Pneumoperitoneum

Physiological consequences
Anaesthetic implications

Suzy Cook
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Overview

• What is it?
• Physiological consequences
  – CV, resp, other
• Why do we bother? benefits vs risks
• Anaesthetic considerations
Pneumoperitoneum

• The presence of gas or air in the peritoneal cavity
  – Spontaneous
    • eg. ruptured abdominal viscous (eg. peptic ulcer)
  – Iatrogenic
    • Laparoscopic surgery
Laparoscopic Surgery

• History
  – early 70’s - gynae for lx/Dx
  – 83 – appendicectomy
  – late 80’s - cholecystectomy

• Intra-abdominal pressures (IAP)
  – initially higher >20 mmHg
  – since ~1990 <15 mmHg

• Now used for increasingly complex procedures
  – GI   colonic, gastric, splenic, hepatic surgery
  – gynae  hysterectomy
  – urol  nephrectomy, prostatectomy
  – vasc  aortic surgery
Laparoscopic Techniques- pneumoperitoneum

choice of gas depends on

- solubility in blood (↑sol → ↓risk gas embolism)
- permeability in the tissues
- combustibility
- expense
- other systemic side effects

- air
  - ↑ risk of gas embolism
  - supports combustion
- N₂O
  - supports combustion
  - associated with sudden cardiac arrest
- O₂
  - supports combustion++
- Helium
  - inert, not absorbed from peritoneum
  - ↑ risk of gas embolism
- CO₂
  - doesn’t support combustion
  - highly soluble in blood/tissues
  - inexpensive
  - seems to stimulate CV system → partial correction of HD Δ due to ↑ IAP
  - causes hypercarbia, resp acidosis, shoulder tip pain
• CO$_2$ insufflated at ~1-2 L/min
• ~25-30L insufflated during average procedure

• eliminated via lungs
  – rapid d2 $\uparrow$ sol/diffusibility in healthy pt’s
    $\downarrow$ if alv vent impaired by mechanics/CV disease
Physiological Consequences
-Factors Involved

- pneumoperitoneum
  - $\uparrow$ PaCO$_2$
  - $\uparrow$ intra-abdominal pressure (IAP)

- patient positioning
  - head down/Trendelenberg
    - eg. for upper abdo surgery
  - head up/reverse Trendelenberg
    - eg. for pelvic/lower abdo surgery
  - lithotomy
    - to facilitate surgical access caudally
Causes of \( \uparrow \text{PaCO}_2 \)

- absorption from peritoneal cavity
  - main factor (partic in young/healthy)
    - minimal \( \Delta \) in \( \text{PaCO}_2 \) when CO\(_2\) not used
  - proportional to CO\(_2\) pressure
  - absorption \( \uparrow \) with extraperitoneal insufflation

- impairment of vent/perfusion by mechanical factors
  - \( \uparrow \) IAP, pt position, IPPV
  - \( \downarrow \) ventilation due to drugs
    - ie. during spontaneous ventilation

- \( \text{PaCO}_2 \) generally returns to normal within 1hr of desufflation
  (may take several hrs following prolonged procedure)
\[ \uparrow \text{PaCO}_2 \text{ also depends on anaesthetic technique} \]

- **Regional**
  - \( \text{PaCO}_2 \) unchanged due to \( \uparrow \uparrow \) min vent

- **Mech vent GA**
  - \( \text{PaCO}_2 \) \( \uparrow \) to reach a plateau \( \sim 15-30 \) mins after start of insufflation
  - \( \uparrow \) by \( \sim 15-25\% \)
  - related to \( \uparrow \) IAP
  - Any significant \( \uparrow \) after this requires Ix for another cause

- **Spont vent GA**
  - \( \uparrow \uparrow \text{PaCO}_2 \) due to
    - anaes induced vent depression
    - \( \times \) \( \uparrow \) WOB due to \( \downarrow \) compliance
    - \( \times \) \( \uparrow \) RR not enough to compensate
CV effects of $\uparrow \text{PaCO}_2$

- $\text{PaCO}_2$ 45-50mmHg
  - no signif HD effects
- $\text{PaCO}_2$ 50-70mmHg
  - indirect effects via sympathetic stimulation
    - $\uparrow \text{HR}$
    - $\uparrow$ risk arrhythmias
      - peripheral vasoconstriction $\rightarrow \uparrow \text{SVR}$
  - counter balanced by direct effects on BV
    - periph vasodilation incl cerebral
  - direct myocardial depressant
CV effects of ↑ IAP
regardless of gas used

↑ IAP

pooling of blood in legs

↑ venous resistance

↑ intraTx press

stimulation peritoneal repts

↑ vasc resist of intra-abd organs

release neurohumoral factors (catecholamines, RAS, ADH)

↓ venous return

↓ inotropism??

↑ SVR

↓ preload

↑ afterload

↓ cardiac output

↑ MAP
CV effects of ↑ IAP

- dependant on vol of gas & pressure
- effects exaggerated in head up position

- ~14 mmHg → CO maintained

- ~20 mmHg → CO maintained
  - small ↑ ICP

- ~30 mmHg → CI falls to ~50% of preop in 5 min
  - ↑ pressure on IVC
  - ↓ CVP (but remains higher than pre insufflation levels)

- ~40 mmHg → CO ↓ by 17% (normovol), 53% (hypovol)
  - ↓↓ VR & R heart filling pressures
• 25% ↑ MAP
• 38% ↑ LV systolic wall stress
• 25% ↓ LV EF
• 18% ↓ LV SV related to ↑ afterload
  – compensated for by ↑ HR
    → CO unchanged (CO = SV x HR)
• impaired LV diastolic function without change in LVEDV
• effect on preload was negligible in adequately volume loaded patients
**CV effects due to positioning**

- head down/Trendelenberg
  - VR, CVP $\uparrow$
  - CO $\uparrow$
  - MAP $\uparrow$
  - intravasc press in lower torso/pelvis
    - $\downarrow$ blood loss
    - $\uparrow$ risk gas embolism
  - barorepts $\rightarrow$ vasoD/$\downarrow$ HR $\rightarrow$ stabilisation
  - $\uparrow\uparrow$ in cor art disease
  - can $\rightarrow$ detrimental $\uparrow$ myocardial $O_2$ demand
• head up/reverse Trendelenberg
  - CO ↓ (proport to degree tilt) due to ↓ VR (↓ preload)
  - MAP ↓
  - venous stasis

- barorepts → ↑ SNS
  → ↑ SVR/HR
  → compounds Δ’s due to pneumoperitoneum

• lithotomy
  - elevation legs
    - acute ↑ VR → exacerbating/precipitating cardiac failure
  - lowering legs
    - acute ↓ VR → hypotension
  - magnitude of effects depend on volume status
Arrhythmias

usaha

vagal tone

- eg. peritoneal stretch, diathermy fallopian tubes

if:

- anaesthesia light
- B-blockers
- preexisting CV disease
- gas embolism
Thromboembolism

• venous stasis
  – immobility
  – head up position
    ↑ IAP

• BUT...
  – incidence of TE doesn’t appear to be ↑
Pre-existing CV disease

- HD changes are less well tolerated
  - ↓ SvO₂ in 50% of ASA 3&4 despite preop HD optimisation
- △↑ afterload is the main factor
- more severe with deplete IV volume
  - ↓ CO
  - ↓ CVP
  - ↑ MAP
  - ↑ SVR
- NB. HD changes are well tolerated in morbidly obese
Resp effects of ↑ PaCO₂

- chemoceptor stimulation of resp centres
  - ↑ ventilation
    - each ↑ of 1mmHg of PaCO₂ → ↑ vent by ~2L/min
Resp effects of ↑ IAP

- ↓ compliance by 30-50%
  - not affected by subsequent tilting/↑ min vent
- ↑ airway pressures
  - peak ↑ by 50%, plateau ↑ by 81%
    ↑ intrathoracic pressure
- ↓ FRC
  - due to diaphragm elevation
- Δ distribution vent/perfusion
- ↑ atelectasis

- in pt’s without CV disease
  → no Δ in physiological dead space/shunt
Resp effects due to positioning

- head down/Trendelenberg
  - FRC ↓
  - total lung vol ↓
  - complicance ↓
  - atelectasis ↑
    ↑ VQ mismatching

- head up
  - only minimal Δ in compliance/gas exchange

- lithotomy
  ↓ FRC

↓↓ in steep head down/obese/elderly/debilitated
Renal effects

- due to ↑ IAP
  - ↓ RBF
  - ↓ GFR
  - ↓ urine output

- urine output signif ↑ after desufflation

\[ \text{to less than 50\% of baseline} \]
Cerebral effects

• due to $\uparrow \text{PaCO}_2$
  – vasodilation $\rightarrow \uparrow \text{CBF} \rightarrow \uparrow \text{ICP}$
  – ICP also $\uparrow \text{independent of PaCO}_2$ to some degree
    • children with VP shunts, pigs
  – IOP
    • not affected in no pre-existing disease
    • slight $\uparrow$ in animals with glaucoma

• due to positioning
  – head down/Trendelenberg
    • ICP/IOP $\uparrow$ due to venous congestion
  – head up
    • ICP $\downarrow$
GI effects

• lower oesophageal sphincter
  $\uparrow$ IAP $\rightarrow \Delta$ LOS $\rightarrow$ barrier pressure maintained

• splanchnic/hepatic blood flow
  – due to $\uparrow$ IAP
    $\not\uparrow$ or $\downarrow$ depending on study =controversial
    • effects not likely to be significant
Patient Benefits

‑ ▼ stress response
‑ ▼ metabolic response
  ‑ lessens ▲ WCC, bsl
  ‑ ▲ better preserved immune function
‑ ▼ pain post-op
‑ ▼ pulm dysfunction post-op
  • avoids prolonged exposure & manipulation of intestines
    ‑ ▼ ileus, earlier return GI function
‑ ▼ adhesion formation
Disadvantages

- greater HD instability
- greater impact on intra-op resp function
- duration of procedure
- surgical learning curve
- difficulty in assessing blood loss
- post op pain due to residual pneumoperitoneum
- post op N+V
- complications

- lap append didn’t offer any significant advantages over open append at 2 weeks post-op wrt:
  - pain scores & medications
  - resumption of diet
  - length of stay
  - activity scores
- however, improved QOL scores
- took longer to perform (& more expensive)
- “choice of procedure should be based on surgeon or patient preference”
Complications of Laparoscopy

• complx related to surgical procedure
  – less obvious than with open
    • concealed hemorrhage, retroperitoneal haematoma
    • delayed: subhepatic/phrenic abscess
• gas where it shouldn’t be
• complx related to positioning of patient
• anaesthetic complx

Recently:

↓ death rate but ↑ complx rate due to ↑ complexity of procedures
  current complication rate ~5%
Gas where it shouldn’t be

- intrapleural
- mediastinum
- pericardium
- subcutaneous
- embolus
pneumothorax, -pericardium, -mediastinum

• caused by:
  ↑ IAP → gas tracking through potential channels due to embryonic remnants or defects in the diaphragm
  – pleural tears during surgery
  – rupture of pre-existing pulmonary bullae
    • due to ↑ alv ventilation/pressures during IPPV
Pneumothorax

- Clinically
  - ↓ compliance
  - ↑ airway pressures
  - → ↓ SaO₂
  - ± sub cut emphysema
- Dx = auscultation (↓ bs, ↑ resonance), CXR
- tension pneumothorax can occur

Capnothorax

▷ ↑ PaCO₂ / ETCO₂ due to ↑ absorb area in pleural cavity
- Guidelines intra-op for capnothorax:
  - stop N₂O, ↑ O₂ to correct hypoxia
  - PEEP
    - ↓ IAP as much as possible, close communication with surgeon
    - avoid thoracocentesis unless necessary
- if CO₂/N₂O used → spont resolution 30-60 mins post desufflation

Pneumothorax from bullae rupture

▷ ↓ ETCO₂ due to ↓ CO
- Guidelines for bullae rupture as for capnothorax EXCEPT:
  - no PEEP
  - thoracocentesis mandatory
Subcutaneous emphysema

- due to extraperitoneal insufflation
  - accidental
  - intentional eg IH repair, HH repair
- extent of emphysema is proportional to gas pressure
  $\Delta \text{PaCO}_2$, ETCO$_2$, CO$_2$ elimination
- $\text{PaCO}_2$ may not be able to be normalised by $\uparrow$ vent
  $\Rightarrow$ pause insufflation to enable correction of $\uparrow \text{PaCO}_2$
  $\Rightarrow$ then resume at $\downarrow$ insufflation pressures
- doesn’t imply pneumothorax
- resolves after desufflation
- doesn’t CI extubation
  $\Rightarrow$ but mech vent recommended until hypercapnea corrected
Gas embolus

- due to:
  - direct needle/trocar injection into bv
  - gas insufflation into abdo organ
- timing:
  - mostly at induction of pneumoperitoneum
- risk ↑ with:
  - head up position
  - prev abdo surgery
  - less soluble gas (lethal dose \( \text{CO}_2 \) ~5x that of air)
- frequency:
  - gas embolus in 68.75% (16 pts ASA 1-3 TOE during lap chole)
    - 45% during peritoneal insufflation
    - 55% during gallbladder dissection
    - mean duration ~170 secs
    - minimal HD instability (no Δ HR, SpO\(_2\))
  - clinically apparent \( \text{CO}_2 \) embolus
    - 0.013% of gynae laparoscopies
effects with:
- size of bubbles, rate of intravenous entry

- ≤0.5ml/kg
  - ↑ PAP
    - change in Doppler sounds
- ~2ml/kg
  - ↓ ETCO$_2$ due to ↓ CO & ↑ dead space
    - after initial ↑ due to ↑ pulm excretion CO$_2$
  - ↑ HR, arrhythmias
  - ↓ MAP
  - ↑ CVP
    - “millwheel” murmer
    - R heart strain on ECG
      - ↓ SpO$_2$ / cyanosis
    - “foamy” blood or gas aspirated from CVC
• 20-30% of people have patent foramen ovale
  – acute ↑ RV pressure → causes FO to open
    → paradoxical embolism to L heart
  – embolism to cerebral & coronary circulations

• treatment
  – cease insufflation, release pneumoperitoneum
  – position steep head down, L lat
  – 100% O₂
    ↑ ventilation to ↑ CO₂ excretion
  – CVC/PAC to aspirate gas
  – CPR can help fragment gas into smaller bubbles
  – CPB for massive embolus
  – HBO₂, partic for cerebral embolism
Complx due to positioning

• endobronchial intubation
  ↑ risk with head down, lithotomy
  → ↓ SpO₂, ↑ airway pressures

• nerve injury
  – lithotomy
    • common peroneal (lat)
    • saphenous (med)
    • obturator/sciatic (excessive flexion hip)
  – shoulder braces
    • brachial plexus
  – arm over-extension
    • ulnar nerve

• compartment syndrome of lower extremities
  – prolonged lithotomy position
Anaesthetic complex

• ~1/3 of deaths assoc with laparoscopic procedures were related to anaesthetic complex during GA without intubation
Anaesthetic considerations
pre-op

• absolute CI = rare
• relative CI
  ↑ ICP
  – hypovolaemia
  – VP or peritoneojugular shunt
    • safe if shunt clamped prior to insufflation
• $H_x/E_x$
  – severe CCF/terminal valve insufficiency
    • more likely to develop CV complx than IHD
  – renal failure
  – resp disease
    • reduced post-op pulm dysfunction
• $I_x$
  – ± echo
Anaesthetic considerations

intra-op 1

• optimisation of HD/volume status
  – partic CV disease, renal failure

• monitoring
  – ETCO$_2$
    • PaCO$_2$:ETCO$_2$ = variable, ↑ more in CV/resp disease
  – SpO$_2$
  – ECG
  – (Pulm art catheter)

• positioning
  – tilting slow & progressive
  – check ETT position after each Δ pt position
intra-op 2

• anaesthetic technique
  – GA
    • ETT & mech ventilation = safest
    • ?LMA → recommendations:
      – short cases, ↓ IAP, ↓ degree of tilt, healthy/thin pts
    • ETCO₂ maintained ~35mmHg
      – (~15-25% ↑ in minute ventilation)
    • ± omission of N₂O
      – no clinical advantage but improves surgical conditions
  – regional
    • need extensive block (T4-L5), doesn’t prevent shoulder tip pain
    • useful for severe resp disease
  – local
    • needs gentle/precise surgical technique, relaxed/cooperative pt, ↓ IAP, minimal port sites
intra-op 3

• induction
  – avoid over inflation of stomach
• drugs
  – vasodilators can improve afterload
  – adequate muscle relaxation to minimise ↑ IAP
• ± NGT to deflate stomach
• ± IDC to deflate bladder (lower abdo surg)
• avoid hypovolaemia
• DVT prophylaxis as usual
Anaesthetic considerations post-op

خفض تدفق الأوكسجين - pulmonary dysfunction
  - 
  - but PaO₂ still ↓, O₂ demand ↑
  - O₂ administration recommended

• post-op
  - usual monitoring
  - analgesia
In summary...

• pneumoperitoneum
• physiological consequences
  – CV, resp, other
• benefits vs risks
• anaesthetic considerations