Critical Neck Position as an Independent Risk Factor for Posterior Circulation Stroke

Abstract

The purpose of this study was to compare the effects of critical neck angulation (rotation and hyperextension) on vertebral artery perfusion in symptomatic and control populations and to determine whether this represents a risk factor for ischemic stroke. In a cross-sectional study, 64 symptomatic individuals with well documented brainstem ischemic events (average age, 70.9 yr) and 37 control subjects (average age, 66.3 yr) were evaluated using a dynamic magnetic resonance angiography technique designed to mimic activities of daily living. Abnormalities of perfusion at the atlantoaxial and atlantooccipital junction and distal vertebral artery were recorded and scored independently by two neuroradiologists.

Volume flow analysis was also recorded at the basilar artery, and distal and proximal vertebral arteries. Symptomatic subjects displayed a consistent pattern (56.4%) of contralateral hypoperfusion at the atlantoaxial and atlantooccipital junction and distal segments (grades 3 and 4) ($p < 0.001$). Unsuspected hypoplasia was noted in 13 % of the symptomatic subjects with a right-sided preponderance (88%), suggesting developmental susceptibility. Occlusion was noted in all subjects with contralateral neck rotation. Postpositional ischemia was present (68%) and correlated with female gender ($p < 0.001$), severity of stenosis (p 0.001), vascular risk factors ($p < 0.001$), and microinfarction on magnetic resonance images ($P < 0.05$). Flow analysis showed low basilar artery perfusion (< 25 ml/min) in 63 6%, and unsuspected with neck motion in 4 subjects. In the neutral position, 42% of symptomatic subjects had asymmetrical reduction of flow (50%) in one vertebral artery compared to the contralateral side. Hyperextension significantly reduced flow in 33%, whereas rotation produced a 42.4% reduction. Warfarin (Coumadin) and antiplatelet medications (70%) provided no protection from mechanical impingement of flow. In conclusion mechanical impingement by neck rotation and hyperextension produce profound vertebral artery hypoperfusion leading to occlusion. This appears to be an independent and modifiable risk factor for stroke. Public and physician education to minimize extreme neck motions during activities of daily living is needed.

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The dimensions of neck-induced mechanical impingement on vertebral artery perfusion as a health problem in the United States has not been defined or described previously. Prompted by the observation of 5 elderly women who had strokes after beauty parlor shampoo treatment [1], as well as a survey of 25 elderly women [2], the hazard of sustained hyperextension and rotation on vertebral artery flow was raised. Thus, concern arises for iatrogenic arterial injury and damage by trivial and extreme neck mobility as this may constitute an *independent,* yet under appreciated risk factor for vertebral artery territory ischemia and stroke

Most published studies on the role of neck motion and cerebral blood flow have been conducted postmortem. DeKleyn and Nieuwenhuyse [3] are frequently credited with the first description relating the influence of rotation on contralateral occlusion and/or reduction of flow at the atlantoaxial (AA) junction. In a subsequent cadaver study, Tatlow and Bammer [4] identified the atlantooccipital (AO) junction as the most susceptible segment. In 1963 Faris and coworkers [5] performed retrograde brachial angiography on 43 asymptomatic young males and identified occlusion in 11 subjects during head rotation. The levels were not specified. Since it is impractical to perform angiography on every individual with ischemic symptoms, the advent of noninvasive magnetic resonance angiography (MRA) provided a unique and novel in vivo technique to determine the role of cerebral hemodynamics in relation to neck mobility.

Materials and Methods

Study Design: Between January 1992 and May 1994, 64 patients were referred to the first author for suspected ischemic cerebrovascular disease and had extensive neurological examination, electroencephalography (EEG), and magnetic resonance imaging (MRI). Vertebrobasilar artery insufficiency or infarction (VBAI) was suspected as responsible for symptoms of vertigo, visual blurring, diplopia, tinnitus, ataxia, dizziness and dysarthria. Women comprised 69% and men 31 % of the study cohort and the mean age was 70.9-years (range, 21-97 yr). Hypertension and cardiac risk factors were present in 87.2% of the symptomatic group.

Thirty subjects who were asymptomatic for ischemic disease comprised the initial

control group. Women predominated, at 67%, with men at 33%. The mean age was 66.3 years (range, 22-80 yr). A herniated disc, spondylitic ridging, or both were present in 30% of the control group and 3 1 % of the symptomatic group.

MRI of the brain was performed with a 1.5-T Signa unit (General Electric). Sagittal images were obtained first, followed by double-echo spin-echo axial slices of whole brain. A three-dimensional (3d) time-of-flight (TOF) imaging technique was utilized for MRA of the head. Neck MRA. was performed in the axial plane utilizing two-dimensional (2D) TOF techniques, with superior saturation pulses suppressing the venous flow. A total of 128 slices 1.5 to 1..8 mm thick were obtained. Four series were performed with the neck in neutral position, hyperextension, and right and left rotation. The angle of rotation was determined on a collapsed image (similar to a base view). When the chin was included, the angle was determined using a vertical line from the inner occipital protuberance to the midchin. When the chin was not included, the carotid foramen were used to measure the angle. Quantitative 2D phase-contrast techniques became available in January 1994 and were utilized in 33 patients and 7 control subjects. Volume flow rate measurements (ml/min) and velocity (cm/sec) were recorded at the proximal and distal vertebral arteries, basilar artery,- and the corresponding common and internal carotid arteries with the neck in neutral, hyperextended, and left and right rotational positions.

The scanning time for each series of neck MRAs ranged between 11 and 13 minutes, whereas MRA of the head took 7 minutes. MRA and MRI were performed at two separate sittings within 1 week, to avoid motion artifact related to the study length

Grading: Initially, grading was based on visual changes in density and in luminal dimensions. All vertebral arteries were evaluated at the following sites: (1) C5-6 entrance of the vertebral artery into the transverse foramina, (2) at the AA segments (C1-2). (3) at the OA junction, and (4) at the-origins of the vertebral arteries and at the vertebrobasilar junction Carotid arteries were evaluated at the bifurcation and at the base of the skull as the carotid artery entered the carotid canal. Each of the above-mentioned segments was evaluated with the neck in neutral position, hyperextension, and left and right rotation..

Based on an arbitrarily defined scoring system, similar to that used for coronary artery disease [61, the following scale was utilized:

Grade I = well-visualized vessels in all views

Grade II = decrease in density with dynamic positions at the AA and AO junction. but no change in distal flow

Grade III = decrease in density of the vessels at the AA/AO junction and decrease in distal flow.

Grade IV = no flow (occlusion) with dynamic positioning.

Imaging results were compared in a blinded fashion by two neuroradiologists and validated independently against the subject's clinical diagnosis and category status. With 2D phase-contrast postprocessing software volume flow rates (ml/min) were analyzed in a corresponding, symptomatic population (N = 33).

Statistics: Differences between groups were assessed with Student's *t* test for continuous variables and Pearson's test for independence for analysis involving categorical variables. *p* Values of less than 0.05 were considered statistically significant. All comparisons were two-tailed.

The *k* statistics were used as a measure of observer reliability between the two blinded neuroradiologists. A *k* of 1 indicates perfect agreement whereas a *k* of 0 means agreement at chance level. A common guideline is that *k* of 0.75 indicates excellent agreement, whereas *k* values from 0.40 to 0.75 indicate fair to good agreement. *k* values of 0.40 indicate poor agreement [7]. *k* values were calculated for hypoplasia, scoring grade, and angle indices.

Pearson's correlation coefficients were computed to assess the relationship among risk factors and symptoms.

Results

The demographic features and vascular risk factors for the groups are listed in the table. There were no significant differences between groups for mean age (symptomatic, 70.9 yr; comparison, 66.3 yr), gender (symptomatic and comparison groups were 69% female) and presence of disc/arthritic ridging abnormality (symptomatic, 31%; comparison, 30%).

The reliability of scoring for stenosis yielded an excellent *k* agreement of 0.75; good *k* levels were found for the following measures: hypoplasia, 0.75; RVOA 0.71; RVAA, 0.65; LVOA, 0.56; LVAA, 0.54.

A *t* test for independent samples was used to assess group difference in stenosis. The level of stenosis in the study groups (mean, 2.49; standard deviation [SD], 0.91) was higher than that in the comparison group (mean, 1.23; SID, 0.68) ($t(67) = 6.29$, $p <$

0.001).

There was a significant association between gender and poststudy dizziness (X^2 (1) ~ 10.85, $p < 0.001$). Thirteen percent of men reported dizziness while 54.3% of women reported dizziness. When dizziness was correlated with selected variables, significant correlations were found for gender ($p < 0.01$), MRI microvascullar changes ($p < 0.05$), grade ($p < 0.001$), and vascular risk factors ($p < 0.001$). Nonsignificant trends for dizziness were found for MRI brainstem infarctions ($p = .07$) and for left angle ($p = .09$).

Of the initial 40 symptomatic subjects, 60% were taking anticoagulant and antiplatelet drugs (X^2 (1) = 26.54, $p < 0.001$). None of the comparison group were taking such medications.

Vascular risk factors were noted in 87.2% of the symp tomatic group compared to 13.3% of the comparison group (X^2 (1) = 37.37, $p < 0.001$). MRI cortical microvascular changes were seen in 87.2% of the symptomatic group whereas 26.7% of the comparison group displayed these changes (X^2 (1) 26.07, $p < 0.001$). Specific brainstem cerebellar, and thalamic infarctions were identified in 48.7% of the symptomatic but only 6.7% of the comparison group ($X^2(1)=14.16$, $P < 0.001$).

Significant vertebral artery compression (grades 3/4) induced by neck angulation was noted in 56.4% of the symptomatic group and 13.3% of the comparison group (X^2 (1) = 13.40, $p < 0.001$). Flow analysis revealed a sig.nificant reduction of vertebral artery flow with the neck inhyperextension and II compression. With the neck in neutral position, the basilar artery perfusion rate was less than 20 ml/min in 46% and less than 25 ml/min in 64%. Unsuspected "steals" (reversal of flow) were identified in 4 patients.

Additionally, poststudy dizziness was disproportionately seen in 68% of the symptomatic group and only 13.3% of The comparison group (X^2 (1) = 16.34, $p < -0.001$).

Hypoplasia was defined as two-thirds reduced vessel size visual measurements. A 13% incidence in the symptomatic group as compared to 3% in the control -,roirp was detected. A right-sided preponderance was noted in 88% of cases. However, right and left vertebral flow analysis detected a 50% reduction of blood flow in 42% symptomatic patients when the neck was in neutral position.

Discussion

To the authors' knowledge, the present investigation is the devoted to in vivo detection of functional hemodynamic impairment associated with neck motion utilizing dynamic MRA. The demonstration that head rotation can consistently produce radiological

compression and occlusion of the contralateral vertebral artery at the AA/OA junction in symptomatic patients indicates that this may be an *independent* cause for arterial damage and stroke. The brunt of the mechanical impingement has been hypothesized as secondary to a downward shift of the contralateral atlas while the vertebral artery segment is fixed. The individual's ischemic threshold depends on numerous factors, including speed and duration of' movements, intactness of collateral blood flow, and extent of atherosclerosis. The provocation of neuronal ischemia (dizziness, vertigo, ataxia) by critical neck angulation was seen in 68% of the symptomatic group and 13% of the control group, and statistically correlated with female sex. high-grade mechanical compression (grades 3 and 4), Vascular risk factors, and abnormal MRIs. Both direct effect of mechanical Occlusion and intimal injury are factors. Repetitive movements during activities of daily living (ADLs) may induce intimal changes (i.e., stretching, spasm, dissection) [8], thereby exposing collagen to circulating platelets and provoking aggregation . This intimal injury has been considered the necessary precondition for future thromboembolism [9].

Anatomical variations of the vertebral arteries are common for example, 13% of the symptomatic group displayed visually a hypoplastic vertebral artery with right-sided predominance. Flow analysis disclosed a 50% reduced volume in one vertebral artery when the neck was in the neutral position in 42% of symptomatic subjects, irrespective of grade. This skewed frequency as well as the consistent pattern of contralateral occlusion (grade 4) with neck rotation identifies a significant cohort at risk. Formerly considered rare, its true incidence is unknown. Although the sample size (N = 101) is too small to make statistical statements, the finding is disturbing. Anecdotal reports of stroke induced by Chiropractic Manipulation suggest an association with a hypoplastic vertebral artery or PICA termination [10]. Thus, individuals with hypoplasia or significant asymmetry of flow who previously would have remained undiagnosed can now he safely identified by MRA and educated to the potential risk of contralateral rotation and hyperextension of the neck. Although conventional cerebral angiography remains the " gold standard" for detecting occlusive lesions [11], it is invasive and expensive and does not detect the physiological abnormalities that may be precipitating symptoms. The noninvasive MRA may become the test of choice in evaluating cerebral hemodynamics. In the absence of criteria for ideal positioning, the first author developed a rigorous protocol using as the end point muscular discomfort rather than ischemia. Its utility appears to be justified by the high degree of positive findings (41/64; 64%) mimicking symptoms compared with the findings using traditional neutral views (2/40). Ruotolo and coworkers [12] using dynamic arteriography, were able to explain VBAI symptoms in up to 50% of patients, whereas conventional views were not diagnostic . Thus, this modification, mimicking ADLs, should represent the *routine* method of testing individuals at risk. Current conventional positions are inadequate and may provide a false sense of security if findings are reported as "negative." With rigorous application and blood flow analysis, MRA may replace angiography as the gold standard. A major concern of this "dynamic" technique is the risk of inducing vertebral artery occlusion or dissection. Anecdotal reports described occurrence of

stroke after 10 minutes[13], 30 minutes [14, 15], and hours of sustained positioning[16]. In the present authors' initial study, the dynamic positioning for 11 to 13 minutes per view (total of 33-39 min) elicited temporary neurological symptomatology without evidence of watershed infarction. dissection, and so on. Protocol refinements further reduced scanning time to 6 minutes per view (total, 24 min). Sustained ischemia for longer than15 minutes appears to be poorly tolerated and may lead to infarction The functional disturbance (ischemia) is transitory and represents a marker for potential circulatory deficiency. However, timely reperfusion and/or restoration of blood flow occurred with this vertebral artery challenge test. This suggests a threshold phenomenon since symptoms did not develop in some symptomatic individuals (36%) and control subjects (66.7%). The physiological basis for this greater susceptibility in middle-aged or older subjects, especially females, is incompletely understood, but may be dependent on the presence of osteoarthritis. atherosclerosis,aspects of speed, rapidity, duration, and so on. While safety cannot be totally assured, there was no evidence of permanent neuronal compromise in the 68% of the symptomaticgroup displaying poststudy symptoms.

Neck trauma has been invoked as a mechanism in the development of dissection in both the vertebral artery [17] and the internal carotid artery [18]. Spontaneous dissectiondid not occur in this series. Hemodynamically, there was no change in caliber of 202 carotid arteries, with mild com pression in 1 artery. This disagreement can probabiv be explained by selection bias of autopsy series. more extensive angulation, and possibly inaccurate estimation.

Warfarin, aspirin, and dipyridamole have been the mainstays of stroke prevention and were used in 70% of the symptomatic group. They did not confer protection again mechanical compression and occlusion produced by contralateral rotation. Since avoidance may be the only method of prevention, education of individuals at risk is important. The optimum degree of rotation and hyperextension is ill-defined and appears to be unique for each individual. Although many motions are considered trivial, they may be significant on individual and a repetitive basis. In the elderly, the safe margin may be indeed guite narrow [19].

Vertebral artery dissection and stroke have been associated with a number of potentially provocative positions such as protracted dental work [20], intubation,[21-24], x-ray positioning [25], and chirooractic manipulation [26, 27]. Likewise, flow-mediated reductions have induced stroke, according to anecdotal reports (28-33].

The functional significance of the AA/AO stasis-hypoperfusion defects, and low basilar artery perfusion quotient is unknown and will require a longitudinal study. However stasis within the vertebral artery can produce small thrombi and subsequent emboli with or without sympto [22-24 34]. Intraarterial embolism has also been observed in subjects with atherosclerotic disease of vertebral artery origin [23, 24, 32, 34].Caplan and Tettenborn [35] estimated that about one-third of VBAls have a

thromboembolic origin .The present data would suggest this to be an underestimate. However, the specific mechanism operant in VBAI is unclear, yet critical neck angulation and atherothromboemboli are not mutually exclusive [36].

The initial visual observations of mechanical compression were strengthened by the addition of quantitative 2D phase-contrast MRA techniques. This novel technology provides reliable and accurate volume flow rates in milliliters per minute, which reflects end-organ perfusion [37]. Flow velocity changes (cm/sec) were also recorded but varied depending on vessel diameter and other factors. Flow pattern disturbances (i.e., steal and enhanced collateral flow through the circle of Willis) were quantified. Measurements were taken with the neck in four different positions and with all grades of compression at the proximal and distal vertebral arteries and basilar artery levels. The accuracy of 2D phasecontrast [37-39] and 2D TOF sequences reliably correlated with conventional angiography [28, 40-42].

By virtue of its unique in vivo depiction of blood flow, the results of dynamic MRA may serve as an *important modifier* of human behavior with avoidance of provocative positioning.

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