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Comparative Forensic Morphology of Pulmonary Changes in Fatal Acute Ethanol Intoxication and Carbon Monoxide (Domestic Gas) Poisoning: A Literature Review

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Abstract

Background. Acute fatal ethanol intoxication and domestic-gas (carbon monoxide, CO) poisoning are common causes of toxicological death that regularly confront the forensic pathologist. In both, the lung is a principal target organ, yet the pulmonary changes are largely non-specific, so that morphology alone rarely establishes the cause of death. Understanding the pathogenesis, histological pattern and confirmatory laboratory correlates of each is therefore essential for correct medico-legal interpretation, particularly because the two exposures frequently coexist.

Objective. To review and compare the macroscopic and microscopic pulmonary changes, underlying mechanisms, quantitative autopsy findings and confirmatory tests of fatal acute ethanol intoxication and CO/domestic-gas poisoning, and to define a practical framework for their differential forensic diagnosis.

Methods. A narrative review was performed in PubMed, Scopus and Web of Science, prioritising articles from 2015–2026 and complemented by key earlier and textbook sources. Fifty-two human, experimental and forensic references were analysed and organised by aetiology, level of biological organisation and diagnostic value.

Results. Acute ethanol intoxication produces respiratory-centre depression, microcirculatory disturbance, venous–capillary congestion, red-cell stasis, interstitial and

alveolar oedema, alveolo-capillary barrier injury with plasmorrhagia and fibrin, and an aspiration risk from diminished protective reflexes; chronic exposure adds alveolar glutathione depletion (80–90%), surfactant and macrophage dysfunction, and pro-fibrotic remodelling. CO poisoning acts chiefly through carboxyhaemoglobin (COHb) formation and tissue hypoxia with oxidative stress, producing swollen, congested, oedematous, often cherry-red lungs with subpleural petechiae. Pulmonary oedema and congestion are the shared, non-specific endpoints; combined ethanol + CO exposure yields a mosaic microcirculatory picture with mutual aggravation. Confirmation requires blood/vitreous ethanol and blood COHb (cherry-red livor typically >30%, death usually >50–60%).

Conclusion. Pulmonary morphology in these poisonings is corroborative rather than diagnostic. A defensible medico-legal conclusion integrates scene investigation, autopsy, histology and—decisively—toxicology (ethanol concentration and COHb). Awareness of the combined-exposure scenario and of analytical pitfalls is critical to avoid misclassification.

Keywords: Forensic pathology; acute ethanol intoxication; carbon monoxide poisoning; domestic gas; carboxyhaemoglobin; pulmonary oedema; alveolo-capillary barrier; differential diagnosis.

1. Introduction

Poisoning deaths are a recurrent and demanding category of forensic practice, and among them acute ethanol intoxication and domestic-gas poisoning are among the most frequently encountered. Carbon monoxide (CO)- the toxic product of incomplete combustion of household fuels- remains one of the leading causes of fatal poisoning worldwide, accounting in some countries for over half of all poisoning deaths and for thousands of fatalities each year [25,36,37]. Acute alcohol poisoning, in turn, is a major contributor to toxicological mortality and frequently coexists with other exposures, complicating the interpretation of cause and manner of death [20]. In both conditions the lung is a principal target organ, and its examination forms a routine part of the medico-legal autopsy.

A central difficulty is that the pulmonary changes produced by these poisonings are largely non-specific. Pulmonary oedema, vascular congestion, subpleural petechiae and interstitial changes can arise from many causes, so that histology alone cannot reliably distinguish one aetiology from another, nor confirm a poisoning as the cause of death [21,34,38]. The forensic value of lung morphology therefore lies not in pathognomonic features but in a consistent pattern that, when integrated with scene findings and- decisively0 toxicological analysis, supports a defensible conclusion. This integrative requirement is heightened by the frequent coexistence of ethanol and CO: alcohol impairs consciousness and escape from a gas-filled environment, and may also confound some analytical methods [24].

1.1. Normal pulmonary structure and the alveolo-capillary barrier

The lung's gas-exchange function depends on the integrity of the alveolo-capillary (aerohaematic) barrier, a thin membrane comprising the alveolar epithelium (flat type I and surfactant-producing type II pneumocytes), a fused basement membrane, and the capillary endothelium, together with the surfactant film and resident alveolar macrophages [3,4]. This barrier maintains alveolar dryness and permits efficient oxygen and carbon-dioxide transfer. Injury to any component- through hypoxia, oxidative stress, increased vascular permeability, or direct cytotoxicity-0 produces the final common pathway of increased permeability, interstitial and alveolar oedema, and impaired gas exchange that underlies acute lung injury in toxicological death [42,43]. Notably, the lung is uniquely exposed to ethanol because a portion of ingested ethanol and its metabolites is excreted unchanged through respiration, sustaining local tissue exposure [1,3].

1.2. Aim of the review

This review synthesises current evidence on the pulmonary morphology of fatal acute ethanol intoxication and of domestic-gas/CO poisoning. It compares their pathogenesis, macroscopic and microscopic features, and quantitative autopsy findings; addresses the combined-exposure scenario; and sets out a practical framework- anchored in confirmatory toxicology- for their differential forensic diagnosis. The emphasis throughout is medico-legal: which findings are suggestive, which are non-specific, and what is required to reach a sound conclusion on the cause of death.

2. Methods

This is a narrative (non-systematic) review. PubMed/MEDLINE, Scopus and Web of Science were searched with combinations of the terms “acute ethanol/alcohol intoxication”, “alcoholic lung injury”, “carbon monoxide poisoning”, “domestic/household gas poisoning”, “carboxyhaemoglobin”, “forensic autopsy”, “pulmonary oedema”, “alveolo-capillary barrier”, “glutathione”, “surfactant” and “differential diagnosis”. Priority was given to peer-reviewed articles from 2015–2026, supplemented by seminal earlier papers and standard forensic-pathology texts. Human autopsy series, experimental (rodent) models and mechanistic and clinical

reviews were included. Data were organised by aetiology (ethanol vs CO), by level of biological organisation (organ, tissue, cell, molecule) and by diagnostic value, and are presented descriptively because heterogeneity of case material, exposure and post-mortem interval precluded quantitative pooling. Fifty-two sources were retained. Quantitative values used in Figures 1–6 and Table 3 were taken from the cited studies; where a value is a representative or midpoint estimate this is stated in the figure legend. The two uploaded source documents were checked against, and integrated with, the independently retrieved literature.

2.1. Overview of the included evidence

Evidence stream	Typical setting / model	Principal read-outs	Representative sources
Forensic autopsy series	Human fatal poisonings	Macro/micro lung findings, COHb, ethanol	Çevik 2026; Liu 2021; Wang 2019 [21,34,20]
Mechanistic reviews	Human + experimental	Barrier, oxidative stress, hypoxia	Rose 2017; Joshi 2007; Mehta 2017 [25,3,4]
Experimental models	Rat lung, ethanol/gas	Histomorphology, microvasculature	Elu 2014; Enikeev 2021; Romanova 2020/23 [23,24,18,19]
Alcohol–lung biology	AT2 cells, macrophages	Surfactant, glutathione, immunity	Yeligar 2016; Simet 2015; Burnham 2003 [5,9,7]
CO toxicology / diagnosis	Clinical + forensic	COHb thresholds, hypoxia mechanism	Weaver 2009; Hampson 2018; Ernst 1998 [26,32,27]
Combined exposure	Rat CO + ethanol	Mosaic dyscirculation, aggravation	Enikeev 2021 [24]

Table 1. Streams of evidence integrated in this comparative review. AT2, alveolar type II epithelial cell; COHb, carboxyhaemoglobin.

3. Pulmonary Morphology by Aetiology

Table 2 summarises the dominant mechanism and the macroscopic and microscopic pulmonary features of each aetiological group, which are then examined in detail.

Aetiology	Leading mechanism	Macroscopic lung	Microscopic lung
Acute ethanol intoxication	Respiratory-centre depression, microcirculatory disturbance, aspiration, hypoxia	Heavy, wet, congested lungs; frothy/serous fluid on section; basal petechiae possible	Septal capillary dilatation and congestion, interstitial/alveolar oedema, red-cell stasis and sludge, fibrin, atelectasis/dysatelectasis
Chronic alcohol effect	Glutathione depletion, surfactant & macrophage dysfunction, oxidative injury	Often unremarkable grossly; features of prior injury/pneumonia may coexist	Alveolar epithelial/endothelial metabolic lesions, plasmorrhagia, protein accumulation, pneumosclerosis, septal collagen

Aetiology	Leading mechanism	Macroscopic lung	Microscopic lung
CO / domestic-gas poisoning	COHb formation → tissue hypoxia + oxidative stress	Enlarged, heavy, swollen, congested, oedematous lungs; cherry-red blood/viscera; subpleural petechiae	Capillary/venular congestion, interstitial & alveolar oedema, hypoxic endothelial injury, focal alveolar haemorrhage

Table 2. Dominant mechanism and pulmonary morphology by aetiology. Synthesised from [1,2,3,5,21,23,24,25,34]. COHb, carboxyhaemoglobin.

3.1. Acute ethanol intoxication: pulmonary changes

3.1.1. Pathogenesis

In acute fatal ethanol intoxication the pulmonary changes are driven chiefly by depression of the central nervous system and the respiratory centre, an acute disturbance of the pulmonary microcirculation, hypoxia, and an increase in vascular-wall permeability [1,2]. Ethanol depresses the respiratory drive, blunts protective airway reflexes and predisposes to aspiration of gastric contents, promoting hypoventilation and hypoxaemia [4,8]. Because the lung excretes a fraction of ethanol and its metabolites directly, alveolar tissue sustains local exposure that increases capillary permeability and allows protein-rich fluid to accumulate [1,3]. The net effect is a non-specific acute lung injury developing in a previously unremarkable lung.

3.1.2. Macroscopic findings

At autopsy the lungs are commonly heavy, congested, wet and oedematous, and the cut surface may exude frothy or serous fluid- features of generalised oedema and venous congestion that are not specific to ethanol [2,20]. Small subpleural (basal) petechial haemorrhages may be present, particularly where an asphyxial component (aspiration, positional airway compromise) is superimposed; documented forensic cases describe congested cut surfaces with basal haemorrhagic spots and alveolar spaces filled with pale-red oedematous fluid [20]. These signs must be weighed together with blood ethanol concentration, scene circumstances and changes in other organs.

3.1.3. Microscopic findings

Microscopically, the alveolar septa are thickened owing to dilatation and congestion of septal capillaries, endothelial swelling and interstitial oedema (Figure 1). Alveolar spaces may contain serous exudative fluid, extravasated erythrocytes, macrophages and fibrin strands [2,20]. Injury to alveolocytes with increased alveolo-capillary permeability and fibrin deposition has been described as a non-specific acute injury of a previously healthy lung. Ventilation–perfusion disturbance produces areas of atelectasis and dysatelectasis, and reported studies note markedly narrowed or barely discernible alveolar spaces in affected regions; erythrocyte stasis and the sludge phenomenon reflect disturbed pulmonary blood rheology and impaired gas exchange [2].

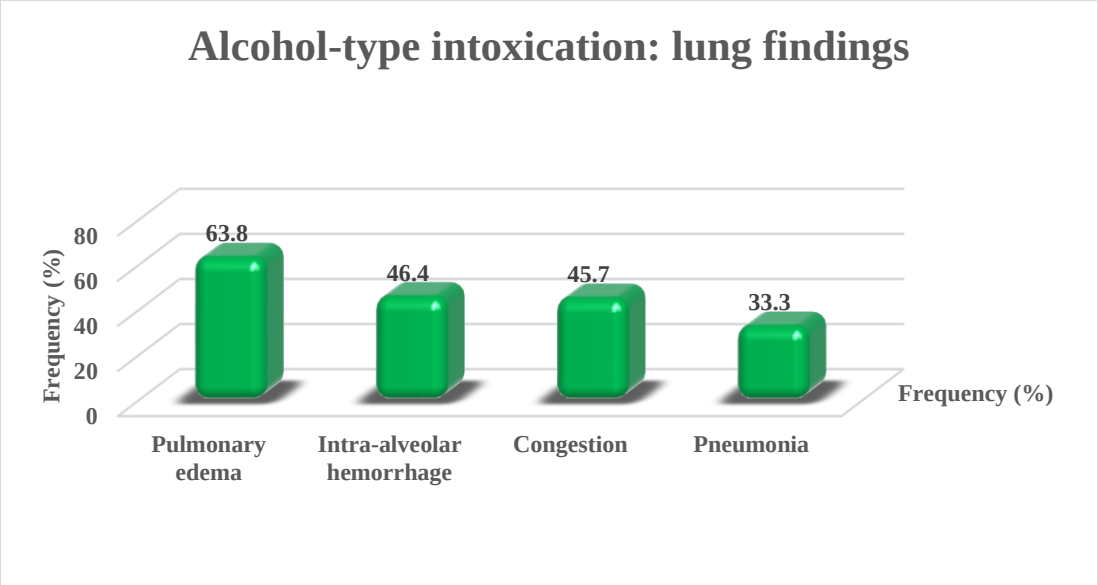

Figure 1. Pulmonary autopsy findings in fatal alcohol-type (methanol) intoxication (n=138, %)

3.2. Chronic alcohol effects on the lung

Beyond the acute event, chronic alcohol exposure primes the lung for injury- the concept of the "alcoholic lung." A cardinal mechanism is depletion of the alveolar antioxidant glutathione, reported at roughly 80–90% of normal in the alveolar space, which renders the epithelium susceptible to oxidant-mediated injury and impairs surfactant production [3]. Chronic ethanol disrupts surfactant phospholipid formation in alveolar type II cells and reduces dipalmitoylphosphatidylcholine, and it depresses the phagocytic activity and motility of alveolar macrophages, impairing pulmonary defence [1,5,13]. Toxic effects of ethanol and its metabolites increase vascular permeability with protein accumulation, dystrophic and destructive parenchymal changes, and subsequent pneumosclerosis [1,2]. At the barrier, ethanol inhibits alveolar fluid clearance through suppression of the epithelial sodium channel, favouring oedema persistence [14]. With chronic exposure, fibrotic remodelling develops, with reported increases of the order of 75% in collagen deposition and 120% in TGF-β1 after lung injury (Figure 2) [12]. Clinically and experimentally, these changes translate into a markedly increased susceptibility to acute respiratory distress syndrome and pneumonia [6,9,10,11].

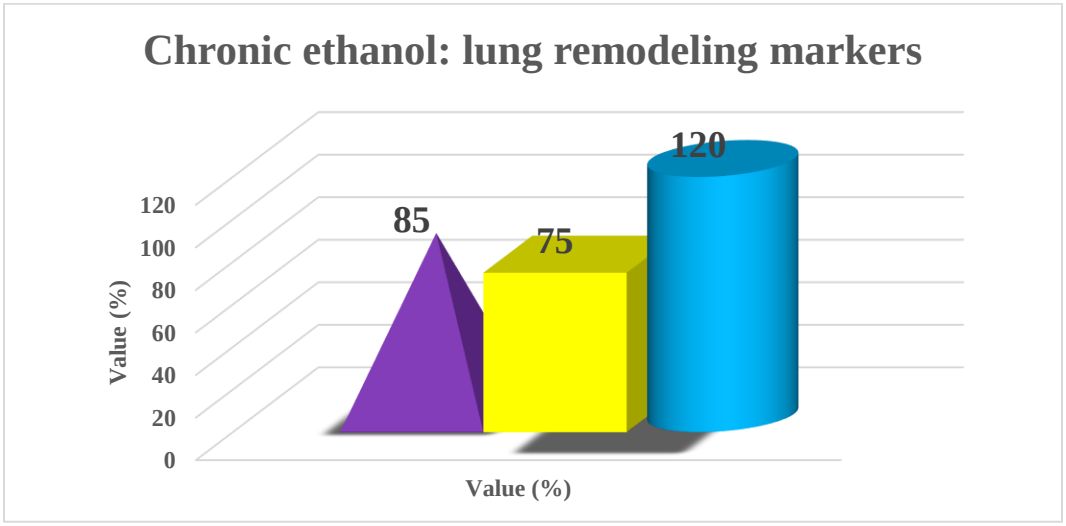

Figure 2. Chronic ethanol lung remodeling / oxidative markers (relative change)

3.3. Domestic-gas / carbon monoxide poisoning: pulmonary changes

3.3.1. Nature of the exposure and mechanism

Domestic-gas poisoning most often results from faulty heating appliances, gas stoves, water heaters, blocked flues or incomplete fuel combustion, generating carbon monoxide- a colourless, odourless gas that binds haemoglobin with an affinity roughly 200–250 times that of oxygen to form carboxyhaemoglobin (COHb) [25,27,37]. This impairs oxygen carriage and offloading and shifts the oxyhaemoglobin dissociation curve, producing tissue hypoxia. Importantly, CO toxicity extends beyond haemoglobin blockade: it binds other haem proteins, promotes oxidative stress, mitochondrial dysfunction and inflammatory-mediator activation, so that injury appears simultaneously in the lung, brain, myocardium and other high-oxygen-demand organs [25,29,52]. A crucial caveat is that domestic-gas poisoning is not synonymous with CO alone: natural gas (methane) can kill by displacing oxygen and causing simple asphyxia, whereas incomplete combustion generates CO; the toxic agent must therefore be defined by toxicology in each case.

3.3.2. Macroscopic findings

The classical external sign is a cherry-pink or bright-red discoloration of livor mortis, blood and viscera, typically evident when blood COHb exceeds approximately 30%, though it is inconsistent and cannot alone establish the diagnosis and is hard to appreciate in anaemic or deeply pigmented individuals [38,39]. The lungs are usually enlarged, heavy, swollen, congested and oedematous, sometimes light-red, with abundant foamy bloody fluid on section and subpleural petechiae [21,25]. Reported autopsy series list pulmonary oedema as the most frequent finding, with emphysematous change, haemorrhage and congestion at lower frequencies (Figure 3); one series documented pulmonary oedema in essentially all cases with deep-red discoloration in about 80% [34, and forensic series].

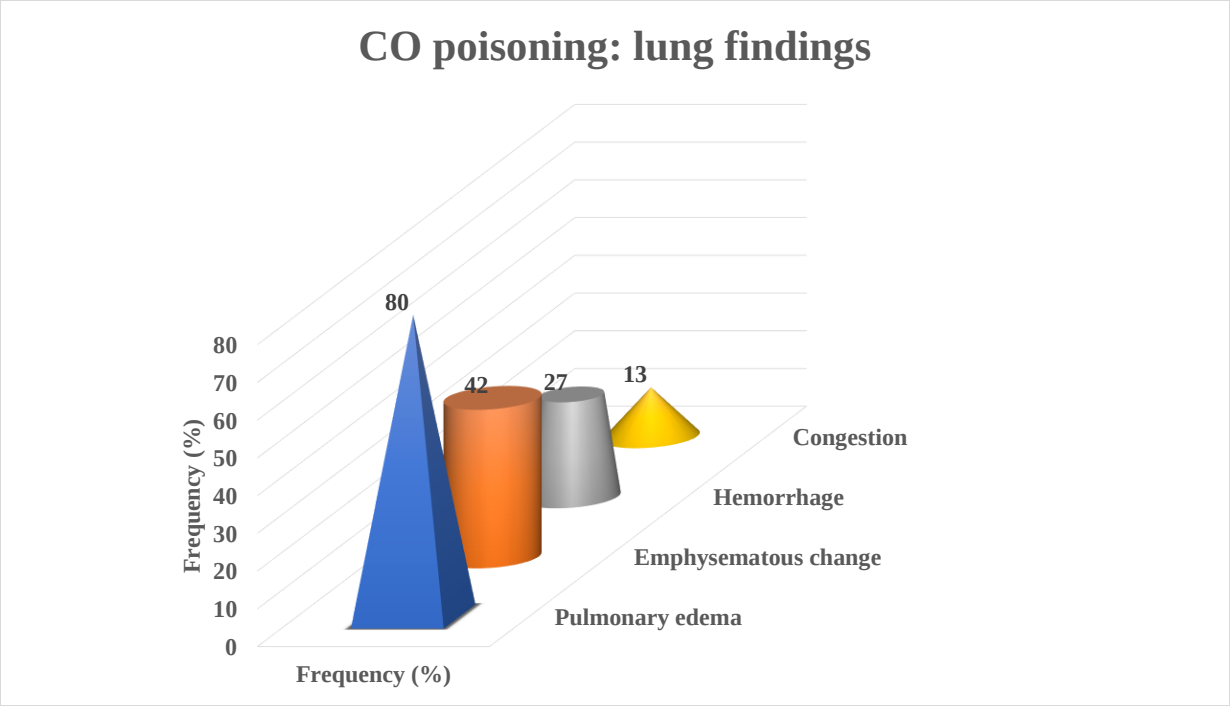

Figure 3. Pulmonary autopsy findings in fatal carbon monoxide poisoning (%)

3.3.3. Microscopic findings

Histology shows congestion of alveolar capillaries and venules, interstitial and alveolar oedema, intra-alveolar serous fluid and, in places, extravasation of erythrocytes. Increased vascular permeability from hypoxic endothelial injury and disruption of the alveolo-capillary barrier is the principal morphological basis of the oedema [25]. These features are the expression of prolonged tissue hypoxia and advanced circulatory failure and, like the macroscopic signs, are non-specific; the diagnosis rests on measured COHb, not on histology [34,38]. Experimental models of acute fatal household-gas poisoning demonstrate ultrastructural damage to bronchial and respiratory epithelium, with mitochondrial and endoplasmic-reticulum injury and destruction of the aerohaematic barrier, helping to explain the mechanism of death [23].

3.4. Quantitative autopsy findings

Table 3 collates representative quantitative findings. Frequencies vary with the study population, duration of exposure, resuscitation and post-mortem interval, and should be read as indicative of pattern and magnitude rather than as fixed values; the corresponding data are plotted in Figures 1–6 of the accompanying editable workbook.

Finding / parameter	Setting	Value	Source
Pulmonary oedema (CO fatalities)	Autopsy series	up to ~80–100% of cases	[34; forensic series]
Emphysematous change / haemorrhage / congestion (CO)	Autopsy series	≈42% / 27% / 13%	[forensic series]
Cherry-red discoloration (CO)	Autopsy series	≈80% (COHb-dependent)	[38]
Fatal CO cases by COHb band (n=307)	Forensic cohort	10–30%: 58; 30–50%: 79; ≥50%: 170	[34]
Pulmonary oedema (alcohol-type intoxication)	Autopsy series (n=138)	63.8%	[21]
Intra-alveolar haemorrhage / congestion (alcohol-type)	Autopsy series (n=138)	46.4% / 45.7%	[21]
Alveolar glutathione depletion (chronic ethanol)	Experimental/clinical	80–90%	[3]
Collagen deposition / TGF-β1 (chronic ethanol)	After lung injury	≈ +75% / +120%	[12]
Mixed toxicological series: oedema / haemorrhage / infiltration	Autopsy series	55.8% / 48.1% / 38.5%	[reviewed literature]
Cherry-red livor threshold / commonly fatal COHb	Forensic	>30% / >50–60%	[27,32,38]

Table 3. Representative quantitative pulmonary and toxicological findings. Values are indicative and drawn from the cited studies; some are midpoint or representative estimates (see figure legends). COHb, carboxyhaemoglobin.

3.5. Combined ethanol and carbon-monoxide injury

Ethanol and CO frequently coexist, and their combination is more than additive. When acute alcohol intoxication and CO poisoning co-occur, the lung shows a mosaic microcirculatory disorder in which areas of pronounced arterial and venous hyperaemia alternate with zones of anaemia and vessel desolation; carbon-monoxide pathology tends to predominate, but both dyscirculatory disturbance and parenchymal pathomorphology are significantly more pronounced than with either insult alone- a mutual aggravation producing more severe tissue damage [24]. Mechanistically the two converge on shared endpoints of barrier breakdown, oedema and impaired gas exchange: alcohol contributes metabolic and immune disruption, aspiration risk and reduced capacity to escape a gas-filled space, while CO drives hypoxic–oxidative injury [1,23,24]. This interaction has direct medico-legal relevance: ethanol may lower the COHb level required for death by impairing escape and increasing susceptibility, and its presence must be quantified and interpreted alongside COHb rather than treated as incidental [24].

3.6. Comparative morphological summary

Criterion	Acute ethanol intoxication	Domestic gas / CO poisoning
Leading mechanism	Respiratory-centre depression, microcirculatory disturbance, aspiration and hypoxia	COHb formation, tissue hypoxia, oxidative stress
Gross lung	Heavy, wet, congested; frothy/serous fluid on section	Enlarged, swollen, congested; cherry-red blood and viscera may be seen
Vascular changes	Venous–capillary congestion, red-cell stasis, sludge phenomenon	Congestion, increased capillary permeability, hypoxic endothelial injury
Alveolar changes	Septal thickening, interstitial oedema, atelectasis/dysatelectasis, fibrin	Alveolar and interstitial oedema, subpleural petechiae, focal alveolar haemorrhage
Confirmatory test	Ethanol in blood / vitreous / urine	Carboxyhaemoglobin (COHb) in blood
Forensic conclusion	Histology auxiliary; ethanol level and circumstances decisive	Histology auxiliary; COHb and scene circumstances decisive

Table 4. Comparative forensic morphology of pulmonary changes in fatal ethanol intoxication and CO/domestic-gas poisoning. Synthesised and expanded from [1,2,20,24,25,34,38].

3.7. Confirmatory laboratory diagnosis and forensic pitfalls

Because the pulmonary changes are non-specific, the diagnosis in both conditions is established by toxicology, not by morphology. For suspected alcohol death, ethanol is quantified in blood (preferably femoral venous), and, to reduce error from post-mortem redistribution or neo-formation, in vitreous humour and urine [20,40]. For suspected CO death, blood COHb is measured; a cherry-red livor is typically associated with COHb above about 30%, and death usually corresponds to levels above 50–60%, although fatalities at lower COHb occur, especially with fire, cardiorespiratory disease or combined exposures (Figure 4, Figure 5) [27,32,34,38]. Table 5 lists indicative COHb bands. Several pitfalls warrant emphasis: (i) baseline COHb is 1–3% in non-smokers and up to ~10–15% in heavy smokers, which must be accounted for [32]; (ii) putrefaction and heating can alter COHb interpretation, and low-COHb fatalities may still be CO-related [34]; (iii) some breath analysers may misread in the presence of ethanol, risking a false-positive CO interpretation; and (iv) natural gas (methane) causes death by oxygen displacement without generating COHb, so a negative COHb does not exclude domestic-gas asphyxia. A sound conclusion therefore integrates scene investigation, full autopsy, histology and quantitative toxicology.

Blood COHb (%)	Typical context	Forensic significance
1–3 (up to ~10–15 in smokers)	Physiological / smoking baseline	Not indicative of poisoning; interpret against smoking status
~20–30	Symptomatic exposure	Consistent with significant exposure; assess with circumstances
≥30	Marked exposure	Cherry-red livor/blood often appears; strongly supportive
≥50–60	Lethal range	Commonly fatal; strong support for CO as cause of death

Table 5. Indicative carboxyhaemoglobin (COHb) bands and their forensic significance. Thresholds are approximate and context-dependent [27,32,34,38].

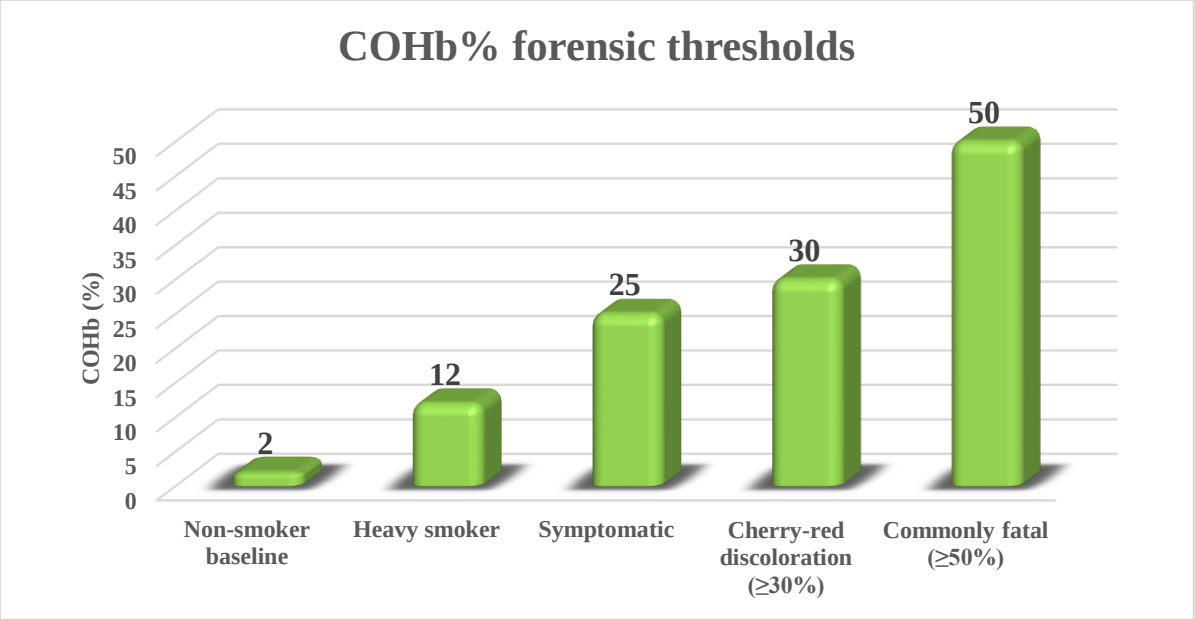

Figure 4. Carboxyhemoglobin (COHb) levels of forensic significance

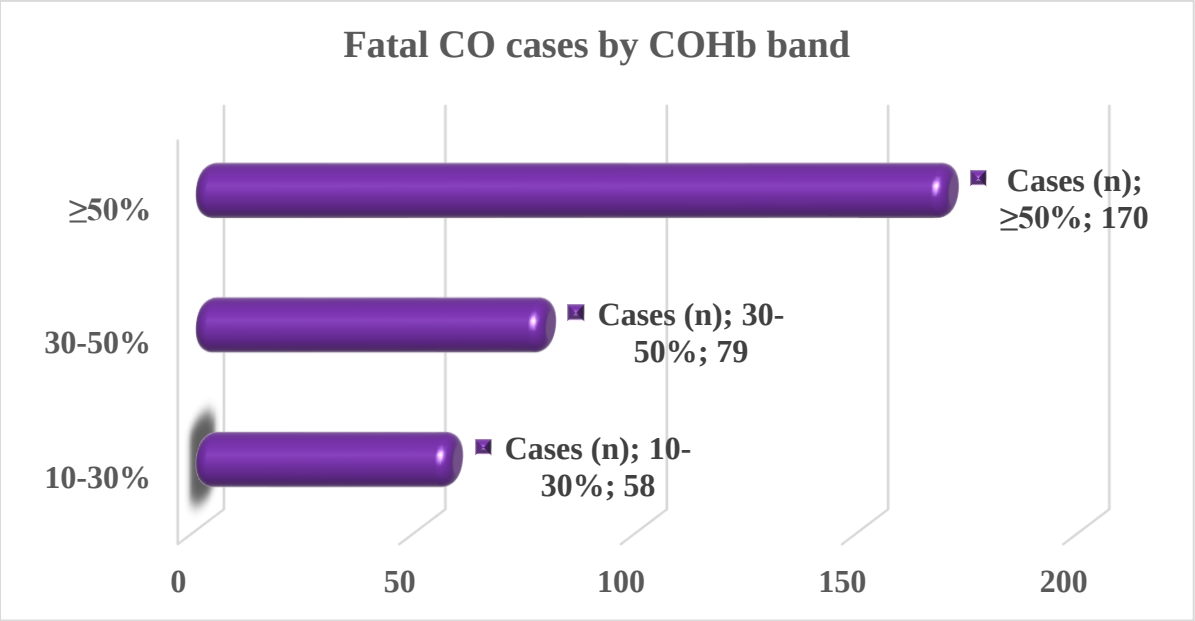

Figure 5. Distribution of fatal CO cases by COHb band (Shanghai forensic cohort, n=307)

4. Discussion

The evidence reviewed here yields a consistent message: in both fatal ethanol intoxication and CO/domestic-gas poisoning the lung reacts through a shared, largely non-specific repertoire- congestion, increased permeability, interstitial and alveolar oedema, focal haemorrhage- so that morphology corroborates but does not by itself establish the cause of death [1,2,21,25,34,38]. What distinguishes the two is mechanism and confirmatory correlate rather than a unique histological signature (Figure 6).

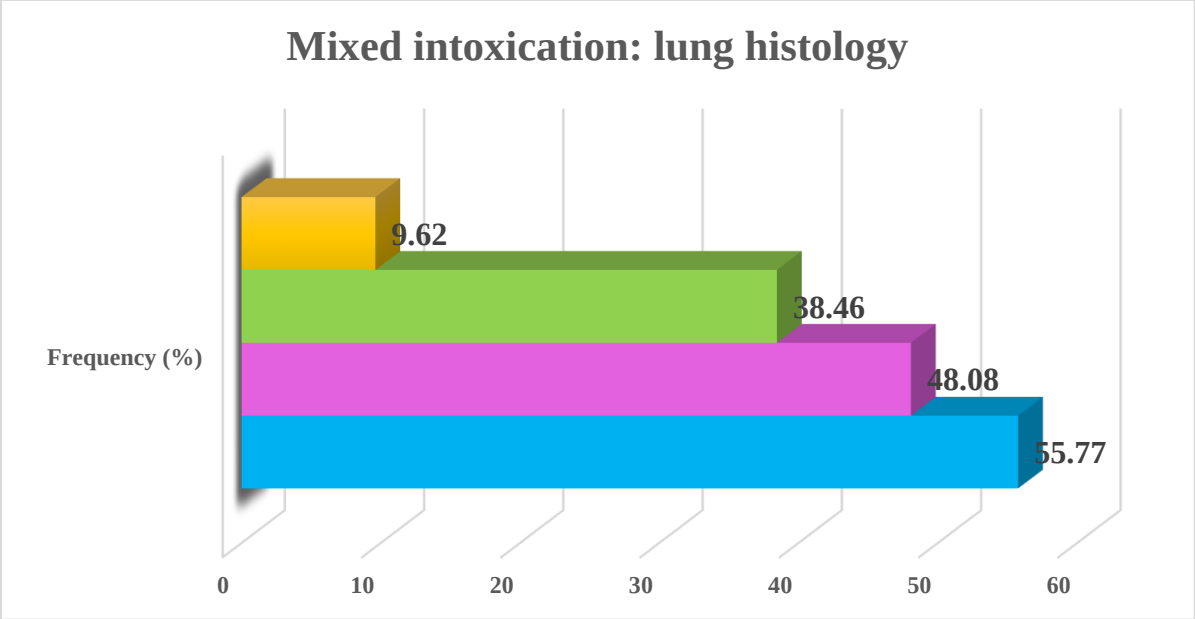

Figure 6. Lung histology frequencies in a mixed toxicological autopsy series (%)

4.1. Distinct mechanisms, convergent morphology

Ethanol acts predominantly through central respiratory depression, microcirculatory disturbance, aspiration and a permeability-increasing local toxic effect amplified by pulmonary excretion of ethanol, with chronic exposure adding glutathione depletion, surfactant and macrophage dysfunction and pro-fibrotic remodelling [1,3,5,12,14]. CO acts through COHb-mediated hypoxia compounded by oxidative and mitochondrial injury [25,29,52]. These distinct upstream mechanisms converge on the same downstream endpoint- alveolo-capillary barrier failure with oedema—which is why the histological pictures overlap so extensively and why a pathologist cannot separate them on morphology alone. This convergence is the single most important interpretive principle for practice.

4.2. The primacy of toxicology and scene context

Because morphology is shared, the discriminating information is toxicological and circumstantial. Blood, vitreous and urine ethanol quantify alcohol exposure while mitigating post-mortem artefacts, and blood COHb quantifies CO exposure with reasonably well-established lethal ranges [20,27,32,34,40]. Scene investigation- gas source, ventilation, heating appliance, presence of fire or soot, and containers- often provides the decisive context [38,39]. The forensic conclusion is thus an act of integration: pattern on autopsy and histology, confirmed and made specific by toxicology, and made coherent by the scene.

4.3. The combined-exposure problem

The frequent coexistence of ethanol and CO is more than a statistical curiosity. Experimentally, the combination produces a mosaic microcirculatory disorder and mutually aggravated parenchymal damage exceeding either exposure alone [24]. Clinically and medico-legally, ethanol reduces awareness and the capacity to escape a CO-laden environment and may render lower COHb levels lethal, while also posing analytical confounding in some CO measurement methods [24,34]. Cases must therefore be worked up for both agents, and a positive finding for one should not close the investigation into the other.

4.4. Limitations and future directions

Much of the morphological literature is descriptive, based on heterogeneous case series and experimental models with differing exposure and post-mortem intervals, and quantitative pulmonary end points are inconsistently reported, precluding meta-analysis. Post-mortem change further limits both morphological and biochemical interpretation. Promising directions include molecular-pathology markers of alveolar barrier injury (for example matrix metalloproteinases, claudin-5, aquaporins and adhesion molecules) that may help characterise oedema mechanisms in otherwise non-specific cases, standardised quantitative histomorphometry, and harmonised protocols coupling scene data, autopsy, histology and toxicology. This review is itself narrative and subject to selection and language bias and did not perform quantitative synthesis; nonetheless, the convergence of independent human, experimental and forensic sources supports its central conclusions.

5. Conclusion

In fatal acute ethanol intoxication and in domestic-gas/CO poisoning the lung shows overlapping, non-specific changes- congestion, increased permeability, interstitial and alveolar oedema and focal haemorrhage- that reflect distinct mechanisms (central respiratory depression, microcirculatory disturbance and local toxic injury in ethanol; COHb-mediated hypoxia with oxidative injury in CO) converging on alveolo-capillary barrier failure. Pulmonary morphology is corroborative, not diagnostic. A defensible medico-legal conclusion requires integration of scene investigation, full autopsy and histology with decisive toxicology- blood, vitreous and urine ethanol, and blood carboxyhaemoglobin- and explicit consideration of the combined-exposure scenario and of analytical pitfalls. Approached in this integrated way, the pulmonary findings become a valuable, if supporting, component of accurate cause-of-death determination.

Declarations

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