Seasonal variation of acute carotid surgery: Does it exist?

Cardiovascular events may show seasonal patterns, with a higher frequency of acute vascular events occurring in winter months.[1,2] We investigated whether seasonal variation exists for acute carotid surgery. Between December 21, 1996, and December 20, 2008, 159 urgent (≤12 hours from the development of symptoms or diagnosis) and emergency (≤1 hour from development of symptoms) carotid interventions were performed in 149 patients in our vascular unit. All patients had at least 1 year of postoperative follow-up.

Interventions were divided into 7 groups: A, internal carotid artery (ICA) thrombosis (22 cases); B, preocclusive (>90%) ICA stenosis (73 cases); C, acute postcarotid endarterectomy (CEA) ICA thrombosis (29 cases); D, acute post-CEA hemorrhage (35 cases); E, pre- and post-CEA ICA thromboses (groups A and C; 51 cases); F, acute pre-CEA events (groups A and B; 95 cases); and G, acute post-CEA events (groups C and D; 64 cases). In group A, 11 of the 22 interventions were ICA embolectomy, followed by CEA. In the other 11 cases, surgery was limited to thrombus removal from the common carotid artery (CCA). Group B underwent 73 interventions and group C underwent 29. All 35 interventions in group D were exploration with control of bleeding sites.

Day of symptom onset was categorized into twelve 1-month intervals and four 3-month intervals (Spring, March 21-June 20; Summer, June 21-September 22; Autumn, September 23-December 20; Winter, December 21-March 20). The distribution of symptom onset within the four 3-month periods was tested for uniformity in the overall population by conventional statistics (tests for goodness of fit). Chronobiologic analysis was performed by applying cosinor analysis and partial Fourier analysis to the monthly data to select the harmonic, or combination of harmonics, that best explained the variance of the time season data.[3] The percentage of rhythm (the percentage of the overall variability of the data on the arithmetic mean that is attributed to fixed rhythmic function) and the probability value resulting from the *F* statistic was used to test the 0-amplitude null hypothesis to estimate the goodness of fit of the approximating model and statistical significance.

The parameters calculated for the overall 1-year end period cosine approximation of the time series data (period of 8766 hours) were the midline estimated statistic of rhythm (the rhythm-adjusted mean over the time period analyzed), amplitude (one-half the difference between the absolute maximum and minimum of the fitted curve), and the peak (acrophase) N-trough (bathyphase) time, respectively, indicating the occurrence of the absolute maximum and minimum values during the year. Significant levels were set at $P < .05$.

The 149 patients underwent 159 interventions. Inferential chronobiologic analysis by month of the year yielded a seasonal variation for groups B, C, E, and F (Fig 1), with respective peaks in October ($P = .030$), December ($P = .018$), December ($P = .037$), and October ($P = .002$). No significant peaks were found for A, D, and G. Analysis by seasons found peaks in Winter ($P = .0157$), Autumn ($P = .011$), Winter ($P = .0086$), and Winter-Autumn ($P = .0266$) for A, B, E, and F, respectively (Fig 2). We did not observe any variation in clinical outcome.

The results seem to confirm a seasonal pattern for carotid urgent and emergency surgery, with peaks in winter and autumn, to a lesser extent. To our knowledge, this is the first report of seasonal variation in acute carotid disease. Several pathophysiologic factors have been associated with increased frequency of cardiovascular events during colder months, including increased blood pressure and arterial spasm, platelet and red cell count, blood viscosity, lipid levels, and clotting activity.[4] Endogenous rhythms may also be a factor (seasonal winter peak of aortic aneurysm dissection is detectable independent of climate).[5]

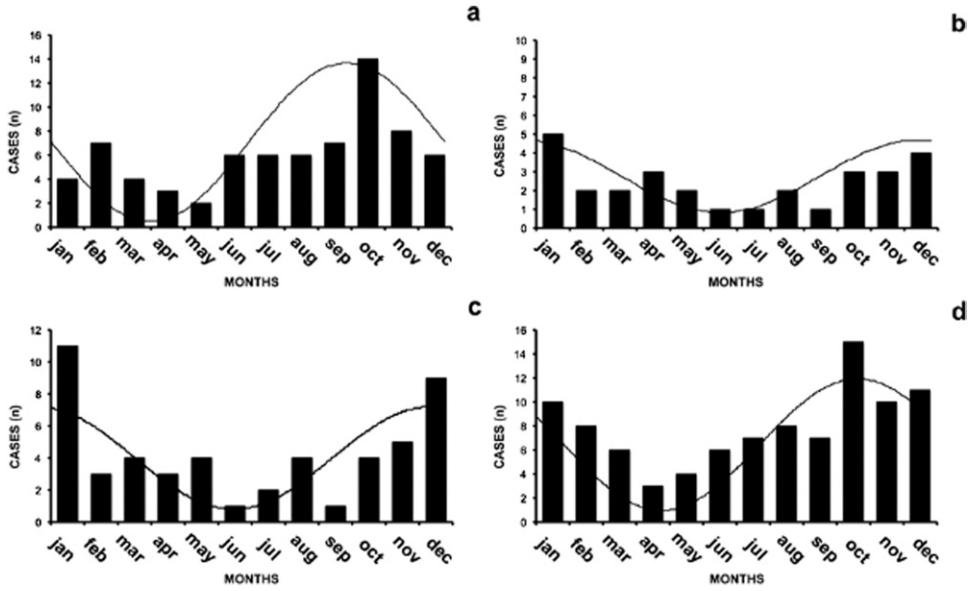

Fig 1. Monthly distribution of carotid interventions for **(a)** preocclusive internal carotid artery stenosis (group B), **(b)** acute postcarotid endarterectomy (CEA) thrombosis (group C), **(c)** total acute thromboses (group E), and **(d)** total acute pre-CEA events (group F). See text for explanations. Superimposed is the best-fitting curve from Fourier analysis.

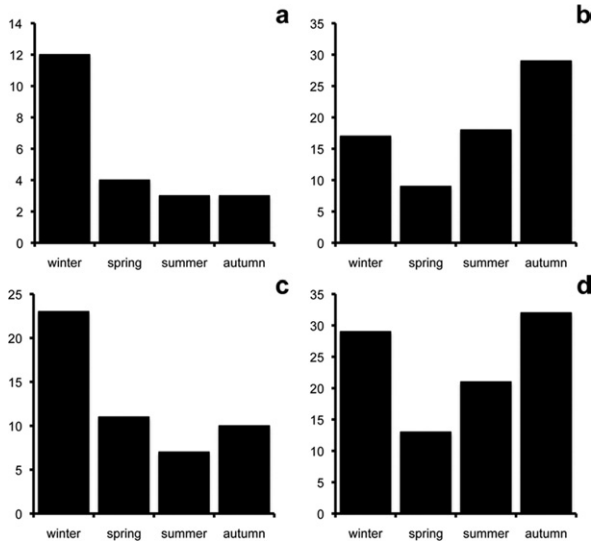

Fig 2. Seasonal distribution of carotid interventions for **(a)** acute internal carotid artery (ICA) thrombosis (group A), **(b)** preocclusive ICA stenosis (group B), **(c)** total acute thromboses (group E), and **(d)** total acute pre-CEA events (group F). See text for explanations.

Further studies, conducted on larger multicenter populations, and aimed to prospectively investigate the possible relationships between seasons and carotid vascular surgery will perhaps add another piece to the complex puzzle of seasonal variation of acute cardiovascular events.

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Submitted Mar 3, 2009; accepted Aug 24, 2009.

doi:10.1016/j.jvs.2009.08.083

Regarding "Analysis of motor and somatosensory evoked potentials during thoracic and thoracoabdominal aortic aneurysm repair"

We read with interest the article "Analysis of motor and somatosensory-evoked potentials during thoracic and thoracoabdominal aortic aneurysm repair" by Keyhani et al.[1] However, several questions arise concerning the interpretation of the statistical tests as well as the motor-evoked potentials (MEP) methodology.

The specificity of the somatosensory-evoked potentials (SSEP) is reported to be extremely good, with a value of 0.97. Yet, appearances can be deceptive. The SSEP showed no permanent changes in 96%, so the á priori chance of correctly identifying a favorable outcome is very high, independent from the actual results. This is probably also reflected in the fact that the SSEP was negative in five of eight patients who later had a neurologic deficit (ND). If we assume in a fictive situation that the neurophysiologist always reports no permanent changes on SSEP to the surgeon independent of what is measured, the test would be completely meaningless; however, the specificity would still be perfect (ie, 100%). So, the seemingly high accuracy that is suggested by a specificity value of 0.97 is in sharp contrast with its practical value. It is not warranted to conclude that, "SSEP monitoring is a reliable tool in ruling out ND state." The same concern applies to the significance of the MEP.

Calculation of the sensitivity probably reflects more realistically the validity of the two monitoring modalities. According to the authors, the difference between SSEP and MEP (0.38 vs 0.63) was statistically not significant; however, no statistical test is presented to substantiate this assertion. In our opinion, it is clinically relevant to know in 5 instead of 3 patients (out of 8) whether or not permanent spinal cord damage is imminent. The higher accuracy of the MEPs to predict an unfavorable outcome is also reflected in a higher odds ratio of 60.8 for MEP vs 21.9 for SSEP.

Also, the MEP methodology deserves further consideration. No information is provided on the minimal amplitude that was required to conclude whether a response was present (ie, this parameter was not clearly defined). The use of isoflurane is likely to cause rather low MEP amplitudes and hence signal to noise ratios, which might account for the six false-positive patients.[2] In addition, using a needle instead of surface electrodes will introduce an unnecessarily high variability of the signals.

Three patients experienced ND postoperatively even though MEP had not shown "permanent changes." Pathophysiologically, this finding is surprising, because the measurability of MEPs at the end of the surgical procedure proves the integrity of the whole motor tract from the cortex to the muscle. A possible explanation for this discrepancy might be "delayed ischemia," that is, a spinal cord lesion that occurs in the intensive care unit presumably due to a drop in blood pressure.[3] So, it would be important to know whether delayed ischemia was observed in the eight patients with ND, if one attempts to correctly ascertain the value of MEP monitoring.

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