

Total vertebral artery flow volume, the 200 mL/min threshold and stroke after isolated coronary artery bypass grafting: a retrospective cohort study

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Abstract

Background.

Duplex ultrasonography measures two things: how narrow a vessel is, and how much blood flows through it. Screening before cardiac surgery uses only the first, in the carotid arteries. Vertebral artery flow volume is recorded but not used in any decision. Our reports compare the total value with a threshold of 200 mL/min. We tested whether both are associated with perioperative stroke. Of 1,377 patients who underwent cardiac surgery between January and August 2022, 915 had isolated on-pump coronary artery bypass grafting (CABG). Right and left vertebral flow volumes from the last preoperative duplex study were summed; both were available for 845 (92.3%).

Results.

Median total vertebral flow volume was 220 mL/min (interquartile range 200–252); the published reference mean is 182 mL/min. Two hundred and ten patients (24.9%) were below the 200 mL/min threshold, but only 15 (1.8%) were below the reference fifth percentile of 102.4 mL/min. Stroke occurred in 19 of 915 patients (2.1%), 17 of them with flow data. Flow volume was not associated with stroke: median 227 mL/min with stroke and 220 mL/min without ($p = 0.789$); adjusted odds ratio (OR) 1.01 per 10 mL/min (95% confidence interval [CI] 0.93–1.09); no difference across tertiles ($p = 0.513$); adjusted OR 0.97 (95% CI 0.31–3.01) below 200 mL/min. The study had 80% power to detect an OR of 3.2; penalised regression and two sensitivity analyses agreed. The posterior circulation was involved in 8 of the 19 strokes (42%). Carotid stenosis, measured in the same studies, was associated with stroke (1.6%, 6.7% and 7.4% by grade; exact $p = 0.007$), including after adjustment for age (adjusted OR 5.15, 95% CI 1.60–16.56).

Conclusions.

Total vertebral artery flow volume was not associated with stroke after isolated CABG. The 200 mL/min threshold used in our reports lies above the mean of a reference population; it marked one patient in four as abnormal and identified no increase in risk. A value below it is not by itself a reason to change the perioperative plan. With few events and almost no patients with genuinely low flow, these data do not settle whether a truly reduced posterior inflow carries risk.

Background

There are two ways to describe an extracranial artery. Stenosis describes how narrow the vessel is, and flow volume describes how much blood passes through it. Duplex ultrasonography measures both, but only stenosis is used for risk assessment. The difference between them is largest in the vertebral arteries, where hypoplasia and asymmetry are common and vessel calibre predicts flow poorly. These

arteries supply the medulla, midbrain, cerebellum, thalami and occipital cortex [1]. Low posterior circulation flow has been linked to atherosclerosis, hypertension, smoking and older age [2], which are also the risk factors of coronary surgery patients. The mechanical properties of these arteries affect cerebral blood supply and can be measured non-invasively [3].

Every preoperative carotid duplex study at our institution also reports right and left vertebral flow volumes in mL/min. The report compares their sum with a threshold of 200 mL/min. This threshold is historical and is not a published limit of normal. Seidel and colleagues measured net vertebral flow volume in an age group similar to patients with cerebrovascular disease. The mean was 182 mL/min and the fifth to ninety-fifth percentile range was 102.4 to 301.0 mL/min [4]. A threshold of 200 mL/min is therefore higher than the reported mean, not lower than the normal range.

The measurement adds no cost but is not used in any decision. Whether it should be is an empirical question, and coronary surgery patients are a suitable group in which to test it: they are screened systematically, then undergo an operation with a defined risk of cerebral injury.

The term “vertebrobasilar insufficiency” has been used for decades for both a haemodynamic state and a clinical syndrome. Chandratheva and colleagues argued that it is not different from posterior circulation transient ischaemic attack, that it leads to symptoms such as isolated vertigo being attributed to vascular compression, and that it should no longer be used [5]. We agree. We therefore analysed the exposure as a flow volume in mL/min.

Stroke is one of the most serious complications of cardiac surgery, with a reported incidence of 2% to 5% [6–9]. Preoperative cerebrovascular assessment focuses on carotid disease, and the posterior circulation receives little attention [10]. Many studies have examined carotid stenosis in CABG patients [11–14]. Data on vertebral artery flow are limited.

The aim of this study was to test whether total vertebral artery flow volume is associated with clinically significant postoperative stroke after isolated CABG. The meaning of such a result depends on where the strokes occur and by what mechanism. We therefore also classified the location and probable cause of every stroke.

Methods

Study design and population

This retrospective cohort study was performed at Dr. Siyami Ersek Thoracic, Cardiac and Vascular Surgery Training and Research Hospital, a tertiary centre in Istanbul, and is reported according to the STROBE recommendations. Duplex Doppler ultrasonography (DUS) of the carotid and vertebral arteries is performed routinely before elective cardiac surgery and was the primary screening method. Computed tomography angiography (CTA) was performed in some patients because of clinical indication or

Doppler findings. These images were reviewed retrospectively, and CTA was accepted when the two methods disagreed.

Between January and August 2022, 1,377 patients underwent cardiac surgery, of whom 930 had isolated CABG with cardiopulmonary bypass and aortic cross-clamping (Fig. 1). We excluded 14 patients with recent carotid stenting or concomitant carotid endarterectomy, and one patient with unilateral total carotid occlusion. One patient with a remote carotid intervention was included because current imaging was available. Combined procedures, off-pump surgery, beating-heart surgery and redo surgery were excluded. The final population was 915 patients. All had a median sternotomy and one to five grafts.

Vertebral artery flow volume

The exposure was total vertebral artery flow volume, defined as the sum of the right and left values in the last duplex study before surgery. The institutional report is structured and gives both values in mL/min. The interval between that study and surgery was short (median 6 days, IQR 3–13) and never longer than six months. Both values were available for 845 of the 915 patients (92.3%).

We treated two different situations separately. In the first, the report documented that a vessel was hypoplastic, occluded, or had retrograde flow due to subclavian steal. That vessel was assigned a flow of zero and the patient was included. This applied to 26 patients. Retrograde flow is not the same as absent flow, so we also performed a sensitivity analysis excluding all 26. In the second situation, neither vessel could be insonated, usually because of calcification. The measurement was then recorded as not obtainable rather than as zero, and the patient was excluded. If these patients had been assigned a value of zero, they would have formed a small group with a total of zero and would have had a large influence on the continuous analysis. Seventy patients (7.7%) were excluded for these reasons or because no preoperative vertebral study could be found.

No duplex report could be found for 60 of the 915 patients, although a carotid stenosis grade had been recorded for them during data collection. These grades could not be checked against a source report. All carotid analyses were therefore repeated in the 855 patients whose report was available.

Flow volume was analysed on three scales: as a continuous variable, per 10 mL/min; by tertile of the observed distribution; and above and below 200 mL/min. The 200 mL/min threshold is the one used in our duplex reports and named in the study protocol. The tertile analysis was exploratory. We also counted the patients below 102.4 mL/min, which is the published fifth percentile of net vertebral flow volume in adults [4].

Carotid artery evaluation

Internal carotid artery stenosis was graded from peak systolic velocity, end-diastolic velocity and the ratio of internal carotid to common carotid peak systolic velocity. We used the Society of Radiologists in

Ultrasound consensus criteria. These define three mutually exclusive categories of diameter reduction, < 50%, 50–69% and $\geq 70\%$, measured at the distal internal carotid artery by the NASCET method [15]. A carotid grade was available for all 915 patients, so the carotid analyses include the whole cohort.

We defined significant stenosis as $\geq 60\%$, the threshold named in the study protocol. This is a treatment threshold and not an ultrasound grading boundary. It was the entry criterion of the randomised trials of endarterectomy for asymptomatic stenosis, and it was later used in vascular surgery guidelines and in trials in CABG patients [12, 14, 16]. It does not match the grading boundaries, and published series have used $\geq 50\%$ or $\geq 70\%$. We therefore added post hoc analyses at both of these cut-offs. The three-grade comparison and the pairwise comparisons were also post hoc.

Evaluation and characterisation of postoperative stroke

Patients with clinically suspected stroke after surgery were referred for neurological consultation and underwent brain computed tomography (CT). The consulting neurologist made the diagnosis from the CT findings and the clinical examination [17]. Patients without neurological symptoms were not imaged or screened. No routine postoperative magnetic resonance imaging (MRI) was performed.

The primary outcome was postoperative stroke as defined above, in any vascular territory, during the index hospitalisation. Anterior and posterior events were counted together, because the clinical question concerns symptomatic perioperative stroke as it occurs in practice.

All brain imaging reports and neurology notes of patients who met the definition were reviewed again by one investigator, who was not blinded to the flow values. Each event was classified by vascular territory, side, timing and probable cause. Territory was recorded as anterior, posterior, both, or no visible acute lesion. Timing was recorded as early if the deficit was noticed within 24 hours of surgery, including deficits present on emergence. Cause was assigned with a modified TOAST classification adapted to the perioperative setting [18]: cardioembolic or aortic atheroembolic, large-artery atherothrombotic, haemodynamic or watershed, or undetermined. This classification is exploratory. There was no second rater and agreement was not measured.

Two additional patients developed a definite postoperative neurological deficit but were never imaged. One died with absent brainstem reflexes. The other was considered unfit for transfer to the scanner. Both were assessed clinically as global hypoxic-ischaemic injury and not as focal infarction, and neither met the imaging requirement of the endpoint. Both were therefore counted as non-events. This decision could reasonably have been made differently, so we repeated the main flow analyses counting both as events. Nineteen patients met the full definition.

Statistical analysis

We used IBM SPSS Statistics version 27, with penalised regression performed in R version 4.6.1 using the logistf package (version 1.26.1). Continuous variables are given as median (interquartile range, IQR)

and categorical variables as number and percentage. Groups were compared with the chi-square test, the Fisher exact test or the Mann-Whitney U test. Stroke was uncommon and several cells in the carotid tables had expected counts below five, so exact tests are reported for those comparisons. Odds ratios (OR) are given with 95% confidence intervals (CI).

Binary logistic regression was used to test the association between vertebral flow volume and stroke, adjusted for age and carotid stenosis $\geq 60\%$. The flow models are based on 17 events, so the estimates are imprecise and confidence intervals are reported throughout rather than p values alone. At this number of events maximum-likelihood estimates and Wald intervals can be biased, so the models were repeated with Firth penalised likelihood, which corrects that bias. We also repeated the main analyses counting the two unimaged neurological events as events, and excluding patients in whom either vertebral artery was assigned a flow of zero. All of these are given in Table 2.

Because the main result is negative, we calculated what the study could detect. Power was computed for the comparison of two independent proportions using the normal approximation, at a two-sided alpha of 0.05. With 210 patients below and 635 at or above the 200 mL/min threshold, and a stroke incidence of 2.0%, the study had 80% power to detect an odds ratio of 3.2. An association weaker than about threefold could not be detected reliably.

Patients included in and excluded from the flow analyses were compared for age, sex, carotid stenosis and stroke incidence. A p value below 0.05 was considered significant. No imputation was performed.

Results

Patient characteristics

Of the 915 patients, 718 (78.5%) were male and the median age was 62 years (IQR 56–68) (Table 1). Postoperative stroke occurred in 19 patients (2.1%). Flow volume was available for 845 patients. The 70 patients without it did not differ from the analysed group in age, sex, carotid stenosis $\geq 60\%$ or stroke incidence (all $p > 0.5$).

Table 1
Characteristics of the cohort and of the flow-analysis subset

Variable	Full cohort (n = 915)	Flow analysed (n = 845)	Flow unavailable (n = 70)	p value
Age, years, median (IQR)	62 (56–68)	62 (56–68)	64 (56–69)	0.513*
Male sex, n (%)	718 (78.5)	665 (78.7)	53 (75.7)	0.665**
Carotid stenosis < 50%, n (%)	828 (90.5)	761 (90.1)	67 (95.7)	
Carotid stenosis 50–69%, n (%)	60 (6.6)	59 (7.0)	1 (1.4)	
Carotid stenosis ≥ 70%, n (%)	27 (3.0)	25 (3.0)	2 (2.9)	
Carotid stenosis ≥ 60%, n (%)	49 (5.4)	47 (5.6)	2 (2.9)	0.576***
Postoperative stroke, n (%)	19 (2.1)	17 (2.0)	2 (2.9)	0.651***
Total vertebral flow, mL/min, median (IQR)	—	220 (200–252)	—	
Right vertebral flow, mL/min, median	—	101	—	
Left vertebral flow, mL/min, median	—	120	—	
<i>IQR</i> interquartile range. *Mann–Whitney U test. **Pearson chi-square test. ***Fisher exact test. Baseline characterisation was limited to the variables held in the operative database; comorbidity, ventricular function and bypass times were not retrievable. Comparison of the flow-analysed and flow-unavailable groups on four variables is weak evidence of exchangeability and is reported as such.				

Vertebral artery flow volume

Median total vertebral artery flow volume was 220 mL/min (IQR 200–252; range 35–438). Flow was below the 200 mL/min threshold in 210 patients (24.9%).

Compared with the published reference range, the values in this cohort were high and not low: the cohort mean of 225 mL/min is higher than the reference mean of 182 mL/min. Only 15 patients (1.8%) were below the reported fifth percentile of 102.4 mL/min. None of these 15 patients had a stroke, and neither did any of the 75 patients with flow below 150 mL/min. One vertebral artery was assigned a flow of zero in 26 patients (3.1%), and one of these patients had a stroke. In 11 of the 749 reports giving both flow values and a summary sentence (1.5%), the summary did not match their sum. All analyses use the measured values.

Flow volume was not associated with postoperative stroke (Table 2). Median flow was 227 mL/min in patients with stroke and 220 mL/min in patients without ($p = 0.789$). The adjusted odds ratio was 1.009 per 10 mL/min (95% CI 0.931–1.094). Stroke incidence across ascending tertiles was 1.8%, 2.8% and 1.5% ($p = 0.513$). Stroke occurred in 4 of 210 patients below 200 mL/min (1.9%) and in 13 of 635 patients at or above it (2.0%), giving an adjusted odds ratio of 0.967 (95% CI 0.311–3.012).

The result did not change under any of the additional analyses (Table 2). Firth penalised likelihood gave an adjusted odds ratio of 1.011 per 10 mL/min (95% CI 0.931–1.093) and 1.041 below 200 mL/min (95% CI 0.313–2.867). Counting the two unimaged neurological events as events, and excluding the 26 patients with a zero-flow vessel, both gave odds ratios close to unity with confidence intervals of the same width.

Table 2 Total vertebral artery flow volume and postoperative stroke (n=845, 17 events)

Analysis	Stroke (+) / n (%)	Crude OR (95% CI)	Adjusted OR (95% CI)	p value
Continuous, per 10 mL/min	—	1.013 (0.935–1.097)	1.009 (0.931–1.094)	0.819
By tertile of flow				
T1, 35–206 mL/min	5 / 284 (1.8)	reference		
T2, 207–240 mL/min	8 / 289 (2.8)	1.589 (0.513–4.919)		
T3, 241–438 mL/min	4 / 272 (1.5)	0.833 (0.221–3.138)		0.513*
Above and below 200 mL/min				
<200 mL/min	4 / 210 (1.9)	0.929 (0.300–2.881)	0.967 (0.311–3.012)	0.954
≥200 mL/min	13 / 635 (2.0)	reference	reference	
Median flow, mL/min (IQR)	227 (205–240) vs 220 (200–252)	—	—	0.789**

Sensitivity analyses

Analysis	n	Events	Per 10 mL/min, adjusted OR (95% CI)	<200 mL/min, adjusted OR (95% CI)
Primary, maximum likelihood	845	17	1.009 (0.931–1.094)	0.967 (0.311–3.012)
Primary, Firth penalised likelihood	845	17	1.011 (0.931–1.093)	1.041 (0.313–2.867)
Counting the two unimaged neurological events as events	845	18	1.012 (0.934–1.092)	0.966 (0.292–2.624)
Excluding patients with a zero-flow vertebral artery	819	16	1.015 (0.929–1.102)	0.907 (0.232–2.703)

CI confidence interval, IQR interquartile range, OR odds ratio. Crude odds ratios are from univariable binary logistic regression and adjusted odds ratios from multivariable binary logistic regression including age and carotid stenosis $\geq 60\%$. The two sensitivity analyses were estimated by Firth penalised likelihood, with profile penalised-likelihood confidence intervals. The tertile analysis was exploratory and its odds ratios are unadjusted. *Pearson chi-square across the three tertiles. **Mann–Whitney U test. The Fisher exact p value for the 200 mL/min comparison is 1.000. With 17 events the study had 80% power to detect an OR of 3.2 at the 200 mL/min threshold, 59% for an OR of 2.5 and 37% for an OR of 2.0; associations smaller than about threefold could not have been reliably detected. Re-expressed on a clinical scale the adjusted continuous estimate is an OR of 1.05 (95% CI 0.69–1.60) per interquartile range and 1.09 (95% CI 0.49–2.46) per 100 mL/min. Measured against published reference values for net vertebral flow volume (mean 182 mL/min, fifth to ninety-fifth percentile 102.4–301.0 mL/min), only 15 patients (1.8%) fell below the fifth percentile and none of them had a stroke.

Carotid stenosis and stroke

Stroke incidence increased with stenosis grade in the whole cohort (Table 3). It was 1.6% in 828 patients with < 50% stenosis, 6.7% in 60 patients with 50–69% stenosis and 7.4% in 27 patients with $\geq 70\%$ stenosis (exact p = 0.007). The difference lies between patients with less than 50% stenosis and patients with any stenosis of 50% or more; the two diseased grades did not differ from each other.

Table 3 Carotid stenosis and postoperative stroke (n=915, 19 events)

Panel A. Ultrasound grade (three mutually exclusive categories)

Grade	Total (n)	Stroke (+) n (%)	Adjusted residual
<50% stenosis	828	13 (1.6)	–3.31
50–69% stenosis	60	4 (6.7)	+2.58
$\geq 70\%$ stenosis	27	2 (7.4)	+1.97

Overall comparison across the three grades: Fisher–Freeman–Halton exact test p=0.007 (Pearson chi-square 11.04, 2 df, p=0.004).

Post hoc pairwise comparisons (Fisher exact, Bonferroni-corrected for three tests): <50% versus 50–69%, $p=0.023$ uncorrected and 0.069 corrected; <50% versus $\geq 70\%$, $p=0.078$ and 0.235; 50–69% versus $\geq 70\%$, $p=1.000$ and 1.000.

Panel B. Stenosis above and below three thresholds

Threshold	Below: stroke (+) / n (%)	At or above: stroke (+) / n (%)	Crude OR (95% CI)	p value
50%	13 / 828 (1.6)	6 / 87 (6.9)	4.644 (1.719–12.547)	0.006
60%	15 / 866 (1.7)	4 / 49 (8.2)	5.043 (1.608–15.815)	0.016
70%	17 / 888 (1.9)	2 / 27 (7.4)	4.099 (0.898–18.707)	0.105

Odds ratios in Panel B are the 2x2 odds ratio with a Woolf (log-transform) 95% confidence interval, and all p values are from the Fisher exact test. Expected cell counts fell below five in every comparison involving the $\geq 70\%$ group (minimum expected count 0.56 in Panel A and 0.56, 1.02 and 1.81 in Panel B), so exact tests are reported in preference to the chi-square approximation; the corresponding uncorrected chi-square p values were 0.001, 0.002 and 0.049. Adjusted for age, carotid stenosis $\geq 60\%$ remained associated with stroke in the full cohort (adjusted OR 5.145, 95% CI 1.598–16.558; Wald $p=0.006$, likelihood ratio $p=0.017$), in the flow-analysis subset (adjusted OR 3.830, 95% CI 1.037–14.152, Wald $p=0.044$) and in the 855 patients whose duplex report could be retrieved (adjusted OR 3.806, 95% CI 1.029–14.072, Wald $p=0.045$). Firth penalised likelihood gave a similar estimate in the full cohort (adjusted OR 5.517, 95% CI 1.609–15.789, $p=0.009$), so the association is not an artefact of the small number of events. Adding carotid stenosis to a model containing age alone raised the area under the receiver operating characteristic curve from 0.512 to 0.559, so this association does not amount to useful discrimination in the individual patient. *CI* confidence interval, *OR* odds ratio. The post hoc comparisons place the difference between patients with less than 50% stenosis and those with any stenosis of 50% or more; the two diseased grades did not differ from one another.

At the $\geq 60\%$ threshold, stroke occurred in 4 of 49 patients (8.2%) and in 15 of 866 patients below it (1.7%), giving a crude odds ratio of 5.04 (95% CI 1.61–15.82, $p = 0.016$). After adjustment for age the odds ratio was 5.15 (95% CI 1.60–16.56, $p = 0.006$), and 5.52 (95% CI 1.61–15.79) by Firth penalised likelihood. The association was similar at $\geq 50\%$ and in the two restricted samples, and was not significant at $\geq 70\%$, where only 27 patients and two events remain (Table 3).

Two points qualify this finding. The confidence intervals are wide, because they rest on four events in the stenosis group. And although carotid stenosis identifies a group at higher risk, it does not separate individual patients: adding it to a model containing age raised the area under the receiver operating characteristic curve only from 0.512 to 0.559.

Anatomical distribution, timing and probable cause

The 19 patients who met the endpoint definition had 35 brain CT examinations in total (Table 4). Ten events (52.6%) were limited to the anterior circulation, three (15.8%) to the posterior circulation, and five (26.3%) involved both. In one patient the acute lesion could not be localised. The posterior circulation was therefore involved in 8 of the 19 events (42.1%). Among the 17 patients with a flow measurement, it was involved in 1 of 4 patients below 200 mL/min and in 6 of 13 patients at or above it. The first CT was performed within 24 hours in 4 patients and later in 15, at a median of 68 hours. In the exploratory classification of cause, 8 events were cardioembolic or aortic atheroembolic, 7 were haemodynamic or watershed and 4 were undetermined. No event was attributed to large-artery atherothrombosis.

Table 4
Territory, timing and probable aetiology of the 19 postoperative strokes

Characteristic	All strokes (n = 19)	Flow < 200 mL/min (n = 4)	Flow ≥ 200 mL/min (n = 13)
Vascular territory, n			
Anterior circulation only	10	3	7
Posterior circulation only	3	1	2
Both anterior and posterior	5	0	4
Acute lesion not localisable on available imaging	1	0	0
Individual territories involved, n			
Middle cerebral artery	9	1	7
Anterior cerebral artery	1	0	1
Internal borderzone / deep white matter	5	2	3
Posterior cerebral artery / occipital	6	1	4
Cerebellar	3	0	3
Brainstem	1	0	1
Laterality, n			
Right	7	2	5
Left	4	1	3
Bilateral	7	1	5
Timing of first postoperative imaging, n			
Within 24 h of surgery	4	1	2
Later than 24 h	15	3	11
Probable aetiology, n			
Two of the 19 patients had no measured preoperative flow volume and appear only in the first column. Posterior circulation involvement was documented in 8 of the 19 events (42.1%), of which 1 occurred in a patient with flow below 200 mL/min and 6 in patients at or above it. Aetiology was assigned using a modification of the TOAST classification adapted to the perioperative setting. Because outcome ascertainment relied on computed tomography, posterior fossa infarcts are likely to be underrepresented; in four patients the acute lesion was documented on repeat imaging or in the neurology record rather than on the first CT.			

Characteristic	All strokes (n = 19)	Flow < 200 mL/min (n = 4)	Flow ≥ 200 mL/min (n = 13)
Cardioembolic or aortic atheroembolic	8	2	6
Large-artery atherothrombotic	0	0	0
Haemodynamic / watershed hypoperfusion	7	2	5
Undetermined	4	0	2
Two of the 19 patients had no measured preoperative flow volume and appear only in the first column. Posterior circulation involvement was documented in 8 of the 19 events (42.1%), of which 1 occurred in a patient with flow below 200 mL/min and 6 in patients at or above it. Aetiology was assigned using a modification of the TOAST classification adapted to the perioperative setting. Because outcome ascertainment relied on computed tomography, posterior fossa infarcts are likely to be underrepresented; in four patients the acute lesion was documented on repeat imaging or in the neurology record rather than on the first CT.			

Discussion

Vertebral artery flow volume is measured in every patient screened before cardiac surgery at our institution and is printed on every report. In 845 patients undergoing isolated CABG it gave no information about the risk of postoperative stroke. Patients who had a stroke had almost the same flow as patients who did not. This was true when flow was analysed as a continuous measurement, in thirds, and above and below the 200 mL/min threshold that our reports use. It remained true after adjustment for age and carotid disease, under penalised regression, and in both sensitivity analyses.

The threshold itself deserves separate comment, because what our data show about it does not depend on how many strokes we observed. The 200 mL/min figure comes from the older vertebrobasilar insufficiency literature and not from a reference series. When Seidel and colleagues measured net vertebral flow volume in patients of the age we operate on, the mean was 182 mL/min and the normal range was 102.4 to 301.0 mL/min [4]. Our threshold therefore lies above the average of a normal population. It marked a quarter of our patients as abnormal, although only 15 of them were genuinely below the normal range. A threshold in that position will label many normal patients and identify few abnormal ones, which is what we observed.

Two findings suggest that the negative result is not simply the consequence of looking in the wrong place. First, carotid stenosis was measured in the same duplex studies, in the same patients, with the same method of detecting stroke, and it was clearly associated with stroke. The examination was not uninformative about vascular risk in this cohort. Second, the strokes themselves involved the posterior circulation in 8 of 19 cases. If every stroke had been anterior and embolic, a negative association with a posterior circulation measurement would have been predictable and would have told us nothing.

The mechanism of stroke after cardiac surgery fits this result. Most events follow embolism from aortic manipulation, cannulation and cardiopulmonary bypass, or a period of hypoperfusion in a vulnerable patient [7, 8]. In our review of the 19 events, cardioembolic and atheroembolic strokes were the most frequent, and none was attributed to atherothrombosis of the vertebral arteries. A chronically reduced resting inflow to one territory is not a plausible cause of embolic occlusion of a distal branch. It might matter more during the operation itself, when perfusion pressure is controlled externally, but a single preoperative measurement cannot describe that.

There are two reasons not to read our result as proof that vertebral flow is unimportant. Nineteen strokes is a small number, and the study could reliably detect only an association of about threefold or more; a moderate association could have been missed. More importantly, this cohort contained almost no patients with genuinely low flow. Fifteen patients were below the published fifth percentile, and none of them had a stroke. We can say that the threshold we use does not identify risk. We cannot say what happens to a patient whose posterior circulation inflow is truly reduced, because we had too few such patients to find out.

Wiberg et al. [19] reported an association between asymptomatic vertebral artery stenosis and cerebral lesions on MRI after CABG. Their exposure was anatomical stenosis and ours was flow volume, and their endpoint was subclinical infarction on MRI while ours was clinically overt stroke. These endpoints differ about tenfold in incidence, so the two results are not in conflict.

One practical observation came out of the data. Each duplex report states the two flow values and then summarises whether their total is below 200 mL/min. In 1.5% of reports the summary did not follow from the values above it. Anyone using this variable for research should take the measured numbers rather than the summary line.

Limitations

The most important limitation is that this cohort included almost no patients with genuinely low posterior circulation inflow. Only 15 patients were below the published fifth percentile, and none of them had a stroke. The study therefore tests an institutional threshold, not the physiology behind it.

The second limitation is the number of events. Seventeen events in the flow analysis allow only a threefold or larger association to be detected, so every estimate here is imprecise.

The third follows from the retrospective design. Brain CT was performed only in patients with clinically suspected stroke, so asymptomatic infarcts certainly occurred and were not counted, and all results concern clinically overt stroke only. Some infarcts are not visible on CT in the first hours, and in four of our patients the acute lesion was seen only on a later study. CT is also less sensitive than diffusion-weighted MRI, and least sensitive in the posterior fossa, where beam-hardening artefact obscures the brainstem and cerebellum [20]. Posterior symptoms such as vertigo and ataxia are more likely than

hemiparesis to be attributed to sedation or postoperative weakness. Both effects reduce the detection of posterior events, which are the events of greatest interest here.

Two patients with a definite clinical deficit were never imaged and were counted as non-events. Current definitions of central nervous system infarction allow a clinical diagnosis when imaging is not available, so this decision was reasonable but not obligatory. We made it because both patients were assessed as global hypoxic-ischaemic injury and not as focal infarction. Counting both as events did not change the results.

Duplex flow volume is operator-dependent and sensitive to insonation angle and vessel calibre. The measurements were made in routine practice by several operators, reproducibility between observers was not assessed, and nearly half of the individual values were recorded as multiples of ten, which indicates rounding at the time of measurement. Imprecision of this kind would tend to hide a real association rather than create one, so it is an alternative explanation for our result.

A duplex report could not be found for 60 patients (6.6%), so their carotid grades cannot be verified against a source document; the restricted analysis above addresses this. Flow volume was not available for 70 patients (7.7%), who did not differ from the analysed patients in age, sex, carotid stenosis or stroke incidence.

Baseline characterisation was limited to the variables available in the operative database, so comorbidity, ventricular function and bypass times could not be tabulated or adjusted for, and residual confounding cannot be excluded. Circle of Willis anatomy was not evaluated, so collateral reserve could not be assessed, although it may determine whether a reduced inflow has any clinical consequence. Postoperative atrial fibrillation and intraoperative haemodynamic data were not available, so the classification of stroke mechanism rests on imaging and clinical review by one unblinded investigator and is exploratory.

Conclusions

Total vertebral artery flow volume, measured routinely before cardiac surgery, was not associated with clinically overt stroke after isolated CABG. Carotid stenosis measured in the same duplex studies was associated with stroke, and the posterior circulation was involved in 8 of the 19 events, so neither the examination nor the endpoint was insensitive to vascular risk.

The 200 mL/min threshold printed in our reports lies above the mean of a published reference population. It marked one patient in four as abnormal and identified no increase in stroke risk. A value below this threshold is not by itself a reason to change the perioperative plan, to request further imaging, or to defer surgery in a patient scheduled for isolated CABG.

This conclusion applies to the threshold, not to the physiology. With 19 strokes the study could not detect a moderate association, and almost no patient in this cohort had flow below the normal range.

Whether a genuinely reduced posterior circulation inflow carries perioperative risk remains open, and answering it will require a cohort selected against published reference values rather than an inherited threshold. Our findings also do not apply to patients with posterior circulation symptoms, in whom the measurement keeps its diagnostic role.

Abbreviations

CABG

coronary artery bypass grafting

CI

confidence interval

CT

computed tomography

CTA

computed tomography angiography

DUS

duplex Doppler ultrasonography

IQR

interquartile range

MRI

magnetic resonance imaging

OR

odds ratio

TOAST

Trial of Org 10172 in Acute Stroke Treatment.

Declarations

Ethics approval and consent to participate. The study was conducted at Dr. Siyami Ersek Thoracic, Cardiac and Vascular Surgery Training and Research Hospital, Istanbul. Institutional permission to review the patient records was granted by the hospital's Medical Specialty and Training Board on 25 August 2022 (document no. E-28001928-604.01.01), before any record was accessed. Approval for the analysis reported here and for its publication was granted by the Hamidiye Non-Interventional Research Ethics Committee, University of Health Sciences Turkey (project registration no. 26/367; decision no. 10/11; 14 July 2026). That approval covers the retrospective use of duplex and computed tomography reports and of consultation notes. The study was conducted in accordance with the Declaration of Helsinki. Because only existing records were used and no intervention was performed, the requirement for written informed consent was waived.

Consent for publication. Not applicable.

Availability of data and materials. The dataset is not publicly available because Turkish personal data protection legislation restricts the sharing of identifiable patient records. A de-identified dataset is available from the corresponding author on reasonable request.

Competing interests. The authors declare that they have no competing interests.

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Authors' contributions. MR conceived and designed the study, collected the data, performed the statistical analysis, oversaw all stages of the project and drafted the manuscript. LC, as responsible investigator, obtained the institutional research permission and the ethics committee approval, took part in data collection and contributed to the interpretation of the results and to manuscript preparation. BK took part in data collection, supervised the project and supported the approval process. TK supervised the project and contributed to the interpretation of the results. MY and SO contributed to the interpretation of the results and to manuscript preparation. FK interpreted the data and critically revised the manuscript for important intellectual content. MSK contributed to the interpretation of the results and critically reviewed the manuscript. All authors read and approved the final manuscript.

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Figures

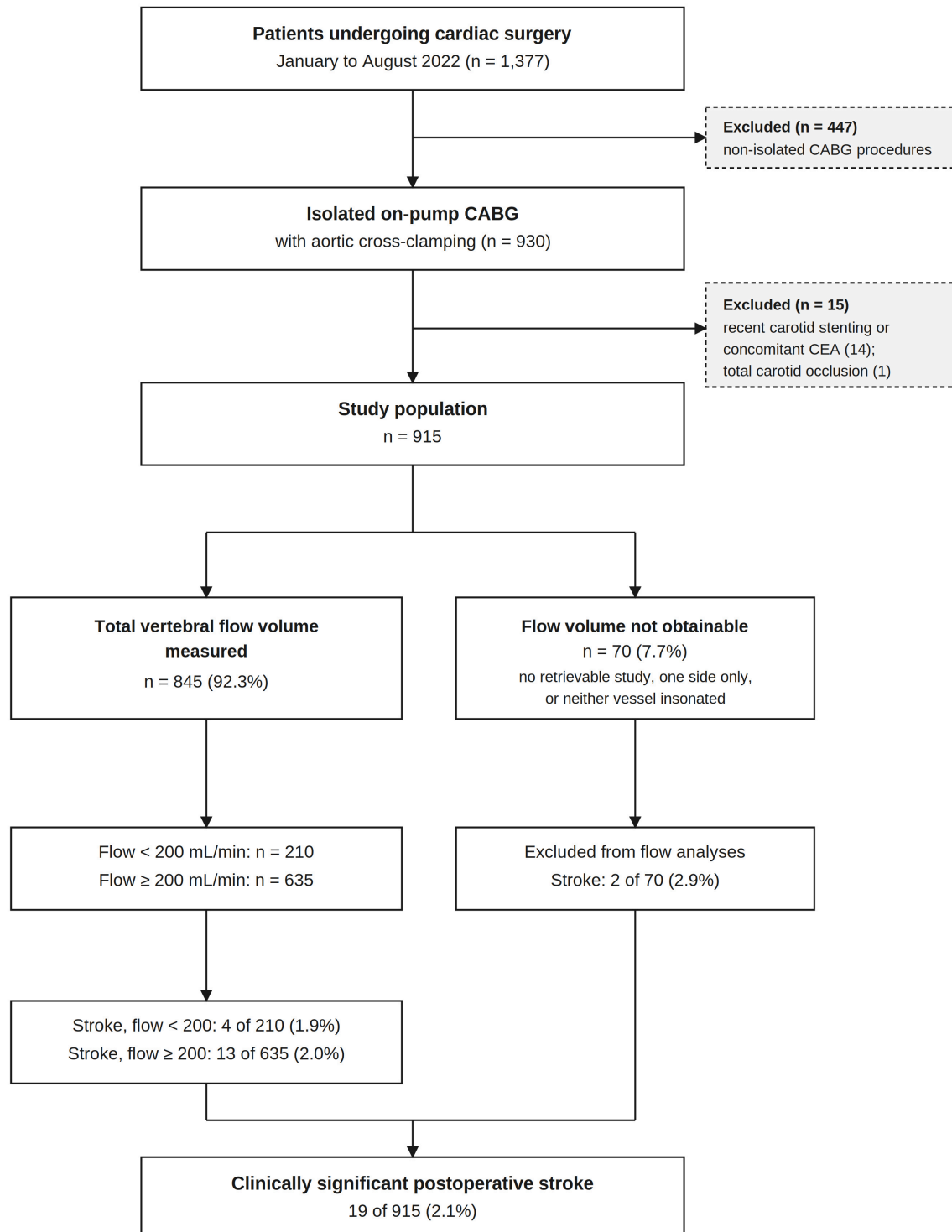


Figure 1

Study flow diagram of patient selection and of the availability of vertebral artery flow data.