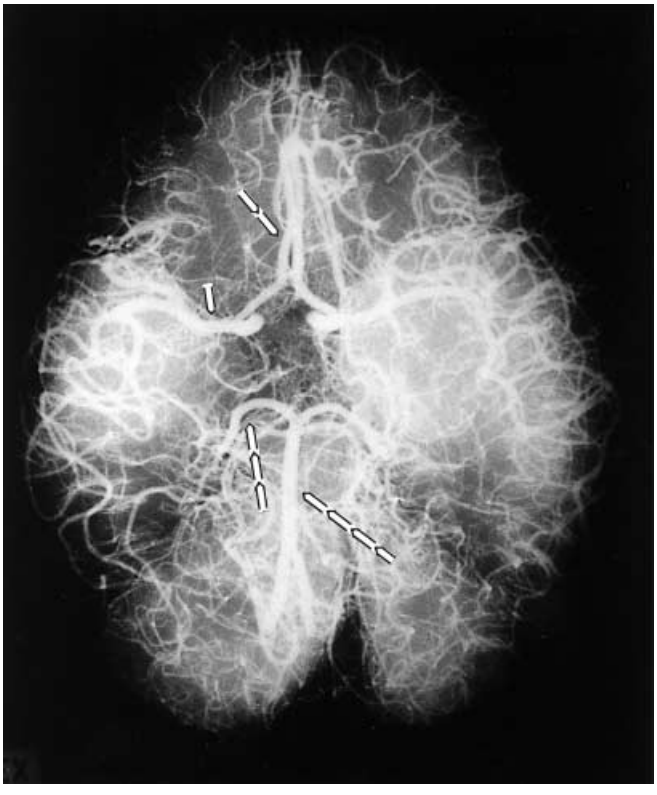

Fig. 1 Normal cerebral anatomy on post-mortem cast angiography of a 50-year-old subject. (⇐ middle cerebral artery, ⇐⇐ anterior cerebral artery, ⇐⇐⇐ posterior cerebral artery, ⇐⇐⇐⇐ basilar artery)

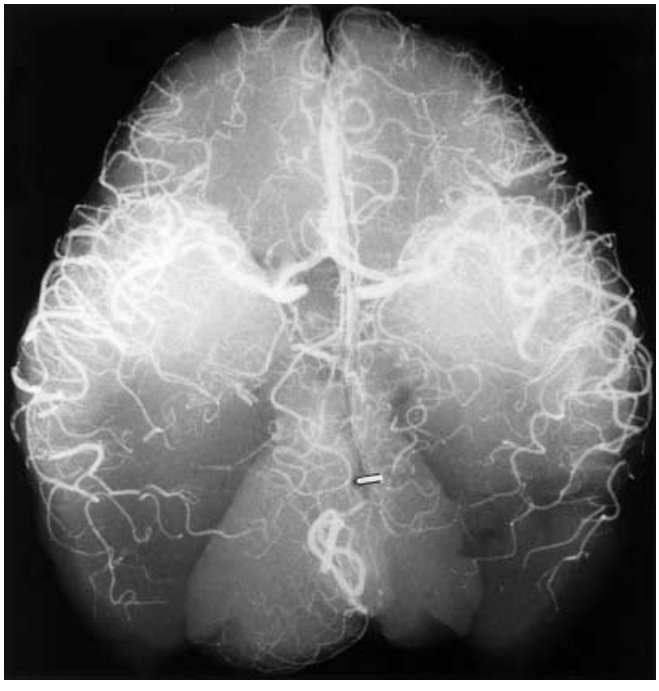

Fig. 3 Post-mortem cast angiography of a 60-year-old subject with basilar artery thrombosis (⇐) after coronary artery bypass grafting with associated infarct of occipital lobes, cerebellum, and brain stem

mer all brain lobes were affected; in the latter, 23 of those 26 (88%) had bilateral defects or defects in both the anterior and posterior parts of the brain. In brief, the angiographic findings from technically incorrect contrast filling did not differ from that of poor filling caused by increased intracranial pressure, except in cases with technical failure caused by massive amounts of air. The filling defects caused by cerebral edema were most prevalent in the frontal lobes (11 cases), followed by temporoparietal areas (6 cases) and occipital areas (4 cases) which were general in 15 of the 26 subjects.

The angiograms failed to show any pathology in 44 out of 72 (61%) cases with brain edema and in 5 of the 22 (23%) having recent brain infarction. Of the six with old cerebral infarcts, four had no cast angiographic changes and two had unspecified changes inconsistent with the site of infarction. In 1 out of the 22 with recent cerebral infarction, the cast angiographic pathology was due to technically inadequate filling, although at autopsy there was thrombosis of the middle cerebral artery. In the angiograms of those five subjects with anoxic brain damage, no specific pathology was evident, although one had poor peripheral contrast filling associated with brain edema.

The variables listed in Table 3 were studied for association with recent ischemic cerebral lesions. The presence of recent cerebral infarction or anoxic cerebral damage was associated with perioperative hypotension ($P = 0.019$), but not with the main cerebral artery stenoses or with variations in the normal vascular anatomy, like poorly developed posterior communicating arteries or posterior cerebral arteries arising from the anterior circulation. (Fig. 4) The patients who died of recent ischemic cerebral lesions survived longer after the operation than did the other patients with fatal outcome ($P = 0.007$).

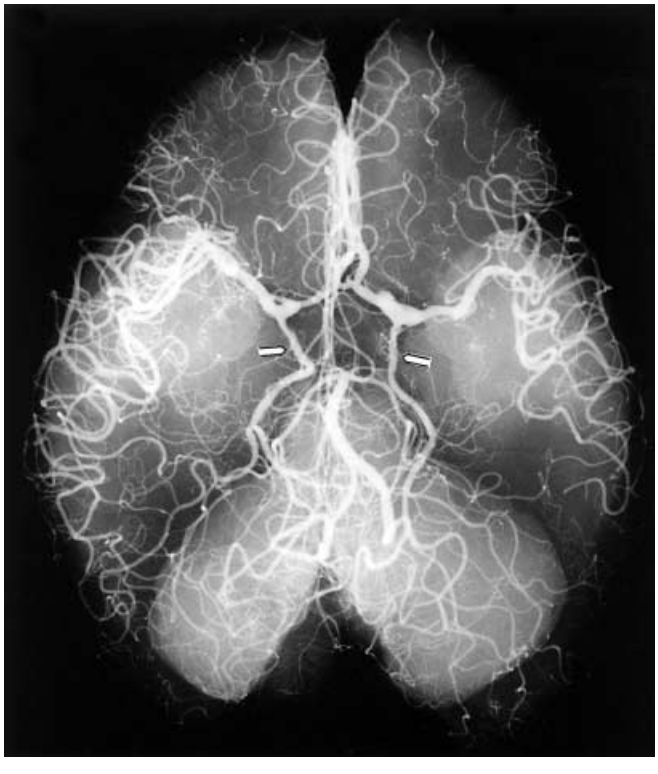

Fig. 4 The posterior cerebral arteries arising from the anterior circulation (⇐) in a post-mortem cast angiography of a 81-year-old subject

In most cases, the immediate cause of death was cardiac (119 cases), cerebral in 20, and in 14 the cerebral damage was a secondary cause (Table 1). Fresh myocardial infarction or myocardial failure co-existed with an ischemic cerebral lesion in 19 cases. In addition, there were three thromboses of the main cerebral arteries along with myocardial failure or infarction. Recent ischemic cerebral lesions were never found in those who died within the first 24 h of the operation, and of the 59 patients surviving the first 24 h but never gaining consciousness only 13 (22%) had such a lesion.

Discussion

The possible advantage of post-mortem angiography is its ability to locate possible cerebral lesions before the autopsy which may subsequently cause distortion of the vascular anatomy. As the cast angiography technique is part of the normal autopsy practice in our department of forensic medicine, it is not possible to judge retrospectively which of the pathology findings would have been missed without the actual cast angiography, because the forensic pathologists performed the final autopsies while aware of the cast angiography findings. However, one aim of this study was to investigate which types of cerebral pathology could be detected by the cast angiography.

Post-mortem cast angiography was a good method for detection of arterial stenoses and seemed to be well suited to detect recent cerebral infarcts associated with main cerebral artery thrombosis (11 out of 12), giving at the same time a clear view of the thrombus propagation from one vessel to another. Its ability to detect other new infarcts was poorer, with 60% presenting with angiographic pathology. These other infarcts seemed to be smaller, situated in the region of end arteries, such as in watershed and atheroembolous infarctions not causing defects large enough to be discerned.

However, cerebral cast angiography is an insufficient technique without an autopsy being carried out. Technical failures occurred in 4.8% of the angiographs compared with the 12.5% major failure rate of coronary cast angiography with the same technique [13]. Sometimes, however, the technical failures were difficult to discern. Small air bubbles were readily noticed, but improper contrast filling, resulting from large amounts of air or from inadequate infusion pressure of the contrast medium, made the interpretation difficult. As this study showed, increased intracranial pressure and technical errors gave very similar angiographic changes in many instances, showing general filling defects in all technically incorrect cases and in 79% of those with filling defects caused by increased intracranial pressure. In recent brain infarcts, the defects tended to be more localized (14 out of 17 with angiographic changes),

though 2 of the 3 with more generalized defects were not readily discernible from defects caused by cerebral edema. The inability of cast angiography to show any pathology in 27 of the 53 (51%) with brain edema as the only cerebral pathology, was probably due to the smaller increase in intracranial pressure in these cases. The necessity of autopsy was also seen in one subject with bilateral middle cerebral artery thrombosis with a post-mortem clot on the right side and ante-mortem thrombosis on the left, as well as in the case with incomplete filling of the region of the middle cerebral artery because of a post-mortem clot.

In a case of thrombosis of cerebral or cervical arteries, the underlying status of the vessel wall and the possible pre-existing stenoses were not always properly given in the autopsy document. This may be why we could find no association between ischemic lesions and haemodynamically significant cervical and cerebral artery stenoses.

In the present study, the proportion of cardiac deaths was higher (83%) than the 27%–59% cited in the literature [1, 14]. The proportion of our cerebral deaths (12%) corresponds well with the 9%–20% in the literature [1, 14], indicating that the cerebral complications were not very overrepresented in our material. [1, 14] However, it is possible that those with suspected cerebral complications had cerebral cast angiographies done more often when an inadequate number of technical staff was present. This can be suspected from the information in Table 2 where in the years 1991 and 1995 when the rate of cerebral cast angiography was at its highest (67% vs 31%–58%), the rate of cerebral complications was lower (8%–13%) than in the other years of the study.

The proportion of main cerebral artery thrombosis (12 out of the 22) in our study was unexpectedly high compared to that in the study of Malone et al. [4] of 20 patients who died after cardiac surgery where none had cerebral infarcts caused by main cerebral artery thrombosis. All nine infarcts were border zone in character. The incidence of anoxic brain damage defined as punctate cortical hemorrhage was 7.5% according to Herczeg et al. [15], but somewhat lower in this study at 4.9%. On the other hand, our incidence of cerebral hemorrhage was low, only 0.7% compared with the 2.8% found by Lee et al. [16] with 108 autopsy patients. Our low incidence of cerebral edema, 51%, compared to the 100% of Herczeg et al. [15] is probably due to the definition of cerebral edema, as in the study of Herczeg et al. the milder forms of edema were also included.

Although not all the non-survivors in our series underwent cerebral cast angiography, we believe that the distribution of various types of cerebral complications, at least clinically overt ones, is representative of the non-survivors of adult cardiac surgery, because in the case of inadequate ability, the angiography was probably done when a cerebral complication was suspected. Cerebral angiography was good at visualizing the intra-arterial stenoses and the distribution of blood in the intracranial arteries. It was especially suited for visualizing cerebral artery thromboses, revealing 92% of the total and giving at the same time a clear image of the thrombus propagation from one vessel to another. For this reason, we encourage the use of cerebral cast angiography as an aid in medico-legal autopsies before the vascular anatomy is distorted by dissection.

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