Vascular Neck Restraint Holds

For the purpose of this discussion vascular neck restraint holds will refer to techniques that apply pressure to the vascular structures of the neck - without applying pressure over the trachea or wind pipe. The primary vascular structure affected by Vascular Neck Restraints are the carotid arteries, indeed the origin of this name links directly back to the effect Vascular Neck Restraints have. Carotid comes from the Greek word καρότιδες, the pleural of καρότις 'drowsiness', this in turn comes from καρούν 'stupefy' - because compression of these arteries was thought to cause stupor.

Anatomy:

Common carotids are arteries that supply most, but very importantly, not all of the blood to the head and neck. There is a minor difference in the origin of the carotid arteries, however for the most part the arteries are identical right and left. Following is a description of the anatomy and origin of the arteries beginning at the left ventricle of the heart.

Oxygenated blood leaves the heart at the left ventricle, flowing into the largest artery of the body - the aorta. From the left ventricle the aorta goes up towards the head (ascending aorta), arches (aortic arch) and then descends into the body (descending aorta) to supply the lower body and legs. It is at the aortic arch that the arteries supplying the upper limbs, head and neck arise. The first artery to leave the aortic arch is the innominate or brachiocephalic artery, (brachiocephalic loosely translates as arm head) and this is a short artery which bifurcates (divides) into the right subclavian and right common carotid arteries. The next artery to leave the aortic arch is the left common carotid artery, followed by the left subclavian arteries. See Figure 1
In its lower section the common carotid artery is deeply set in the neck, well protected by muscles. As the artery progresses up the neck it becomes more superficial. So as by the time the common carotid bifurcates into the internal and external carotid arteries at approximately the level of the fourth neck vertebrae, it has become palpable with minimal protection. This level is just below the point of the chin (see figure 2).

Figure 2 - Fourth Cervical Vertebrae (highlighted in red)

After bifurcation the internal carotid arteries travel upwards, becoming deeper and more protected on their way to the brain. External carotid arteries remain quite superficial bifurcating at approximately the level of C2 into the maxillary artery and the superficial temporal arteries. The maxillary artery supplies the deep structures of the face whilst the superficial temporal arteries supply the face and scalp. (Figure 3)

Figure 3 – Arteries of the Neck
Physiological effects of Vascular Neck Restraints;

Vascular neck restraints affect more than just the carotid arteries. Other structures affected with these techniques include: the nervous system, muscles, bones, cartilage and the jugular veins. However the main structure for rendering the subject unconscious is the carotid arteries.

It is extremely important to note that VNR will not completely cut off the blood supply to the brain (cerebral anoxia) of a healthy person. This is due to the fact that the vertebral arteries also supply (albeit a lesser volume) blood to the brain. These arteries branch off the subclavian arteries and then progress to the head via holes or foramen in the vertebrae of the neck (Figure 4). As such they are highly protected from external forces by the bony structure of the vertebrae. Once within the skull there is a complex interrelationship of the arteries, such that if there is restriction or occlusion in a major artery the supply can be somewhat accommodated for by arterial connections (Figure 5). LOC occurs as a method of the body protecting the most important parts of the brain, i.e. higher level functioning and consciousness is sacrificed to ensure sufficient blood supply is directed to the brain stem to ensure vital functions (breathing, heart beat etc.) are sustained.

There have been many ideas put forth to explain what happens when the carotid arteries are compressed. Unfortunately many of these are hypothetical with little or no evidence to support the fact.

Research conducted by Mitchell et al. \(^1\) looked into possible causes of unconsciousness listing the following as the more likely causes they wanted to test;

Hypothesis tested:

1. VNR compresses the carotid arteries and decreases cerebral blood flow sufficiently to produce cerebral hypoxia* and Loss of Consciousness (LOC).

* Cerebral hypoxia and hypoxia are two distinct conditions.

Hypoxia is a reduction in the amount of oxygen in the blood supplying an organ. Hypoxia occurs in instances such as high altitude where the air is thin (reduced partial pressure) and the body is unable to perform efficient gas exchange between the lungs and blood stream. This results in decreased oxygen saturation - \(O^{2}\)sat (the percentage of oxygen the blood is carrying compared to the maximum it can carry).
Cerebral Hypoxia is caused by an event that reduces the brain's ability to receive or process oxygen. There are different types of cerebral hypoxia;

- Hypoxic hypoxia - caused by a hypoxia i.e. a decrease in $O_2$ sat in the arterial blood, e.g. Divers ascending from deep water dives, exercise at high altitudes, asthma attacks.
- Hypemic hypoxia - inadequate oxygenation of the blood despite adequate oxygen in the environment, e.g. CO poisoning, anemia.
- Ischaemic hypoxia - inadequate blood flow to the brain, e.g. fainting, VNR.
- Histotoxic hypoxia - $O_2$ is present in the brain but cannot be metabolised by the brain tissue e.g. cyanide poising.

A very clear clinical difference between hypoxic and ischaemic hypoxia is that whilst loss of consciousness via hypoxic hypoxia can take minutes, loss of consciousness via ischaemic hypoxia takes seconds.

2) Carotid sinus baroreceptor reflex may cause bradycardia (slowed heart rate) and hypotension (decreased blood pressure) and decreased cerebral blood flow.

Baroreceptors are simply receptors that monitor pressure, just as a barometer measures pressure in the atmosphere. Carotid baroreceptors are located in the carotid sinus, which itself is located at the bifurcation of the carotid artery. These receptors monitor the blood pressure in the carotid arteries and send the information to the brain stem – if pressure drops, the brain stem responds by stimulating the heart to increase its rate and stroke volume. If pressure rises then the brain stem response is to slow the heart rate and lower stroke volume. This is an extremely fast acting and sensitive system which prevents large swings in blood pressure due to external stimuli. A good example of how quickly this system adapts is when on occasion you quickly stand up and get light headed (postural hypotension), the system is fast and sensitive enough to respond so you do not lose consciousness, nor does your blood pressure spike too high.

3) The valsalva maneuver (moderately forceful attempt to exhale against a closed airway) might contribute to hypotension via an increase in intrathoracic pressure putting pressure on the heart and reducing the hearts ability to fill, leading to a reduction in stroke volume (the amount of blood pumped in each beat of the heart), with a decrease in the arterial blood pressure.

Method of Testing;

Testing involved placing fluid filled balloons over the carotid bifurcations, to measure pressure, and then a certified VNR instructor maximally performed a VNR on the subjects. The VNR was performed until ocular fixation occurred (subject not able to follow the movement of a pen with their eyes) or the subject indicated a desire to terminate the experiment or after a prolonged period (approx 20 seconds) without ocular fixation.

The results of increased the study were as follows;

1) Rapid decrease in middle cerebral artery flow velocity in the order of 80% reduction (measured by transcranial Doppler ultrasound) – measuring the flow of arterial blood within the head. With rapid recovery in arterial flow, post restriction.

2) No significant change in mean arterial pressure.

3) No significant change in heart rate.

4) Slight decrease in stroke volume.

5) Most subjects recorded zero airflow during VNR despite the airway being unobstructed, indicating they were most likely holding their breath.
Clinical effects of VNR

1) Occular fixation - inability to continue to follow the movements of a pen with the eyes, despite the eyes being open
2) Brief periods of myoclonic jerking (involuntary twitching of a muscle or group of muscles)
3) Eyes turning upwards in some subjects
4) Narrowing of visual field with colour changes
5) Entering dreamlike state
6) Near immediate recovery with release of VNR with no observed or reported adverse effects

Conclusions drawn from the study were that;

1) “the most important mechanism causing unconsciousness during VNR is decreased blood flow due to bilateral artery compression”, i.e. Ischaemic cerebral hypoxia.
2) The lack of change in mean arterial pressure, heart rate and minimal change in stroke volume excludes baroreceptor reflex from being a significant contributor to LOC.
3) The absence of change in mean arterial pressure and only a small decrease in cardiac output suggest Valsalva manoeuvre did not contribute greatly to LOC.
4) VNR is a safe and effective force intervention

In summary whilst the blood flow to the brain is generally well protected, there is a specific region in the neck where the arteries are superficial and protection is minimal. VNR techniques exploit this area to compress the arteries and significantly reduce blood flow to the brain. This restriction in blood flow appears to be the primary mechanism through which LOC is induced. Properly performed VNR does not seem to have any significant side effects, and recovery from LOC is rapid following VNR application.
References.

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Further Reading

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