Various Forensic Authors' Commentary on Sudden Vagal Death, Jay Wiseman (2010)

I continue to believe that the best way to move this discussion forward is to provide information. One source of information that I have more access to than many others do is forensic pathology textbooks. As I've mentioned a time or two, I've had the experience of having several such textbooks simultaneously open on a table in front of me to the same general sections, noting the similarities and differences in what the various authors have to say. It's made for interesting reading.

What follows below are excerpts from six different forensic pathology textbooks (other than Knight). The page upon which excerpt appears is included. The excerpts cover matters such as asphyxia in general, and vagal-induced cardiac arrest from a multiplicity of causes specifically. They also deal at some length with compression of the neck by various means, and the results thereof. I think it can be quite fairly said that there is a lot of "back and forth" in the literature on this subject. With this material, I hope to share some of that back and forth.

Given the sheer size of the overall material, these excerpts necessarily involved something of a judgment call on my part. Undoubtedly, a different person would have chosen to excerpt at least some material differently. That said, I think the material below does represent a reasonable cross-section of the debate in the forensic pathology literature, particularly on the question of whether or not sudden, vagal-induced death from being strangled or choked is or is not a reasonably foreseeable result. To help ensure that "the other side" was reasonably well represented, I have pointedly included some writings in their entirety which say that such a death is not reasonably foreseeable.

I have presented the material below without editorial comment. The only alterations I have made are 1) when there was not particularly relevant material between two sentences in the same paragraph, I put in three dots...like this, to show that some material was omitted, and 2) when the context of the commentary might not have been clear, I have included some brief explanatory material in brackets, for example: choking [such as on a piece of food].
I hope you find this material interesting and useful.

"Pathology of Neck Injury" by Vanezis. Copyright 1989
Chapter 5 -- Compressive neck injury I: Signs of mechanical asphyxia (Pg. 44)
Death usually results from a combination of mechanisms involving the various structures which are compressed, i.e. airway, blood vessels and nerve structures. The most common mechanism is a direct result of acute airway and vascular obstruction, rather than by airway, vascular, or nerve compression alone. Sawaguchi et al (1976) carried out selective compression on unanesthetized rabbits and dogs and reached the following conclusions. When strangulation (predominantly airway obstruction) was employed the cause of death was acute respiratory and ventilatory failure due to anoxemia, hypercapnia, and acute respiratory acidosis. With cervical blood vessel compression alone (internal jugular, common carotid artery, and vertebral artery) the cause of death resulted from disturbance to the cerebral circulation and paralysis of the vasomotor nerves. With pressure on the cervical nerves (vagus, phrenic, cervical, and brachial plexus and sympathetic trunks) the cause of death was cardiac (the ECG showed absent P waves with is strong evidence of vagal inhibition). Pulmonary insufficiency also occurred and was secondary to disturbance of the respiratory center.

The time required for death to occur in mechanical asphyxia due to compression is one of the most important medico-legal considerations, and yet one that can seldom be measured with any degree of accuracy.

Extent of struggle, length and force of application (page 61)
The question of how long the assailant's grip needs to be maintained for death to occur is complex, but it is crucial from the point of view of indicating intent.

Occasionally one may find that although death may have occurred very rapidly, the assailant's grip is maintained for a long time. In one case a man admitted squeezing his wife's throat for five minutes, although the autopsy findings suggested that death occurred when the pressure was first applied. (R. v Pye, Essex Assizes 1952, cited by Camps and Hunt, 1959).

Survivability and complications (page 61)
Complications, however, can arise in survivors and be potentially fatal. This is especially the case where laryngeal fractures are unrecognized, particularly in strangulation cases because they are usually present without obvious mucosal disruption of submucosal hematoma formation. Delayed life-threatening airway obstruction, or long-term vocal disruption, may be the end result.

Neurological complications may ensue if the carotid artery has been injured. Milligan and Anderson (1980) encountered two female patients of reproductive age who presented with strokes one day and three days respectively after attempted strangulation by their husbands. Both had sustained a traumatic occlusion (thought to be thrombotic, with possible distal embolization) to the cervical internal carotid artery.

Compression of the neck from neck holds (page 61)
Neck holds are taught in the United States of America by law enforcement agencies as a means of subduing suspects resisting arrest or to control prisoners who are combative and unmanageable.
Two types of hold are generally recognized: the "carotid sleeper" and the bar arm control or "choke hold" (Figure 6.25). The carotid sleeper is promoted as the hold to be used, whereas the choke hold is strongly condemned.

The carotid sleeper (Page 62) The carotid sleeper is designed to compress the common carotid arteries and produce transient cerebral ischemia...With correct application the airway should remain unimpeded during the maneuver. The vertebral arteries, because of their relatively protected position are unaffected by this hold, thus continuing to supply blood to the brain. Permanent neurological damage should therefore not occur even though cerebral ischemia and transient unconsciousness results because the occluded carotid circulation is not completely compensated by the vertebral arteries.

A second factor involved in producing cerebral ischemia with this hold is carotid artery sinus stimulation, which can produce bradycardia and sometimes cardiac arrest (Daly, et al, 1979).
The bar arm control or choke hold (Page 62-63)

The choke hold is designed to occlude the airway by forearm compression of the exposed anterior aspect of the neck. The technique is similar to the carotid sleeper, except that the forearm is placed directly over the airway.

The situation may be made worse by the resistance of the person on whom the hold is applied. By struggling to remove the obstruction from his (or her) neck and improve his breathing he inadvertently contributes to increasing the force on his neck. Death in such cases can result from hypoxia with its attendant cardiac arrest. In this respect, the role of the carotid body is important; carotid body chemoreceptors are stimulated by a reduction in Pa02; and also by a rising PaCO2, and hydrogen ion concentration. Any condition of hypoxia which involves excitation of the carotid bodies and which is accompanied by decreased ventilation will enhance chemoreceptor bradycardia and endanger the person's life (Daly et al 1979).
Page (63)

Fatal consequences of neck holds can be anticipated because of their physiological effects. Pre-existing natural disease increases the likelihood of a fatal outcome, even when a hold such as the carotid sleeper is applied correctly.

According to Koiwai (1987), if the chokehold is applied correctly then fatalities should not occur. This has certainly been the case in judo, which since its inception in 1882, has not experienced any fatalities related to such holds...Nevertheless, perfection conditions to apply the hold do not pertain when restraint is attempted, usually on subjects who are at risk particularly from alcohol drug abuse. Furthermore, even if the desired effect is to produce only temporary unconsciousness, albeit for a few seconds, the practice, needless to say, is a very hazardous one.

Mechanism of death [in hanging] (Page 75)
Leaving aside judicial hanging, which is discussed below, one should appreciate that the mode of death in suspension may be complex. These are the main factors to be borne in mind, namely:
Obstruction of the airway
Occlusion of the cerebral circulation
Effect of the vagus nerve Mechanism of death in choking [as in on a piece of food]
Mechanical asphyxia is the most common mechanism of death in choking. Nevertheless, occasionally deaths occur very rapidly with no visible asphyxial signs. When a full post-mortem examination is carried out and there are no physical findings to account for death, one must consider whether death has resulted from cardiac arrest following vagal inhibition. Such a conclusion, however, should not be arrived at lightly, and then only after careful consideration of the circumstances surrounding death.

"Spitz and Fisher's Medicolegal Investigation of Death, Fourth edition" Copyright 2006
(Page 810)
A karate chop to the neck is particularly damaging in tearing the airway regardless of level, causing obstructive edema or instantaneous death as a result of vagal reflex.

Neck holds (Page 818)
Neck holds are used in Judo and by law enforcement and correctional personnel to subdue combative individuals who resist arrest can cause death...Two types of holds are recognized:
[1] Choke hold or arm bar control
[2] Carotid sleeper hold

The use of the choke hold or bar arm control is being discouraged, because of its potential for injury of the airway...Choke holds can cause serious damage or death within seconds. Laryngeal fractures, internal hemorrhage and swelling of the airway lining due to edema may cause delayed complications, hours, even days later.
(Page 821)

In contrast to choke hold, a carotid sleeper hold does not constrict the airway and breathing continues...Significantly less force is required to subdue a combative individual with this type of hold. Despite the apparent harmless-ness of the carotid sleeper hold occasional deaths do occur as a result of application of this form of restraint. Movement during struggle may turn a sleeper hold into a choke hold with serious, sometimes fatal, consequences.
Death Due To Carotid Sinus Reflex (Page 826)
The carotid sinus, situated at the bifurcation of the common carotid artery on both sides of the neck, consists of nerve tissue, stimulation of which regulates blood pressure and pulse rate. Strong pressure on the neck in the area of carotid sinus, can elicit a drop of the blood pressure by as much as 20 mm Hg. in a normal individual.

In older people, with advanced arteriosclerosis involving the carotid arteries and in rare instances of marked hypersensitivity of this structure, excitation of the carotid sinus, as by a fall or blow on the side of the neck may cause fainting, even cardiac arrest. In extreme cases of hypersensitivity of the carotid sinus the individual is unable to tolerate a necktie or tight collar.

Criminal defense attorneys sometimes argue that the death of the victim was unpredictable, without malice or foresight on the part of their client, consequently accidental, in cases of obvious manual strangulation. The following must be considered in determining whether a death may have been caused by carotid sinus stimulation:
1. Deaths as a result of carotid sinus stimulation are infrequent.
2. The victim is likely to be elderly with advanced arteriosclerosis involving the carotid arteries.
3. The victim was known to be susceptible to carotid sinus stimulation with previously documented manifestations of dizziness, headache, weakness and fainting associated with pressure on either side of the neck.
4. The presence of injuries, especially fingernail marks (whether the victim's or the assailant's) on the skin of the neck, usually rules out death by carotid sinus stimulation.
5. Injuries in the soft tissues under the skin should be limited to the areas of the carotid sinus.
6. Absence of petechial hemorrhages in the conjunctivae and the facial skin.

Interestingly, no death due to carotid sinus stimulation as a result of attempted self-strangulation has as yet been reported.

Since Brouardel (1896) the term carotid sinus reflex has permeated the forensic literature and served as argument in the defense of strangulation. However, it remains a mystery of how a claim of death due to carotid sinus reflex may be applicable to cases of obvious strangulation. Carotid sinus stimulation was a defense claim in the Preppy Murder trial in New York City (1987).

Many, in fact probably most cases of strangulation in women are sex related and we have often heard the defense claim in cases of obvious manual strangulation that the death was unexpected during amorous foreplay,without intent to cause harm.

No doubt sudden cardiac arrest elicited by carotid sinus stimulation is a serious and valuable defense tool. However, such a claim appears persuasive only at face value. A closer view of the autopsy findings and the infrequency of occurrence of carotid sinus stimulation, as a cause of serious consequences, suggest the contrary.

"Forensic Medicine: Clinical and Pathological Aspects" by Payne_James, et al. Copyright 2003
Causes of Death [by hanging] (Page 266)
The most frequent cause of death by hanging is obstruction of the cervical blood vessels.
Another important cause of death may be the stimulation of the vagus nerve and, more particularly, the fibers responsible for the carotid sinus reflex. Pressure on the vague nerve was used for therapeutic purposes as early as the end of the last century. In cases of cardiac dysrhythmia, the reflex cardiac arrest or tachycardia can be provoked by pressure from the fingertips or massage on the carotid sinus one one or both sides; generally, cardiac contraction started again but some cases of complete, and thus final, cardiac standstill also occurred.

Connections between the superior laryngeal nerve and the vagus nerve may least to intense stimulation of the first leading to simultaneous stimulation of the latter, thus resulting in fatal slowing reflexes on the heart. This should also be borne in mind, especially in cases in which laryngeal trauma is marked.

Throttling (Page 270)
Compression of the neck with one or both hands or by clasping the neck between the upper and lower arm is described as throttling.

If the neck is compressed in a so-called "headlock" or by pressure of the lower arm against the area of the head and neck, thus keeping it immobilized, there may be no externally visible injuries. Also, in cases in which fatal reflexes were triggered from the nerves and nerve plexuses in the neck, strangulation and congestion marks may be present only to a small extent or not at all.

The occurrence of fatal reflexes must always be considered in cases of throttling. Pressure on both the carotid sinus and stimulation of the superior laryngeal nerve and fracture of the larynx have all been suggested as capable of triggering a reflex-induced cardiac arrest. In the clinical literature, a number of cases have been described in which reflex-induced cardiac arrest occurred when therapeutic attempts were made to treat cardiac dysrhythmia by exerting pressure by hand on both sides of the carotid sinus.

Numerous tests on humans and animals have not shown that reflex induced cardiac arrest conclusively and furthermore, the autopsy investigation in such cases and reports in the literature are not conclusive. It is therefore generally considered that unless there happens to be a pre-existing carotid sinus syndrome or a hypersensitive carotid sinus, killing by reflexes triggered in the process of throttling, is unlikely.

"Forensic Pathology Principles and Practice" by Dolinak, et al. Copyright 2005
Vagal stimulation and rapid death [from choking on food] (Page 207)
It is interesting to note that food and foreign bodies can cause death not only by choking, but also rarely by acute and pronounced distension of the esophagus. In this scenario, the mechanism of death is not airway obstruction but rather a vagally mediated event, namely, bardycardia, cardiac dysrhythmia, bronchospasm, seizure, or some other mechanism. The proposed mechanism is esophageal distension-mediated stimulation of tensoreceptoers in the wall of the esophagus, causing vagal outflow that terminates in the medulla, where the impulse pathway overlaps with those of the respiratory and cardiac pathways, causing bradycardia, dysrhythmia, or bronchospasm. This is the mechanism by which significant esophageal distension may elicit a detrimental -- and possibly fatal -- cardiopulmonary response.

Vagal reflexes arise not only from the esophagus, but also from the pharynx and larynx. This may also help explain why in some cases of choking due to upper airway obstruction death appears to ensue quicker than might be expected from an asphyxial event alone.

Carotid Sinus Stimulation (Page 223)
Whenever significant neck compression occurs, there may be compression and stimulation of the carotid sinus, which is situated just cephalad to the bifurcation of the common carotid arteries. Stimulation of this structure may lead to vagal effects, such as bradycardia and hypotension, or may lead to other types of dysrhythmia. The vagal effects may rarely be overwhelming and possibly lead to a rapid onset of cardiac standstill and sudden death. Hence, one may not need to necessarily occlude the airway or the vasculature in order to cause death by neck compression. This may be the mechanism of sudden death in individuals who collapsed immediately during the application of a neck hold or an impact to the neck, and in whom no significant pathologic findings are identified as autopsy. However, because during a struggle, most individuals are tachycardic and hypertensive, vagal stimulation will more likely lead to normal cardiovascular parameters or mild hypo tension and/or bradycardia and not be a factor in causing sudden death. This is likely true because in normal individuals, stimulation of the carotid sinus produces only mild bradycardia and mild hypotension.

The individuals who are believed to be susceptible to rapid vagal death are likely older people with significant cardiovascular disease who were known to be symptomatic from previous episodes of carotid sinus stimulation such as fainting spells or dizziness associated with pressure on their neck. Additional evidence of violence such as abrasions resembling fingernail marks on the victim's neck and significant internal neck injury combined with conjunctival petechiae effectively rules out death by carotid sinus stimulation.

Note on carotid sinus stimulation (Page 310)
A factor to consider with any type of neck compression is stimulation of the carotid sinus, which is located just cephalad to the bifurcation of the common carotid arteries. Compression of the carotid sinus may result in bradycardia and rarely cardiac arrest. As such, carotid sinus stimulation may be used to explain sudden death occurring rapidly after the application of a neck hold. However, most descriptions of vagal stimulation under controlled conditions lead only to mild bradycardia and/or mild hypotension, and because most individuals involved in struggles are tachycardic and hypertensive, vagal stimulation will likely lead only to more normalized cardiovascular parameters, or only mild bradycardia and hypotension.

Death attributed to excessive vagal stimulation is not likely, and if it does occur, is probably restricted to an older individual with significant cardiovascular disease who was known to be symptomatic from previous episodes of carotid sinus stimulation such as fainting spells or dizziness associated with pressure on his or her neck.

"Forensic Pathology" by DiMaio and DiMaio. Copyright 1989
Deaths Occurring Following the Application of Choke or Carotid Holds (Page 248)
Rarely, one will encounter a death alleged to have occurred due to application of either a choke [arm bar control] or a carotid sleeper hold. These terms are often used interchangeably, but, in fact, refer to two different types of hold whose purpose is to produce transient cerebral ischemia and unconsciousness. Neither involves use of a mechanical implement. Rather, the arm and forearm are used to compress the neck, producing the cerebral ischemia and unconsciousness. Occasionally, a baton, large metal flashlight, or some other device will be used to compress the neck. The authors have seen a number of deaths with such instruments with fracture of the hyoid bone or larynx. Since a device is used rather than the arm, they are not really deaths due to choke holds.

With choke [arm bar control] holds, compression of the neck by the forearm is used to occlude the upper airway. Incapacitation is due to collapse of the airway and the carotid arteries with a resultant decrease in oxygen to the brain. The forearm is placed straight across the front of the neck. The free hand grips the wrist, pulling it back, collapsing the airway. If too much force is used, there may be fracture of the larynx or hyoid bone. In the two cases reported in the literature by Reay and Eisele and in a recent case seen by the authors, there were unilateral fractures of the greater cornu of the thyroid cartilage. The authors' case also had a fracture of the hyoid bond on the same side. In the two cases reported by Reay and Eisele, both fractures are n the left side of the neck. These were produced using the right forearm across the neck and the left hand to pull it backward. Thus, pressure was eccentrically transferred to the neck, predominately to the left side. In the case seen by the authors, the left forearm was used and the fractures were on the right side of the neck.

Choke holds can also cause death by another mechanism. In a choke hold, incapacitation is produced by lack of oxygen to the brain. This hypoxia is generalized, however, due to compression of the airway. Hypoxia sensitizes the heart to arrhythmia. The carotid sinus is a structure located in the internal carotid artery, just above the bifurcation of the common carotid artery.

Stimulation of this structure by pressure to the neck can cause bradycardia and/or a fall in arterial blood pressure. Thus, we have two factors working on the heart predisposing to arrhythmias: the hypoxia from occlusion of the airway and the bradycardia from stimulation of the carotid sinus. There is, in addition, a third factor: release of catecholamines. Choke holds are used to restrain an individual who is struggling. On placement of the hold, the individual usually continues to struggle. This results in release of catecholamines, specifically, norepinephrine and epinephrine. These have an arrhythmogenic action on the heart. Thus, the combined actions of hypoxia and catecholamines, which are both arrhythmogenic, plus the bradycardia produced by the carotid sinus stimulation, may result in a fatal cardiac arrhythmia.

In the carotid sleeper hold, symmetrical force is applied by the forearm and upper arm to the sides of the neck such that there is compression of only the carotid arteries and jugular veins and not the trachea. The arm is placed about the neck with the antecubital fossa or crook of the arm centered at the midline of the neck. The free hand grips the writs of the other arm and pulls it backward, creating a pincher effect. This impeded blood flow in the carotid arteries by pressure exerted on both sides of the neck by pincher effect of the arm and forearm. If properly applied, the compression of the carotid arteries will cause loss of consciousness in approximately 10–15 s. On relaxation of the hold, cerebral blood flow will be restored and consciousness will return in approximately 10–20 s, without any serious side effects. Maintenance of the pressure is essentially manual strangulation and if continued long enough will, of course, cause death. Experiments by Reay and Holloway demonstrated that during application of the carotid sleeper hold, blood flow is decreased an average of 85% to the head. The range in five subjects was 82 to 96%. The time to minimum blood flow averages 6 s (range 3.2 to 7.2 s).

In theory, the carotid sleeper hold will cause rapid unconsciousness without injury to the individual. Unfortunately, in violently struggling individuals, a carotid sleeper hold can easily and unintentionally be converted into a choke hold as the individual twists and turns to break the hold.

A properly applied carotid sleeper hold can also cause death. One would not expect any trauma to the structures of the neck, however. The compression of the carotid arteries, with resultant decreased cerebral blood flow, can theoretically precipitate a stroke in an individual with atherosclerotic disease of the carotid and/or cerebral vasculature. The pressure may cause dislodgement of atherosclerotic material with a stroke sue to an embolus. Blood flow to the brain is from both the carotid and the vertebral arteries. If the vertebral arteries have impaired blood flow due to atherosclerosis then occlusion of the carotid arteries may compromise an already compromised circulation with resultant thrombosis and stroke.

Compression of the neck by a carotid sleeper hold may also cause stimulation of the carotid sinus with bradycardia. Application of the hold to an individual who is agitated and struggling may increase the struggling, with increased release of catecholamines. The catecholamines working with the carotid sinus stimulation may produce a cardiac arrest. In addition, if the individual has intrinsic heart disease, he may be even more sensitive to bradycardia and the arrhythmogenic activity of the catecholamines.

All of the aforementioned potential complications of these holds do not mean that they are totally objectionable. The fact is that any action involving force always has the potential of producing severe injury and death. One must weigh how much force is acceptable in a situation.

”Autoerotic Fatalities” by Hazelwood, et al. Copyright 1983
(Page 26)
In a 1942 paper on sudden death, Gardner mentioned the case of a boy ”who had died from hanging during a masochistic experiment.” Although the boy had passed his head through a noose and suspended himself from a beam, Gardner inferred that he had not intended to kill himself, for ”he had arranged an ingenious quick

release device by which he could slacken the rope whenever he decided to do so; but as the rope took the weight of his body, unconsciousness and death took place too quickly for him to have time to use it." (Gardner, 1942) Gardner suggested two likely mechanisms for the rapidity of such deaths: pressure upon the vagus nerve or upon the carotid sinus. Both are consistent with current knowledge of the physiology of hanging, and in either case, death is mediated through vagal stimulation (see Chapter 4).

Hanging and strangulation (Page 60)
In both hanging and strangulation asphyxia results from compression of the neck by a ligature.
It is a common misconception that hanging and strangulation cause death by compressing the airway (trachea)...In the majority of cases, compression of the neck kills through effects on the blood vessels of the neck rather than interference with respiratory exchange through the trachea.
The effects of neck compression on the blood vessels of the neck include interference with the return of blood through the veins to the chest, interference with the supply of fresh blood through the major arteries to the head, and effects not widely known to laymen that involve pressure-sensitive areas in the neck (see the following).

Experiments conducted in Europe in the late nineteenth century and replicated several times since (see Polson 1965, pp. 293-294) have shown that the degree of tension necessary to achieve these effects varies in a predictable sequence. With slight force (less than 5 lbs.) the jugular veins are closed; with somewhat higher force (7 to 11 lbs.) blood flow through the carotid arteries is reduced to a trickle, and then eliminated; and at forces greater than 30 lbs., the trachea and vertebral arteries are closed. Detailed accounts of other effects of neck compression and the signs of death from hanging and strangulation are available in standard textbooks of forensic pathology, although perhaps the best is that in Polson and Gee.

Some unknown portion of deaths from neck compression occur not as a result of obstruction of blood flow in the blood vessels of the neck but rather from the effects of the neck compression on a pressure-sensitive organ in the neck known as the carotid baroreceptor. Just above the division of the common carotid artery into the internal and external carotid arteries, there is a widening of the internal carotid artery known as the carotid sinus. This sinus includes pressure-sensitive cells and chemically sensitive cells. The pressure-sensitive carotid baroreceptors respond to pressure by slowing the heart. Forceful pressure upon this area is believed to result in a precipitous fall in heart rate and blood pressure, causing unconsciousness. The chemically sensitive carotid body responds to changes in oxygen tension in the blood (as well as changes in hemoglobin concentration, carbon dioxide tension, and pH). Under conditions of decreased oxygen (hypoxia), the carotid body generates nerve impulses that increase respiration.

According to Adelson (1974), pressure to the neck overlying the carotid sinuses can cause practically instantaneous death "in the predisposed victim" (p. 526). Although we are unaware of solid evidence that individuals differ in their pressure sensitivity, there is evidence suggesting that normal individuals may differ in chemical sensitivity and that biological responses to hypoxia may be controlled genetically (Hudgel and Weil, 1974); Moore et al. 1976; Mountain et al 1978). Since the carotid bodies apparently have evolved from our reptilian ancestors (Anonymous, 1978), there has been ample opportunity for genetic diversity to develop.
Sudden death resulting from pressure on the carotid sinuses occurs through changes in nerve impulses conveyed by the vagus nerve, one of the major actions of which is to slow the heart. Sudden death conveyed through these vagal impulses is variously known as "death from inhibition," "reflex cardiac death," "instantaneous physiologic death" (Adelson 1974, pp. 526-527), and death from vagal inhibition.
Death from vagal inhibition has been proposed as a mechanism of sudden death in autoerotic asphyxias for at least thirty years (Gardner, 1942). Gardner suggested that the contributory role of carotid-sinus pressure in sudden death was better recognized in North America than England, although writings of the time do not seem to support that green-grass view.

Sexualization Of Strangulation

In any case, Gardner had noticed occasional tears across the carotid sinus in cases of hanging. This observation led him to consult a Japanese instructor in jujitsu in London about the "knockout holds" in jujitsu exercises.
"He told me that he could render anyone unconscious in 3 seconds or kill him in 20 by pressure on the sinus, and he passed a silk belt about two inches wide across the back of my neck and brought the ends in front; then holding one he pressed the other against the side of my neck high up under the angle of the jaw, and as far as unconsciousness went he spoke truly, but I think it happened in under the 3 seconds."

Airway Obstruction [on food and suchlike] (Page 62)
Although the term choking is sometimes used to describe manual strangulation (throttling), we restrict the usage to cases in which the breathing passages have been internally blocked. Although familiar in the form of the cafe coronary, in which an individual's airway is obstructed by food, and among infants and young children, choking is unusual among autoerotic asphyxias. Quiroz-Cuaron and Reyes-Castillo (1971) suggest that the case they reported may have involved obstruction of the airway with a foreign body; they are unclear on this point. The usual mechanism of death by choking is inability of the victim to take in oxygen or expire carbon dioxide. Thus death takes two to five minutes if the airway obstruction is complete and longer if the obstruction is partial. Occasionally, however, choking results in sudden death from cardiac inhibition (Simpson 1965; Polson 1965) since the vagus nerve has sensitive branches around the airways.