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Asphyxial Deaths and Petechiae: A Review

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ABSTRACT: Conjunctival and facial petechiae, although nonspecific findings, are considered hallmarks of asphyxial deaths. Consensus in the literature suggests that their pathogenesis is related to the combined effects of increased cephalic venous pressure and hypoxic damage to endothelial cells. Despite the common knowledge that they are neither predictable findings in all asphyxial deaths nor rare in natural, nonaphyxial deaths, the belief persists that petechiae are corroborative evidence of asphyxia. We suggest that a clear, physiologically based understanding of the pathogenesis of petechiae of the head is critical for their appropriate interpretation. We present a review of the literature and the basis of our conclusion that conjunctival and facial petechiae are the product of purely mechanical vascular phenomena, unrelated to asphyxia or hypoxia.

KEYWORDS: forensic science, petechiae, asphyxia, conjunctival petechiae, facial petechiae

Conjunctival and facial petechiae have been regarded as classic signs of asphyxial deaths (1–11). First described by Tardieu in the nineteenth century, external and visceral petechiae have since been interpreted as hypoxia-related sequelae of asphyxia, sometimes being attributed in part to "mechanical obstruction of the upper airway" (12,13). Despite consensus in the literature that cephalic petechiae are not found in all asphyxial deaths, and that they are observed commonly in natural deaths without an asphyxial mechanism (2–7,13–15), the view that petechiae and asphyxia are causally linked continues to be perpetuated, without regard to the inherent inconsistencies central to that theory. Potential confusion generated by the foregoing misconception bears out most precariously in courts of law, where forensically and scientifically sound pathogenetic conclusions are critical for the proper adjudication of criminal proceedings. In that setting, the presence of conjunctival and facial petechiae traditionally has been used as compelling evidence in support of an asphyxial death, while their absence has cast serious doubt on that possibility. We suggest that a clear understanding of the pathogenesis of petechiae of the head increases the likelihood of their appropriate interpretation.

Many authors have described the incidence of conjunctival petechiae in nonasphyxial and asphyxial deaths, but there is a dearth of literature comprehensively addressing their pathogenesis. Furthermore, much of the literature on petechiae cannot be compared and contrasted because studies report petechiae as either present or absent, one being as good as a million. Such an either/or criterion disregards the reality of common experience and equates a single, subtle conjunctival red dot with showers of conjunctival and facial petechiae too numerous to count. Granted, in rare instances, an isolated petechia may be an important finding in the context of a complete case study, but to base our understanding of an entire subject on three standard deviation outliers is the equivalent of a tiny tail wagging a large dog. Therefore, this paper describes the pathogenesis of conjunctival and facial petechiae on the basis of mechanical phenomena in order to clarify inconsistencies and vagaries in the literature. First we present a review of the literature and then our conclusions regarding petechiae of the head as they pertain to asphyxial and nonasphyxial deaths. We will not address the pathogenesis of visceral petechiae in our review.

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Definition

Asphyxia is broadly defined as the interference with the intake or utilization of oxygen, combined with the failure to eliminate carbon dioxide (6). Although all end organs are ultimately affected by asphyxia, the brain is most sensitive to it effects. The array of circumstances leading to asphyxial deaths varies widely. Together, they present some of the more vexing challenges in medicolegal death investigation and include such disparate mechanisms as cervical vascular obstruction, lower airway obstruction, smothering, chest compression, suffocation by asphyxiant gases, entrapment, and drowning.

Literature Review

Tardieu was the first to describe minute hemorrhages due to rupture of small blood vessels on the surfaces of the lungs, heart, and other body parts, and regarded them as "characteristic of death from suffocation" (12). The discussion within the forensic community that followed (10,16–21) became the forerunner of the controversy and confusion that remains today, including that surrounding the pathogenesis of the thoracic visceral petechiae sometimes observed in Sudden Infant Death Syndrome (SIDS). There does exist, however, wide agreement today that what are now known as "Tardieu spots" are the result of intense lividity, leading to postmortem rupture of dependently engorged blood vessels, entirely unrelated to asphyxia or any other mechanism of death. The occasional reference still made to them in the literature as antemortem petechiae or "asphyxial signs" betrays a misconception of their current meaning (6–7,10,21).

Luke was the first to propose that increased intracranial vascular pressure is the basis for the development of petechiae of the head in deaths involving compression of the neck or chest. In his 1967 review of strangulations in New York City, he reported that external petechiae, found in approximately 50% of the cases reviewed, were more prevalent on the conjunctivae than the face, slightly more prominent on the face than on the scalp, and seemed to be enhanced by the use of a ligature (14). He later theorized that the in-

creased prevalence of petechiae in ligature strangulations was due to the probability that "disproportionate venous/arterial compromise may be more effectively accomplished" with a ligature than with the hands alone (15). He explained that these petechiae occurred as a result of impairment of intracranial venous egress while arterial flow to the head continued, a "practically universal" phenomenon in ligature strangulations, partial suspension hangings, and thoracic compressions (1). Despite his seeming understanding, however, he went on to say that the pathogenesis of petechiae in these deaths had yet to be fully elucidated. In 1985, Luke and his colleagues elaborated on this theme in a retrospective study of hanging deaths (15). They concluded that the small vessel and intracapillary pressures in the head leading to the formation of petechiae of the conjunctivae and face should reflect the extent of carotid and vertebral artery occlusion, and that this, in turn, was dependent upon the amount of compressive ligature pressure produced by the degree of body suspension. The effects of the consistency and size of the ligature were not specifically addressed. Other authors have supported this contention, but have further suggested that the petechiae result from elevated venous pressure combined with hypoxic injury to endothelial cells caused by venous stasis and tissue acidosis (2,4,5,9,10,13,16,18,22).

Rao and Wetli were the first to apply the mechanical theory of the formation of conjunctival petechiae to nonasphyxial deaths in which there is increased cephalic venous pressure without neck or chest compression, per se (13). They included sudden cardiovascular deaths, particularly those with acute right heart failure, and instances in which individuals die with their faces prone as examples. They found, in fact, in their review of 5000 autopsy reports over nearly a two-year period, that conjuctival petechiae were observed most frequently in deaths due to natural causes. Their study contained no information or description of the number of petechiae observed in "positive" instances. When seen in asphyxial deaths, however, specifically, "homicidal asphyxiation" and mechanical chest compression (traumatic asphyxia), they agreed with other authors' contentions that the petechiae resulted from a combination of hypoxia and increased cephalic vascular pressure. This theory was further applied to nonasphyxial deaths in a study by Hood et al. that described the presence of conjunctival petechiae in individuals who had undergone attempted cardiopulmonary resuscitation prior to death (2). All decedents had petechiae of the conjunctivae, eyelids, and/or cheeks that were felt to be caused by perimortal resuscitative efforts, and unrelated to the mechanisms of death. Interestingly, the causes of death that were thought to be unrelated to the development of the petechiae in these decedents included atherosclerotic cardiovascular disease with an acute myocardial infarct, epilepsy, and gunshot wounds to the head, all known occasionally to be associated with conjunctival petechiae (5,13). The authors concluded that petechiae of the head occurred when repeated forceful resuscitative chest compressions caused increased pressure in small blood vessels that had been damaged as a result of hypoxia in a dying individual, leading to vascular rupture and blood extravasation into the surrounding tissues. They went on to say that hypoxia alone, without increased vascular pressure, was insufficient to produce such petechiae. They suggested that the combined amount of hypoxia and pressure needed to produce conjunctival petechiae was "not great," even in the living subject. They based this contention on a study in which healthy volunteers were placed in a head-down, vertical position as a means of determining the ocular manifestations of gravity inversion (23). After only one minute of inversion, the resultant ocular findings included orbital congestion, conjunctival hyperemia, and petechiae of the conjunctivae and upper eyelids. However, no subject lost consciousness during testing. Moreover, intraocular pressures were found to reach 80% of their maximal level within ten to fifteen seconds of inversion, and no statement was made suggesting that vascular hypoxic damage was contributory to the development of the petechiae. The forensic literature mentions some of these same physical findings in descriptions of fatalities due to positional asphyxia in victims of accidental, head-down "reverse suspension," but without specific discussions of the pathogenesis of cephalic congestion or petechiae of the head (24,25).

Other scenarios have been described as mechanical causes of conjunctival and facial petechiae in living patients and in victims of natural, nonasphyxial deaths, including status epilepticus, labor and delivery, and severe or sustained episodes of vomiting, coughing, sneezing, or respiratory stridor, as seen in bronchial asthma or croup (5,13,26,27). The underlying pathophysiologic mechanism suggested in these settings is the prolonged and/or forceful abdominal and thoracic muscular contractions resulting in reflux of blood from the right heart, which causes increased pressure in the valveless veins of the head and neck. The mechanical contribution of a concurrently closed glottis in association with thoracoabdominal compression (the Valsalva maneuver) in the production of increased cephalic venous pressures also is mentioned in the literature describing the facial plethora and petechiae accompanying prolonged chest compression in instances of traumatic asphyxia (26,28). The authors of these papers suggest that the glottis closes as part of a "pre-impact fear response" or panic.

The characteristic distribution of petechiae in cases of chest or neck compression has been addressed infrequently in the dermatology and forensic literature (7,26). In a case report describing the pathophysiologic features of traumatic asphyxia, Lowe et al. theorized that is was the lack of tissue support around conjunctival capillaries that accounted for the predictable subconjunctival hemorrhage, a nearly constant feature of traumatic asphyxia.

Asphyxial deaths in which facial and conjunctival petechiae are distinctly uncommon include those due to smothering (facial wedgings, those involving plastic bags or gags, and all forms of homicidal smothering), overlaying of children, choking, suffocating gases, entrapment, and drowning (5,6). Interestingly, however, at least one author has noted the occasional finding of very fine facial/conjunctival petechiae in deaths that involve the gagging or homicidal smothering of elderly individuals; an explanation for this observation, however, was not offered (5). Moreover, in those few deaths due to plastic bag suffocation in which conjunctival petechiae have been observed, neck ligatures usually were used to secure the bag in place; in those deaths without petechiae, no such ligatures were used (22,29). In accidental autoerotic deaths, the presence of petechiae correlates with the mechanism of asphyxia; specifically, in incomplete suspension hangings and ligature strangulations, petechiae more commonly are found, whereas, in deaths involving plastic bags or gags, they are not.

The presence or absence of petechiae is not specifically addressed in textbook discussions of deaths due to carotid sleeper and bar arm control/choke holds. Suggested mechanisms of these deaths include anoxia, particularly with the airway obstruction of bar arm holds, carotid sinus massage leading to an arrhythmia, a catechol surge due to "air hunger," and carotid and/or jugular vascular occlusion (5). Reay and Eisele found conjunctival petechiae, more prominently on the left side, in two cases of carotid sleeper hold deaths that occured during law enforcement activities (30). In each case, the hold was used for an unspecified "brief" period, during which time an intense struggle took place. An explanation for

the development of petechiae in this setting, however, was not offered.

Discussion

Excluding those related to infectious, coagulopathic, or microembolic etiologies, we conclude that petechiae of the head are the product of purely mechanical vascular phenomena: namely, impaired or obstructed venous return in the presence of continued arterial input. As pressure builds in venules and capillaries, particularly those with little surrounding connective tissue support, such as the conjunctivae and eyelids, vascular rupture produces petechiae. The likelihood of this occurrence is directly proportional to the degree of venous obstruction and inversely proportional to that of arterial compression at or above the level of the heart. Nearly 4.5 lb (2 kg) of pressure is required to compress the jugular veins, whereas 11 and 66 lb (5 and 30 kg) are required to compress the carotid and vertebral arteries, respectively (15,31); therefore, an intermediate amount of force simultaneously applied to both results in venous compression before arterial (2,6,15). This is similarly applicable to the right and left sides of the heart. If the compressive pressure to the chest or neck is great enough to obstruct venous return from the head, but not enough to obstruct arterial flow to it, cephalic venous pressure will rise, as will the probability of small vessel rupture. A violent struggle that increases cardiac output and raises blood pressure therefore enhances the occurrence of petechiae. Alternatively, if the applied force is sufficiently great to obstruct arterial flow, venous engorgement and rupture will not occur. An analogous mechanism resulting in elevated cephalic venous pressure without compression occurs with a precipitous impairment of venous return to the heart, such as that seen in acute right heart failure.

A clinical illustration of this vascular pressure phenomenon has been demonstrated elegantly with the capillary fragility test, or tourniquet test. Although now replaced by laboratory diagnostics, the tourniquet test was used years ago to assess vascular integrity by maintaining sphingomanometric pressure between that of systolic and diastolic for a duration of 5 min on an upper extremity. The number of petechiae observed within a 3 cm diameter distal to the tourniquet served as an indicator of mechanical vascular integrity; the presence of less than five petechiae was considered normal. Since arterial perfusion continued during testing, the petechiae were correctly interpreted as products of vascular rupture due to obstructed venous egress, rather than hypoxia (32).

Most authors agree that increased venous pressure is requisite in the development of conjunctival and facial petechiae, irrespective of whether the death is due to an asphyxial or natural, nonasphyxial cause. This conclusion is supported by the fact that petechiae characteristically and predictably are present in deaths that are preceded by impairment of venous egress from the head, while continuous or intermittent arterial perfusion to it continues. Examples of such deaths include those involving partial suspension hanging, ligature or manual strangulation, traumatic asphyxia/chest crushing injury, plastic bag-ligature suffocation, carotid sleeper holds with a concomitant struggle, acute right heart failure, status epilepticus, and those deaths preceded by a vigorous cardiopulmonary resuscitative effort, or prolonged or violent paroxysms of coughing or vomiting. Equally supportive of the vascular pressure mechanism is the fact that asphyxial deaths either with no impairment of venous return from the head or with arterial obstruction at or above the level of the heart characteristically lack facial and conjunctival petechiae (e.g., full suspension hanging, plastic bag suffocation, laryngotracheal obstruction, including bar arm holds and choking, asphyxiation by suffocating gases, smothering, overlay deaths in children, entrapment, and drowning).

Considering all of the foregoing observations, it is our contention that no relationship exists between the development of petechiae and the presence or absence of asphyxia. Rather, it is venous congestion without arterial obstruction that pathogenetically links the development of petechiae in these deaths, otherwise often disparate in their causes, mechanisms, and manners. Typically, the more intense the facial plethora, the more florid the petechial eruption. A possible explanation for the occasional observation of conjunctival petechiae in homicidal smotherings of elderly victims is the increased cephalic venous pressure of the Valsalva effect caused by struggling and screaming against an obstructed upper airway. This, together with increased cardiac output, elevated blood pressure, and the fragile vasculature and inelastic surrounding connective tissue of elderly persons, may lead to facial and conjunctival microvascular rupture. Conversely, the occasional absence of facial plethora and petechiae in victims of chest compression (traumatic asphyxia) is best explained by an overwhelming crushing force that effectively compresess the left ventricle and arrests further cardiac output, thereby precluding cephalic venous congestion.

Further support for a purely mechanical basis for the development of petechiae lies in the fact that, when observed on the head, they are more likely to be found on the conjunctivae and eyelids (7,9,14,17,22,26,28). Luke found, in fact, in his 1985 review of hanging deaths, that although conjunctival petechiae were commonly observed as isolated findings, petechiae of the facial skin were never seen without those of the conjunctivae (15). This distribution is best explained by the relative lack of support and resistance offered by the surrounding tissues in and around the conjunctival and palpebral capillaries. As pressure mounts in the microvasculature, the likelihood of rupture is inversely correlated with the tenacity of the surrounding connective tissue and its ability to prevent and/or tamponade blood extravasation. Surely, the capillary endothelium of the conjunctivae and eyelids is no more susceptible to the effects of hypoxia and tissue acidosis than is the capillary endothelium of the thick skin of the face. Yet, petechiae are distinctly more prevalent in the conjunctivae and eyelids.

The literature suggests that it is the combined effects of increased vascular pressure and hypoxic microvasculature that lead to petechiae of the head. Although this is difficult to disprove, the addition of hypoxia to the pathogenetic equation is unnecessary and misleading. Without invoking venous stasis and hypoxia-related increased vascular permeability, the pathogenesis of conjunctival and facial petechiae is sufficiently explained by vascular pressure increases and microanatomy alone; all available data support this mechanism. Moreover, conjunctival petechiae have been observed in surviving victims of attempted strangulation, suggesting cephalic venous congestion, rather than life-threatening asphyxia, as the mechanism of microvascular rupture. A study examining both living and dead victims of strangulation reported conjunctival petechiae in 14 out of 79 surviving victims, only half of whom had suffered sufficient oxygen deprivation to lose consciousness during the assault (33). Some of these surviving victims actually had a more florid and pronounced petechial eruption than that of their deceased counterparts. Despite these data, the speculation that hypoxia must play a role has generated the erroneous conclusion that petechiae and asphyxia are causally related. The perpetuation of this belief betrays an attachment to dogma that has no basis in science or logic. Some authors have actually suggested an instanta-

neous neural mechanism of death (as opposed to complete arterial obstruction) in pale-faced victims of full suspension hangings, as a means of explaining the absence of the expected "asphyxia-induced" petechiae in those cases (8).

We conclude that hypoxia is unrelated to the pathogenesis of petechiae in deaths due to compression of the neck or chest. Facial and conjunctival petechiae develop in a variety of circumstances in which cephalic plethora, not asphyxia, is the final common pathway. We know of no evidence whatever to support the contention that hypoxia of a duration up to a few minutes, i.e., sufficient to be lethal, produces capillary fragility or any hemorrhage-inducing alteration of the physical properties of capillary endothelium in the conjunctivae or any other location. In fact, compelling evidence exists to the contrary. The presence of conjunctival petechiae in living victims of attempted strangulation and experimentally inverted subjects supports our contention that pressure phenomena, not asphyxia or hypoxia, leads to their development. Moreover, hundreds, if not thousands, of persons who have accomplished suicide by placing a plastic bag over their heads, without a concomitant drug overdose, have performed "human experiments" by inducing pure hypoxial deaths. Unless the bag is fastened around the neck by a ligature with sufficient tension to obstruct venous return from the face, in our experience, such persons never have facial or conjunctival petechiae. Persons with tight fastenings around the neck are recognizable instantly by their facial plethora and numerous petechiae. We are aware that some observers may have seen an occasional petechia in rare instances of plastic bag suicide (22). However, we have not seen detailed descriptions of such observations that permit another person to evaluate the variables that might have produced an isolated petechia in a rare victim. Conversely, in New York City, an average of approximately 15 persons per year accomplish suicide by plastic bag, and we never have observed petechiae in a person who did not tightly fasten the bag around his/her neck (19).

The importance of defining the pathogenesis of conjunctival and facial petechiae lies in the potential implications of their misinterpretation. They are simply markers of increased cephalic venous pressure. In and of themselves, they should not be regarded as supportive evidence of asphyxia; in a vacuum, conjunctival and facial petechiae point to no particular cause of death. Only in conjunction with a complete autopsy and thorough death investigation can their potential importance be ascertained, and only with an understanding of their pathogenesis can they lend insight into the circumstances of a death. An erroneous interpretation of this clearly mechanical phenomenon, which can occur in a heterogenous array of perimortal circumstances, creates a slippery slope. In the best-case scenario, confusion is perpetuated; in the worst-case scenario, the adjudication of criminal cases is severely impaired, and fertile soil having the potential to create false grounds for conviction or acquittal is laid.

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