### **Review Article**

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### Threshing grain of mechanism from chaffy myth of air embolism in laparoscopy - lux et veritas

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### **ABSTRACT**

Biomechanical processes involving exact mechanism of air embolism (AE) or gas embolism (GE) in pneumoperitoneum (PP) in operative laparoscopy (OL) causing morbidity and mortality lie in air lock (AL) or gas lock (GL) in right side of heart resulting in traffic jam (TJ) for blood flow in pulmonary tree (PT). This also leads to poorly understood cascade of immunopathological pulmonary reactions. These issues are critically reviewed and discussed to thresh the grain of mechanism from husk of myth of ghost of AE or GE in OL for its dreaded moribund complications. Physics of gas is defined with analysis that venous GE is not exclusive to ambient air (AA) in OL but occurs with CO<sub>2</sub>, N<sub>2</sub>O, O<sub>2</sub> and other gases also. It is important since use of AA in OL is useful or ostensibly better than CO2 in situations like pregnancy, camps, cardiopulmonary cripples, cirrhotics, war fields, etc and economic needing no cylinders as AA is ubiquitous. Even when gas used was or is CO2 or O2 but blemish is labelled on air for embolism imprudently needing scientific scrutiny.

Keywords: Air lock, Air or gas embolism, Ambient air, CO<sub>2</sub>, Operative laparoscopy, Pneumoperitoneum

### INTRODUCTION

Venous air embolism (AE) is a fairly common event but death or moribund events are rare. Natal and Doty reported that many cases of venous AE are subclinical with no adverse outcome and thus go unreported.1 It carries very high mortality and morbidity in severe cases irrespective of gas and begs a high index of awareness. This analytic review tries to thresh the grain of mechanism from chaffy myth of AE or GE from Air lock (AL) or Gas lock (GL) in right side heart (RH) and Traffic Jam (TJ) in pulmonary tree (PT) in operative laparoscopy (OL), unscientifically implicating air for embolism even when CO<sub>2</sub>, O<sub>2</sub>, N<sub>2</sub>O, etc were used.

Technically, all gases are not synonymous to air or ambient air (AA); vide infra. AE or GE occurs for disparate mechanism in OL than in open surgery. In past, it used to occur in neurosurgical procedures conducted in the sitting position, orofaciodental surgery, caesarean section or vaginal delivery, orogenital sex, central venous catheterization, scalp incision, cervical spine fusion, penetrating and blunt chest trauma, high pressure mechanical ventilation, thoracocentesis, hemodialysis and several other invasive vascular procedures, even in diagnostic studies, such as during radiocontrast injection for computed tomography, even from pressurized intravenous infusion of normal saline in professional football players, etc.1 Yet, the blemish is extended to AA in OL by undue corollary.

#### Surgical smoke vis a vis nature of venous embolus

During OL with any gas, it is difficult to know exact nature of peritoneal gaseous admixture, i.e., surgical smoke (SS) due to electrosurgical pyrolysis at fateful time of embolism forming the mixture of gases, nonviable particles and viable viruses including blood fragments from 0.1 to 5.0microns known as lung damaging dust.<sup>2</sup> Gas embolus formed in venous system is product of such SS and is most important object, studied by host of workers.<sup>3</sup> SS is called by variety of names, including cautery smoke, plume, diathermy plume, smoke plume, aerosols, bio-aerosols, or just vapour. It is visible with odious odour.

# Mechanism of formation of surgical smoke and its nature

SS includes matter created when energy is transmitted to tissue cells during surgical procedures. Heat created by laser or electrosurgical energy is transferred to cells; vaporizes cellular fluid that increases pressure inside cell causing cell membrane to rupture. Cell disruption releases cellular fluid that vaporises and ejects cell contents forming SS with water vapor. Heat also causes thermal necrosis in neighbouring cells releasing diverse organic matters.

More than 600 chemicals are estimated in SS and few are hydrogen cyanide, acrylonitrile, isobutene, methane, benzaldehyde, benzene, benzonitrile, acetonitrile, acetylene, butadiene, butene, 2 methyl propanol, 3butenenitrile, carbon disulphide, pyrrole, indole, polyaromatic hydrocarbons, CO, phenol, creosols, ethane, ethyl benzene, ethylene, ethynyl benzene, formaldehyde, etc.2 Two highly toxic chemicals of concern are acrylonitrile and hydrogen cyanide.4 Acrylonitrile is volatile colourless chemical and liberates toxic hydrogen cyanide. CO forms in OL due to chemical reaction between hydrogen ions produced by combustion of cells and CO<sub>2</sub> i.e., H +CO<sub>2</sub>→OH+CO.<sup>5</sup> It then forms carboxyhemoglobin (HbCO) and methemoglobin (Met-Hb).6 Viruses of HIV, HbsAg, HCV, Human Papillomavirus and tubercle bacilli are also found.

It is observed that few cells remain viable also. Lower energy and shorter bursts are more likely to generate viable cells in SS. Cells may also be liberated by instrumental manipulation of tissues during OL. These cells are transported by gas flow in PP to remote ports due to gas leaks by chimney effect; harbinger of metastases at other than retrieval ports.<sup>7</sup>

Leakage of gases occur in and out of sides of cannulae from various ports mixing AA even when  $CO_2$  is used, against surgical will, compounding SS, which determines final chemistry of venous GE. Thus, precise composition of SS, hence ensuing embolus, in various stages of OL is difficult to reckon at the fateful moment of venous embolus origin.

#### Physical properties of gas and embolism

Lack of awareness of physical properties of matter and its phases in discussing their role relating to etiology and pathophysiology of venous GE shall only compound the confusion. Broadly, Gas represents one of four phases of matter, i.e., solid, liquid, gas, plasma. Gases are again subdivided into four types, viz. monatomic (noble), e.g., Neon, Argon; simple molecular (diatomic), e.g., O<sub>2</sub>, N<sub>2</sub>; compound molecular, e.g., CO<sub>2</sub>, N<sub>2</sub>O; or mixture, e.g., AA, SS. They are governed by plethora of physical laws, e.g., Boyles Law, Charles Law, Avogadro's law, Ideal Gas Law, Ideal Gas constant etc. Gas is governed by four characteristics, i.e., volume, temperature, pressure, number of particles or moles which affect their behaviour. They are difficult to quantify and qualify at a given moment of time in vivo in OL. Detail account of physics, chemistry of gases is beyond scope here, but these features make AE or GE a rather complex issue in OL. We still do not know exact amount or type of gas, mechanism of AE or GE, and pathophysiological events of AL or GL in RH and PT.8

# Early scientific evolutionary phase and developmental bumps

Such Phrases, "...air embolism associated with carbon dioxide insufflation few deaths from air embolism using carbon dioxide...". Abdomen tightly distended, ordinary unfiltered air, keep the cavity distended with air during the entire procedure" and "insufflation with a pressure of over 250mmHg indicate indicate little idea of effects of tight distension on biophysics and etiopathology of intraabdominal pressure (IAP) on AE or GE or infection from unfiltered air in nascent stages of early twentieth century. Plot 11 Faux pas of AE or GE perpetuates even when CO2 is used as late as 2005, 2012. 7.12

Kelling used air in dog to create IAP of 100mmHg in Lufttamponde published in 1901 as Kolioskopie. <sup>13-17</sup> Jacobaeus performed abdominal endoscopy in 1910 and carried out 115 cases with one serious complication of bleeding without AE. <sup>13</sup> From the time of Kelling, most gases viz. air, O<sub>2</sub>, He, CO<sub>2</sub>, N<sub>2</sub>O, were tried but none is found satisfactory. CO<sub>2</sub> is used empirically even when complications attributed to air are occurring with it. <sup>7,15</sup> It prompted technical review of biomechanics of CO<sub>2</sub> vis-avis AE or GE versus AL or GL in RH and PT, and other evolving pulmonary immunopathological factors.

### Few notable observations on gas or air embolism

Venous embolism is baffling despite current study on epidemiology and mechanism. Gottlieb et al state, "While the mechanism of death is now clear, the amount of air necessary to produce it is not clear." They further asserted, "This observation is no less true today." Hare observed that air necessary to cause death was an amount that could scarcely enter a wounded vein during operation by any possibility unless it was deliberately forced into

it.<sup>19</sup> Mirski et al stated, "Gas embolism may occur not only in an anterograde venous course, as is most typical, but also via epidural spaces, via tissue planes, and in a retrograde fashion either arterially or by venous channels."<sup>20</sup>

Serious consequences are rare in fairly common venous GE.<sup>21</sup> Fatal venous AE or GE can occur in OL under following conditions.

- A pressure gradient favoring passage of gas into circulation is required.
- Direct connection between source of air or gas and veins should exist.
- Mere presence of gas bubbles in venous tree is common yet inconsequential unless pathognomonic of moribund effects irrespective of gas. Coefficient for AE or GE should be yardstick of morbidity and mortality.
- Gas insufflation in or over raw area on solid organ is ominous. It can occur during solid organ surgery, e.g., laparoscopic cholecystectomy, OL on liver, kidney, uterus, etc with bleeding and if high IAP is used.
- High insufflation pressure is ominous during OL, e.g., >15 to 20mmHg.
- Gas trapped in portal venous circulation results in systemic GE by yet ill understood mechanism.
- Occurrence of paradoxical embolism without embryological anomaly, i.e., patent foramen ovale, patent ductus arteriosus, ductus venosus or some intrapulmonary arteriovenous malformations causing right to left shunt.<sup>22-24</sup>
- Others, yet ill understood miscellaneous mechanisms.

# Mechanism of gas lock versus embolism and pulmonary reactions

There is fundamental difference between AL or GL and AE or GE. Its basic understanding has profound impact, since it carries very high mortality rate of 28% once fully developed and 30% when it happens after central venous catheterisation. <sup>25,26</sup> Mere gas in venous tree goes without consequence since there is no clinical mishap irrespective of gas, e.g., AA, CO<sub>2</sub>, N<sub>2</sub>O, He, O<sub>2</sub>.It is GL and TJ in RH and PT that results in mortal event of GE, irrespective of gas. Gas bubbles are compressed during systole and not pushed in PT like liquid blood in right lateral or supine positions. In diastole, gas bubbles expand impeding blood flow by GL and TJ.<sup>27</sup>

It causes diminished cardiac output, myocardial and cerebral ischemia, arrhythmias, hypercapnia, hypoxia, and hypotension with cardiovascular collapse. Some vasoactive factors and bronchospasm are also incriminated to compound mechanical obstruction in RH. How and why gas causes symptoms or fatality in some and spares others is baffling?

It is not the type of gas (O<sub>2</sub>, CO<sub>2</sub>, He, N<sub>2</sub>O, AA) but the size and speed of creation of GL and TJ in RH and PT that matters. Thus, GL and TJ form main facets for and hemodynamic cardiopulmonary disturbances engendered by massive GE besides its secondary lung immunopathological reactions.<sup>28</sup> Whatever the gas, once inside venous system; travels in RH, where it remains trapped innocuous or cause GL and TJ or pass down PT? Its pathological dispensation and outcome depends on position of patient, size of gas bolus, degree and place of GL, speed of gas entry, competent anaesthetist, diligent surgeon, prompt diagnosis, suitable auxiliary support, expedient management and cardiopulmonary status of patient, but type of gas seems less relevant practically.

Durant et al postulated on mechanism of fatal AE: 1) amount of air entry to the circulation, 2) speed with which it enters, 3) the position of body at the time of embolic accident, 4) efficacy of pulmonary gaseous exchange mechanism.<sup>27</sup> Death occurs due to circulatory obstruction resulting from AL or GL in RH and PT. Displacement of AL by turning the body in left lateral position may save life even when right ventricular contractions have become feeble. They hypothesize that fatal syndrome of GE is hidden in GL or TJ against flow of blood in and out of RH, especially if body is in supine or right lateral position. But Smulders, Zheng et al and Mehlhorn et al contend other forces operate than body posture.<sup>28-30</sup> Smulders deliberated on the issue of pulmonary vasoconstriction and bronchospasm. "Furthermore, bringing about a strictly mechanical obstruction by cross clamping the left or right pulmonary artery during a surgical procedure, or by unilateral balloon occlusion, causes only a modest rise in pulmonary artery pressure (PAP), and almost never results in right sided heart failure, whereas PE (pulmonary embolism) with obstruction of only ±25% of the pulmonary vascular tree can cause marked pulmonary hypertension. Also, major PE can be found during autopsy in patients who never had any clinical manifestations of PE." This is a very powerful observation.

### Brief lung immunopathophysiology in venous gas embolism

Gas embolus in pulmonary vascular system can initiate myriads of complex molecular, cellular and physiological changes in endothelium and pulmonary alveoli due to vet ill understood factors besides mechanical obstruction to hemodynamics.<sup>28</sup> It can trigger serious inflammatory changes in pulmonary vessels by direct injuries, e.g., endothelial damage, accumulation of various blood components, platelets, neutrophils, fibrin etc.<sup>1</sup> It also initiates secondary changes due to complement activation, release of inflammatory mediators and free radicals, e.g., reactive O<sub>2</sub> and N<sub>2</sub> species, release of cytokines (TNF-α and interleukin-1 degranulation of mast cells, activation of macrophages, liberate prostaglandins, interferon, histamine, serotonin, leukotrienes, kinins (e.g., bradykinin), etc.<sup>31</sup> There is increased vasodilation, capillary permeability with noncardiogenic pulmonary edema, right to left shunting; more dead space, hypoxia, hypercapnia, ventilation perfusion mismatch, wheeze and bronchospasm; accentuating TJ further.<sup>29,30</sup>

It can lead to acute respiratory distress syndrome, systemic inflammatory response syndrome aggravated by infection leading to multiple organ dysfunction syndrome and disseminated intravascular coagulation with diffuse fatal bleeding. <sup>32,33</sup>

### Experimental studies of clinical relevance

Lot of experimental studies have been done with a view to determine the precise mechanism of GE and GL and amount of gas needed for fatal complications but without conclusions. Durrant et al added, "Our experience has been that the dosage which will produce fatality is extremely unpredictable, and, while there is undoubtedly some relationship to the size of the animal, many other factors are involved which greatly complicate any attempt at calculation." Gordy and Rowell stated, "The lethal dose for humans has been theorized to be 3-5ml/kg and it is estimated that 300-500ml of gas introduced at a rate of 100ml/sec is a fatal dose for humans." Ambiguity on type and amount of gas still persists for inability of replication of animal in vitro studies into in vivo human species.

Lethal volume is suggested six times more for  $CO_2$  than AA. Reasons cited are (a) High solubility of  $CO_2$  (b) Buffering capacity of blood for  $CO_2$  (c) rapid excretion of  $CO_2$  through lungs. Acidosis and production of  $CO_2$  caused by  $CO_2$  is ignored. These theoretical imputations deduced from controlled experimental studies in vitro appear prima facie favourable for  $CO_2$  over air, but in vivo events occur under totally uncontrolled and unknown conditions.

In OL, exact time, amount of gas or SS entering blood stream, its speed, several other biomechanical, kinetic and pathophysiological factors remain beyond our control in PP. In human beings, kind of control and experimental conditions needed is difficult to impute since there are multiple uncertain reactions occurring instantly, when study may be envisaged. As stated earlier, true incidence of venous GE is unknown during OL due to silent emboli. Further it will be difficult to decide cause of death and amount of SS involved due to absorption of SS from venous tree postmortem, prior to autopsy.<sup>34</sup> It is notable that human beings are not dispensable pari passu laboratory animals, which seriously limit application of bioexperimental experience to humans.

### Some measures for prophylaxis of venous gas embolism

Normal IAP in man varies from -5 to +7mmHg with average of  $\pm 2$  mmHg.<sup>35</sup> Insufflation of peritoneum de

facto is tension PP. High index of suspicion and awareness of GE or AE in OL is most important.<sup>36</sup> Some cautions are advised for surgeons and anaesthetists for prophylaxis of AE or GE.

- Careful veress needle entry especially in patients with previous surgery, very thin patients with a narrow costal angle, obesity, slim elderly patients with flail muscles especially multiparous females with patulous belly, paediatric setting.
- Select port site away from expected abdominal adhesions, e.g., Palmer's point in left hypochondrium or left iliac fossa.
- Advisable to lift anterior parietes up while introducing Veress needle which should be checked for its sharpness, spring action and patency.
- Initial gas insufflation must be slow, e.g., ≤1L per minute and increase slowly to allow hemodynamics to settle in next few minutes.
- Check liver dullness and four quadrant resonance of abdomen throughout insufflation carefully, especially in patients with expected adhesions.
- IAP in PP should not exceed 10 to 12mmHg. IAP >15mmHg runs risk of embolism irrespective of type of gas used.
- If procedure is prolonged or there is too much smoke generated or plasma ion gas like Argon is used; gas should be renewed often for (a) keeping IAP as low as possible (b) refreshing quality of contaminated gas or SS after use of electrosurgery (c) Gases like Argon diffuse in large volume causing sharp rise of IAP, warranting extra care.
- Deep anaesthesia for good muscle relaxation to thwart fictitious high IAP.
- Similarly avoid leaning over or resting of arm on abdomen intraoperatively.
- Use diligent surgical technique to avoid opening undue surgical planes or vascular injuries causing GE by direct gas injection or Venturi effect.
- Preload of suitable intravenous fluid preoperatively is advisable for volume compensation at every cost. Little overhydration is preferable if cardiovascular status permits, to counter postural effects on cardiac output.
- Close liaison between surgeon and anaesthetist is essential in every patient. Surgeon should explain his plan to anesthetist alongwith anticipated unplanned events.
- Surgeon, anaesthetist competence factor.
- Awareness of AE or GE is vital despite all precautions or type of gas.
- Anaesthetist must beware of emergent resuscitation to deal with embolism efficiently shifting patient in Trendelenburg and left lateral position.<sup>26,27</sup>
- If CO<sub>2</sub> is used, COHb and metHb formation is more warranting care in old, cardiorespiratory cripples and pregnant patients wherein AA may be preferable.

- Toxic products of tissue pyrolysis are more in anerobic milieu using CO<sub>2</sub> during cutting and coagulation; making AA preferable.
- Hasson technique or optiview trocars are no safer substitutes for selection of proper site of first entry point of trocar to obviate complications.
- In modern anaesthesia where positive pressure ventilation forms an integral part in major surgery, it can lead to pulmonary air leak syndromes; harbinger of GE.<sup>37</sup> Inappropriate ventilatory pressure can cause iatrogenic lung barotrauma resulting in pulmonary interstitial emphysema, respiratory distress syndrome and GE, unrelated to gas used in OL.
- Careful use of N<sub>2</sub>O in anesthesia to save diffusion in embolus and intestines.
- Role of transesophageal echocardiography in gas Embolism is limited only to diagnostic value and is a highly technically demanding and invasive tool. Its experts are very few and available in selected centres globally. It has no direct role in treating or preventing AE or GE, where expert anesthetist surgeon team is vital.

### CO<sub>2</sub> versus ambient air debate and human psyche: jus naturale

Once vox populi was established against AA in OL; psychology en mass carried it forward like Law of Natural Justice or Jus Naturale. Even a whimper of embolism would cast instant revolt against AA. Sudrania having used both CO<sub>2</sub> and AA feels that AA in OL appears safer, better, economic since it is available ubiquitously free, hassle free of cylinders and less painful. Park et al observed, "...carbon dioxide can cause hypercapnea, metabolic acidosis, cardiorespiratory compromise, and greater postoperative pain as well as having adverse effects on intraperitoneal immune function, even increasing the risk of port-site tumor metastases in experimental models. Consequently, alternative gases have been investigated to reduce negative side effects."

 $CO_2$  is irritating to peritoneum, pungent, more painful than air besides acidotic effects for carbonic acid formation ( $CO_2 + H_2O \rightleftharpoons H_2CO_3$ ). As stated earlier, CO also forms more with use of  $CO_2$  in OL. Fan et al also confirm, "The production of CO is enhanced by presence of high carbon dioxide levels in the peritoneal cavity and increased intraabdominal pressure during laparoscopic surgery." ...CO level in patient's blood is significantly increased after a laparoscopic cholecystectomy even in healthy non-smokers."<sup>38</sup>

### Surgeon team factor

Laparoscopic surgeon should be patient and calm as OL is very taxing. Cushieri deliberates on surgeon factor that appears to be the solution of most problems in OL including embolism.<sup>39</sup> The renowned report '*To Err is Human*' by Kohn et al led to increased awareness to

focus on reducing preventable iatrogenic surgical errors including surgeon factor. 40 Surgical team is least discussed factor but seems the most important factor in the surgical outcome. 27,41,42 It amply reflects in differing results with different surgeons and OL is no exception. Makary and Daniel (2016) cautions on medical errors as third largest cause of death after heart disease, cancer, the science of safety has matured to describe how diagnostic errors, poor judgment, and inadequate skill can directly result in patient harm and death. 43 This is further emphasised by, advances in the prevention and control of HAIs (AHRQ): advancing the science of patient safety and quality improvement to the next level under aegis of U.S. health and human services. 44

Laparoscopic surgeon, anaesthetist and operation theatre staff get tired and tend to rush during later stages of OL.<sup>39</sup> Factors that disturb concentration, e.g., overcrowding, noise or muttering in theatre, exhaustion from lack of sleep or overwork, stimulants like nicotine or caffeine which induce mental hyperactivity; diminish mind acuity and patience; should be avoided. Surgeon may take a break of five to ten minutes after every hour of OL if needed. This paradoxical delay pays out in increased dexterity and improved ergonomics in prolonged surgery. Taking small refreshing sweet drink to replenish depleting blood sugar to energise brain cells is preferable. If the procedure is likely to take longer than two to three hours, it is advisable to work with two coordinated surgical teams or take longer break to escape acute fatigue syndrome.

Attempt should be made to identify factors for optimal performance of surgical team, e.g., personnel with major impact on surgical team work and implement them in practice to improve the dexterity of the whole team. Also identify nursing staff, surgical assistants, anaesthetic team and other ancillaries concerned, which should be acknowledged and encouraged.

### **DISCUSSION**

Precise mechanism of fatal AE versus AL still remains enigmatic and silent emboli go undetected with imprecise incidence. It is postulated that fatal venous AE or GE is due to large gas bubble causing AL or GL and TJ in RH and PT, besides some nascent immunopathological reactions in lungs. AL or GL must be cleared within time limit of cerebral anoxia for irreversible brain death that is extremely variable for individuals between five to twenty minutes or so in normothermic ischemic anoxia. 45,46 This short-stipulated time may not be enough for any gas to clear in RH devoid of other venous outlets than PT and without active interference. By the time, symptoms start declaring, events of embolism may have far advanced for our cognitive senses to salvage life. Fast CO<sub>2</sub> solubility diffusion coefficient hypothesis may appear consolation to us instead of a solution for fast worsening patient. In evolving laparoscopy history, high IAP was used for lack of knowledge of mechanism of fatal GE. Often gases used were other than AA, yet air or gas was and is blamed for embolism instead of CO<sub>2</sub>.<sup>7,12</sup> Venous GE may result even without a plausible cause, e.g., young woman died of CO<sub>2</sub> insufflation for diagnostic laparoscopy.<sup>47</sup>

### **CONCLUSION**

Use of AA appears common and lot of colleagues had reported it to Sudrania (Personal Communications) but hastened to say, "We do not talk of it.". Its legitimacy may help surgeons globally who may be using AA under compulsion where cost and availability of CO2 is prohibitive. Thus, extend benefits of OL to the needy to save long painful postoperative convalescence of open surgery, especially the poor daily wage earners. Analogically, electrosurgery had lot of serious complications earlier but its use was not abandoned; instead, it was refined and improved to make it safer for clinical use. Today electrosurgery is most important tool of modern surgery, without which surgery inconceivable. Similarly use of AA in OL should be considered empathetically in light of the current scientific, technological developments and improved knowledge of the physics of tension PP besides newer anesthetic technological equipments and advancements.

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