Mechanism of loss of consciousness during vascular neck restraint

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Mechanism of loss of consciousness during vascular neck restraint. J Appl Physiol 112: 396–402, 2012. First published November 17, 2012; doi:10.1152/japplphysiol.00592.2011.—Vascular neck restraint (VNR) is a technique that police officers may employ to control combative individuals. As the mechanism of unconsciousness is not completely understood, we tested the hypothesis that VNR simply compresses the carotid arteries, thereby decreasing middle cerebral artery blood flow. Twenty-four healthy police officers (age 35 ± 4 yr) were studied. Heart rate (HR), arterial pressure, rate of change of pressure (dP/dt), and stroke volume (SV) were measured using infrared finger photoplethysmography. Bilateral mean middle cerebral artery flow velocity (MCAVmean) was measured by using transcranial Doppler ultrasound. Neck pressure was measured using flat, fluid-filled balloon transducers positioned over both carotid bifurcations. To detect ocular fixation, subjects were asked to focus on a pen that was moved from side to side. VNR was released 1–2 s after ocular fixation. Ocular fixation occurred in 16 subjects [time 9.5 ± 0.4 (SE) s]. Pressures over the right (R) and left (L) carotid arteries were 257 ± 22 and 146 ± 18 mmHg, respectively. VNR decreased MCAVmean (R 45 ± 3 to 8 ± 4 cm/s; L 53 ± 2 to 10 ± 3 cm/s) and SV (92 ± 4 to 75 ± 4 ml; P < 0.001). Mean arterial pressure (MAP), dP/dt, and HR did not change significantly. We conclude that the most important mechanism in loss of consciousness was decreased cerebral blood flow caused by carotid artery compression. The small decrease in CO (9.6 to 7.5 l/min) observed would not seem to be important as there was no change in MAP. In addition, with no significant change in HR, ventricular contractility, or MAP, the carotid sinus baroreceptor reflex appears to contribute little to the response to VNR.

unconsciousness; baroreceptor

VASCULAR NECK RESTRAINT (VNR) is a technique that police officers may employ to control combative individuals. When properly applied, the typical subject loses consciousness within 5–11 s, followed by a full recovery with no lasting medical complications (15, 16). The technique applies lateral compression to the vascular, muscular, and neural structures of the neck, suggesting several physiological mechanisms that might play a role in rendering the subject unconscious. We hypothesized that VNR compresses the carotid arteries and decreases cerebral blood flow sufficiently to produce cerebral hypoxia and loss of consciousness (3, 10, 16). However, the carotid sinus baroreceptor reflex may also cause bradycardia and hypotension and decreased cerebral blood flow. The Valsalva maneuver (characterized by forced strain against a closed glottis) might also contribute to hypotension (5). Increased intrathoracic pressure might increase external constraint to the heart and cause blood to pool peripherally, thereby limiting ventricular filling and reducing stroke volume (SV) and arterial blood pressure (11, 12).

To test the hypothesis that simple compression of the carotid arteries is the main mechanism by which VNR causes loss of consciousness, we measured bilateral middle cerebral artery blood flow velocity (MCAV) and bilateral external neck pressures during VNR in healthy volunteers. To assess the potential contributions of various other mechanisms, we collected noninvasive hemodynamic data and air flow measurements.

METHODS

The study protocol was approved by the Institutional Conjoint Health Research Ethics Board. All subjects provided written informed consent prior to their participation.

Twenty-four healthy police officers (27–40 yr of age, 3 women) who had no prior relevant medical history and who were free of medications were recruited to attend one clinical laboratory session. Exclusion criteria included a history of orthostatic hypotension, cardiac or cerebrovascular disease, or cardiac rhythm disturbances. Subjects refrained from consuming caffeine-containing foods or beverages after the previous midnight. Height, weight, and neck circumference were recorded and body mass index (BMI) was calculated. Arterial pressure was measured from the middle finger of the nondominant hand referenced to mid-heart level with an infrared finger photoplethysmograph while heart rate (HR) was measured with an electrocardiogram (Nexfin, model 1, BMEYE Cardiovascular Intelligence, Amsterdam, The Netherlands). Blood flow velocity was measured in the right (R) and left (L) MCA by transcranial Doppler ultrasound (TCD; TCD100M, Spencer Technologies, Seattle, WA) using probes positioned bilaterally against the temporal skull above the zygomatic arch. Once a satisfactory velocity signal was obtained, the probes were fixed in position using a cranial harness. Bilateral external neck pressures were measured using flat, fluid-filled balloons taped in place over the carotid bifurcations and connected to pressure transducers (model P23 ID, Statham Gould, Oxnard, CA). The balloons were placed at the projected intersection of the superior border of the thyroid cartilage and the lower angle of the mandible (8). In 12 subjects, additional balloons were taped in place over the largest projection of the L and R sternocleidomastoid muscles (SCM). To monitor respiration, nasal airflow was recorded with an airflow pressure sensor connected to a nasal cannula (Ultima Dual Airflow Pressure Sensor, model 0585, Braebon Medical, Carp, ON).

Experimental protocol. After instrumentation, baseline data were collected for 60 s (baseline 1). A single certified VNR instructor from the Calgary Police Service Skills and Procedures Unit positioned himself behind the seated subject placing his right (dominant) arm in VNR position without applying any pressure. As the positioning of the instructor may have changed baseline con-
Table 1. Subject characteristics

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th>Women</th>
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<tbody>
<tr>
<td>Number</td>
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<td>3</td>
</tr>
<tr>
<td>Age, yr</td>
<td>34 ± 4</td>
<td>36 ± 1</td>
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<tr>
<td>Weight, kg</td>
<td>92 ± 13</td>
<td>66 ± 9</td>
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<tr>
<td>Height, m</td>
<td>1.82 ± 0.04</td>
<td>1.72 ± 0.07</td>
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<tr>
<td>Body mass index, kg/m²</td>
<td>27.9 ± 3.6</td>
<td>22.0 ± 1.4</td>
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<tr>
<td>Neck circumference, cm</td>
<td>41 ± 3</td>
<td>34 ± 1</td>
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</table>

Data expressed as mean ± SD.

Table 2. Responses to VNR

<table>
<thead>
<tr>
<th></th>
<th>Ocular Fixation</th>
<th>Subject Stop</th>
<th>Instructor Stop</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>16</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Time, s</td>
<td>9.5 ± 0.4</td>
<td>11.0 ± 3.8</td>
<td>23.3 ± 2.1</td>
</tr>
<tr>
<td>Time to minimum</td>
<td>1.6 ± 0.3</td>
<td>0.7 ± 0.0</td>
<td>3.4 ± 0.9</td>
</tr>
<tr>
<td>MCAVmean, s</td>
<td></td>
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</table>

Data expressed as means ± SE. MCAVmean, middle cerebral artery flow velocity.
respiratory efforts (small airflow deflections) during VNR. On VNR release, phasic inspiration and expiration resumed in both examples.

Figure 3, A and B, shows that R and L MCAVmean decreased in both the Ocular Fixation and Instructor Stop Groups during VNR, compared with baseline 2 values. On release of VNR, R and L MCAVmean promptly returned to baseline 2 levels and remained constant. There was no change in MAP (Fig. 3C). In the Instructor Stop Group, HR decreased during VNR and remained depressed compared with baseline 2 values (Fig. 3D). SV decreased during VNR in the Ocular Fixation Group (Fig. 3E).

Figure 4 shows percent changes in hemodynamic measurements resulting from the application of VNR. CO decreased similarly in both the Ocular Fixation and the Instructor Stop Groups. However, note that these decreases in CO resulted from different changes in HR and SV. MAP and dP/dt were not different between groups and did not change during VNR. Both groups had reductions in R and L MCAVmean but L MCAVmean decreased more in the Ocular Fixation Group compared with the Instructor Stop Group.

External bilateral neck pressure. Figure 5 shows pressures over the carotid arteries and the SCM muscles in both groups. In the Ocular Fixation Group, pressure over the R carotid was greater than over the L carotid while pressure over the L SCM was greater than over the R SCM. Similar trends were seen in the Instructor Stop Group. There were no differences between the groups at either the carotid artery or SCM muscle locations. Note that pressures over the SCM are underestimated because values could only be measured up to 600 mmHg; when

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Fig. 1. A and B: positioning of the vascular neck restraint (VNR) instructor in relation to the subject and the bilateral positioning of the balloon pressure transducers over the left (L) and right (R) carotid bifurcations and sternocleidomastoid (SCM), respectively.

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Fig. 2. Ocular Fixation Group (A) and Instructor Stop Group (B) illustrating representative responses in beat-to-beat changes before, during, and after VNR. The vertical hatched lines indicate the start and release of VNR. MCAVmean, mean middle cerebral arterial velocity (cm/s); MAP, mean arterial pressure (mmHg); HR, heart rate (beats/min); SV, stroke volume (ml); dP/dt, rate of change of pressure (mmHg/s).
Anthropometric relationships. Figure 6, A and B, respectively, shows the relations between time to ocular fixation and neck circumference and BMI for each subject in the Ocular Fixation Group. There was a tendency ($P = 0.08$) for those subjects with a larger neck circumference to fixate more quickly (Fig. 6A). There was an inverse relation between time to ocular fixation and BMI (Fig. 6B).

There was no relationship between the changes in R and L MCAVmean and BMI or external carotid pressures (data not shown).

**DISCUSSION**

Our results demonstrate that the most important mechanism causing unconsciousness during VNR is decreased cerebral blood flow due to bilateral carotid artery compression. The substantial decrease in MCAVmean (Ocular Fixation Group: R 83%, L 80%) can be presumed to translate into an acute period of cerebral ischemia sufficient to render subjects unconscious. Thus 11 subjects in the Ocular Fixation Group had no detectable flow on at least one side while six subjects had no detectable flow on both sides. The small decrease in CO (9.6 to 7.5 l/min) observed would not seem to be important as there was no change in MAP. In addition, with no significant change in HR, ventricular contractility, or MAP, the carotid sinus baroreceptor reflex appears to contribute little to the response to VNR.

Carotid artery compression. Interruption of cerebral blood flow rapidly produces cerebral hypoxia, and loss of consciousness can occur within seconds (3, 10, 16). Cerebral oxygenation has been related to cerebral perfusion as indicated by transcranial Doppler flow velocity (10, 19). Njemanze (13) determined that if mean flow velocity is reduced by more than 50%, unconsciousness would ensue. In our study, 16 subjects (Ocular Fixation Group) were rendered unconscious with an 83% and 80% reduction in R and L MCAVmean, respectively. The four subjects who did not lose consciousness (Instructor Stop Group), had a 74% reduction of R MCAVmean, but only a 40% reduction in L MCAVmean. Thus our data support the
changes in MAP and the small decrease in CO observed have affected hemodynamics. However, the absence of the question of how changes in intrathoracic pressure might measure intrathoracic pressure, we cannot directly address of subjects, breath holding likely occurred. Since we did not indicate that the trachea was patent and that, in the majority respiratory effort and therefore a breath hold occurred during the application. In the small number of subjects who did exhibit respiratory efforts during VNR, the presence of airflow could contribute to a loss of consciousness. Despite a properly applied VNR protecting the patency of the trachea, we measured zero airflow in most of our subjects suggesting an important vasovagal response to VNR. We could not confirm the findings of Reay and Holloway in that the critical lower limit of MCAVmean of 50% below baseline must be met bilaterally to induce unconsciousness.

The Valsalva maneuver. Reduced venous return sharply lowers SV and blood pressure during Phase II of a Valsalva maneuver. Although most normal subjects do not lose consciousness during a Valsalva maneuver, decreased blood pressure could contribute to a loss of consciousness. Despite a properly applied VNR protecting the patency of the trachea, we measured zero airflow in most of our subjects during VNR. This could be explained by a lack of respiratory effort and therefore a breath hold occurred during the application. In the small number of subjects who did exhibit respiratory efforts during VNR, the presence of airflow indicates that the trachea was patent and that, in the majority of subjects, breath holding likely occurred. Since we did not measure intrathoracic pressure, we cannot directly address the question of how changes in intrathoracic pressure might have affected hemodynamics. However, the absence of changes in MAP and the small decrease in CO observed suggests that a Valsalva maneuver did not contribute substantially to loss of consciousness.

Clinical effects of VNR. Ocular fixation with VNR was commonly associated with brief periods of myoclonic jerking and, in some subjects, the eyes turned upward. Many subjects reported a narrowing of the visual field with color changes or having entered into a dreamlike state. Importantly, recovery occurred almost immediately on release with no observed or reported negative side effects. Nine subjects sensed mild tracheal compression and one subject noted slight residual neck pain at the conclusion of the experiment. As previously described, despite most subjects showing no air flow during VNR, some subjects had infrequent respiratory efforts with measured airflow, thus confirming tracheal patency.

Practical implications. We were able to quantify the pressure applied by a police instructor over the carotid bifurcation and, in 12 subjects, simultaneously measured pressure over the largest portion of the SCM muscle. For consistency, we purposely used the same right hand-dominant instructor to apply all VNRS. This translated into a greater pressure over the R vs. L carotid and L vs. R SCM (statistically significant in the Ocular Fixation Group; Fig. 5). This pattern of pressure measurements may vary among police officers. Since pressures over the R and L carotids and SCM did not correlate with the decrease in MCAVmean (greater applied pressure did not result in a greater decrease in MCAVmean) in either artery (data not shown), we do not anticipate that the effectiveness of a properly applied VNR to change if similar pressures are achieved, i.e., if mean bilateral external neck pressures are maintained at greater than ~100 mmHg, there should be an effective decrement in MCAVmean leading to unconsciousness in most subjects.

Relations between VNR and body habitus. There was a negative correlation between time to ocular fixation and BMI in that critical lower limit of MCAVmean of 50% below baseline must be met bilaterally to induce unconsciousness.

Carotid sinus baroreceptor reflex. The lack of an important decrement in contractility (dP/dt), SV, and CO implies little or no important contribution of the carotid sinus baroreceptor reflex to loss of consciousness during VNR. The absence of correlations between changes in HR, SV, MAP, and CO and the decrement in MCAVmean or time to ocular fixation supports this conclusion (data not shown).

We could not confirm the findings of Reay and Holloway in which two of five subjects who had the greatest decrease in HR also had the shortest time to minimum flow, suggesting an important vasovagal response to VNR. We found no significant correlation between decreased HR and time to minimum MCAVmean (r = 0.44, P = 0.09) or time to ocular fixation (r = 0.07, P = 0.79; data not shown). Perhaps the mandatory termination of VNR before ocular fixation and use of facial blood flow velocity as a surrogate for brain perfusion in their study account for the different results.
pressure and decreased MCAVmean (data not shown) does not support that speculation. Although it is possible that vertebral arterial blood supply was compromised during VNR, we find this unlikely. Licht et al. (9) measured blood flow at the unprotected arterial region midway between the origin of the vertebral artery from the subclavian artery and its entry into the foramen of the sixth transverse cervical process and found no significant volume flow changes for four multiple rotational head positions. What appears to be more relevant to VNR is a forward tilting of the head in the mid-sagittal plane. Jargiello et al. (7) found no instances of vertebral artery compression using power Doppler imaging during head anteflexion in 428 patients when symptoms of vertebrobasilar insufficiency were brought on by specific head positioning. Although the above studies only measured vertebral arterial flow with different head positions without external neck compression, it would seem that it would be difficult to compromise vertebral arterial flow during VNR.

Limitations. There are several limitations to the study. First, we recognize that the SV measurements were obtained noninvasively and not validated against an invasive, calibrated flow measurement. However, Harms et al. (4) showed nonsignificant differences throughout a range of SV changes induced by an orthostatic stress when comparing invasive thermodilution-determined SV to noninvasive Modelflow SV finger pressure measurements. Second, we used mean flow velocity as a surrogate for cerebral perfusion assuming laminar flow and that the vessel diameter did not change enough to explain the profound decreases in flow velocity. Third, the recorded minimum MCAVmean for the 5-s period did not necessarily occur exactly at the time of ocular fixation and was taken to accurately represent the period (mean time 9.5 ± 0.4 s; Table 2). Most subjects did not experience minimum flow for at least 1–2 s after VNR application, implying that the 5-s period does represent the important 6- to 7-s period during VNR. Moreover, Fig. 2A shows a representative example of beat-to-beat changes that demonstrate minimal flow changes occurring exactly at ocular fixation (1–2 s preceding VNR release). Last, we could not explain why four subjects were resistant to loss of consciousness, or more specifically, why they only had a 40% reduction in L MCAVmean. Presumably, anatomical differences in neck structures may have accounted for the differences in MCAV, despite no significant differences in applied neck pressure. Moreover, since we could not measure vertebral artery blood flow, we cannot exclude between-group differences. We also cannot explain why the Instructor Stop Group had a significant decrease in HR and not SV while the Ocular Fixation Group showed the opposite changes. The small number of subjects in the Instructor Stop Group make such comparisons difficult.

Conclusions. Although many police agencies have adopted VNR, there are no studies describing the physiology of VNR with only two early limited investigations (15, 16). Our results indicate that the most important mechanism in causing unconsciousness was decreased cerebral blood flow due to carotid artery compression. The slight decrease in SV and CO should not be considered physiologically important enough to cause unconsciousness. Since there were no significant changes in HR, ventricular contractility, or arterial pressure, the carotid sinus baroreceptor reflex appears to contribute little or nothing to the response to VNR. With the majority of subjects rendered unconscious and, importantly, no serious adverse events in our subjects, we conclude that VNR is a safe and effective force intervention; however, outcomes could vary in different populations (i.e., unhealthy or older subjects).

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DISCLOSURES
No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS
REFERENCES


