NON-INVASIVE BLOOD PRESSURE - NIBP

a.	palpatory	- measure at pulse return ~ systolic ± 5-8 mmHg
b.	limb flush	~ systolic ± 10 mmHg
c.	oscillometric	- systolic & mean, no diastolic - mean very accurate - systolic ± 5 mmHg
d.	ultrasonic	- systolic & diastolic accurate
e.	pulse arrival	- time interval QRS - peripheral pulse
f.	auscultatory	- Korotkoff's sounds
	i. phase I	- snapping tones ~ systolic
	ii. phase II	- murmurs (may be low pitch/inaudible)
	iii. phase III	- thumping
	iv. phase IV	- muffling
	v. phase V	- silence ~ diastolic

ARTERIAL LINES

- there are no absolute, only ***relative indications***,
    1. continuous arterial pressure monitoring
    2. arterial pressure waveform analysis
    3. repeated arterial blood tests
    4. research

- ***Advantages***

    a. ease of cannulation
    b. accessibility
    c. usually adequate collateral circulation
    d. low complication rate

- ***Absolute Contraindications***

    a. AV shunt for dialysis
    b. severe Raynaud's disease
    c. absence of collateral circulation (but not +'ve Allen's)
    d. Buerger's disease
    e. Wegener's granulomatosis

- ***Factors Which Optimise Signal Fidelity***

    a. equipment factors
        i. high frequency transducer response
        ii. short, stiff, non-compliant tubing
        iii. elimination of air bubbles from tubing < 0.25 ml → underdamping
        > ? → overdamping
        iv. a slow continuous flush device
        v. correct calibration of the transducer against Hg column
    b. patient factors which reduce signal fidelity
        i. rapid heart rates → resonance
        ii. reflection of high frequencies within circulation
            - elderly, high SVR, atherosclerosis
        iii. site of catheter insertion - radial vs. femoral
        - axillary vs. aortic
        iv. profound vasodilatation - post-CPB

- ***Information Gained***

  a. systolic and diastolic trends

  b. accurate mean

  c. hypovolaemia
     - increased pulse paradox
     - lower dichrotic notch
     - steeply peaked systolic wave

  d. decreased contractility
     - reduced upslope
     - reduced peak pressure

  e. indication of myocardial O_2 supply/demand
     - pulse pressure product
     - systolic area vs diastolic area

  f. pulsus paradoxus
     - hypovolaemia
     - δSAP up/down
     - tamponade, constrictive pericarditis
     - high intrathoracic pressure
     - severe CCF, myocarditis
     - RV AMI
     - PTE
     - ascites
     - pregnancy

  g. hyperdynamic pulse
     - sharp rise & fall
     - sepsis
     - AI
     - AV fistula
     - anaemia
     - thyrotoxicosis
     - pregnancy

  h. pulsus alternans
     - pericardial effusion
     - severe LV dysfunction

  i. pulsus bisferens
     - AI

  j. access for frequent blood analyses

- ***Misinformation***

  a. poor guide to ***perfusion***

  b. poor guide to ***myocardial performance***

  c. ***errors***
     i. calibration / drift
     ii. amplification
     iii. resonance
     iv. damping

- ***Complications***

a. ***thrombosis*** ~ 60% overall incidence
   i. duration ~ 50% at 2/7, but minimal < 24 hours
   ii. size of canulae - 20G better in adult
   iii. wrist size - approximates arterial diameter, < 18 cm ↑ incidence
   - female > male
   iv. catheter material - teflon/vialon are best
   v. flush system
   vi. systemic hypotension
   vii. technique - number of cannulation attempts
   - Seldinger > direct

b. ***haematoma*** ~ 50%

c. accidental haemorrhage

d. sepsis - related to technique and duration
   - increases after 4-5 days

e. distal emboli ~ 2-4%

f. thumb/hand ischaemia - transient in 10%

g. proximal forearm ischaemia

h. aneurysm

i. AV fistula

j. inadvertant drug administration

- ***Calibration of Transducers***

a. ***static calibration***
   i. zero
   ii. gain
   iii. linearity
   iv. stability with time, temperature variation

b. ***dynamic calibration***
   i. high frequency response - ideally ~ 10x fundamental frequency
   ii. damping coefficient, x **~ 0.677**

CENTRAL VENOUS CATHETERS

- ***Indications***

a. CVP measurement

b. vascular access - difficult or prolonged

c. hypertonic or irritant fluids - TPN, HCl
- inotropes

d. infusion of large volumes - ***not*** for rapid administration

e. other therapies - pacemaker
- PA catheter
- haemodialysis, haemoperfusion, plasmapheresis

- ***Complications***

a. ***during insertion***

i. failure to site in SVC ~ 55% cephalic / 35% basilic
~ 10% EJV / ***0-4% IJV***
~ 5% subclavian (some ≤ 25%)

ii. haematoma

iii. arterial puncture ~ 5% with subclavian approach
~ 1-2% with IJV approach

iv. pneumothorax ~ 2% with subclavian approach
< 1-2% with IJV approach

v. damage to other structures - vagus/recurrent laryngeal nn.
- stellate ganglion, cervical plexus
- thoracic duct, trachea, ETT cuff !

b. ***during use***

i. venous thrombosis - hypertonic solutions
± thromboembolism

ii. colonisation, infection, bacteraemia, septicaemia
- ~ 10% of colonised → bacteraemia

iii. accidental removal

iv. venous perforation - catheter stiffness
- duration & site

v. embolisation - air, thrombus, septic thrombus
- catheter tip (shearing)
- AV fistula

c. ***during removal***

i. embolisation

ii. haematoma formation

PA CATHETERS

• 1953 Lategola & Rahn used hand-made, balloon-tiped catheter for PA catheterisation & occlusion measurement in dogs
• Swan *et al.,* 1970 NEJM, reported used of multilumen, balloon-tipped, radio-opaque PVC catheter with the following charcteristics,

1. reliable, prompt passage into the PA
2. minimal arrhythmias
3. passage without fluoroscopy

• balloon occluded PA pressure, ***pulmonary artery occlusion pressure (PAoP)***, showed good correlation with traditional ***pulmonary capillary wedge pressure*** measurements
• the later using traditional stiff right heart catheters "wedged" into small pulmonary vessels
• subsequent studies confirmed correlation between PAoP, PWP and LAP by direct measurement

• since then a multitude of catheters have been described, uses including,

1. in-vivo oximetry
2. pulmonary angiography
3. paediatric catheterisation
4. His-bundle electrocardiography
5. thermodilution CO estimation
6. LV pacing

Indications

1. optimisation of ***LV preload***, where the CVP will not reflect LVEDV
   - i. LV dysfunction - present or anticipated
     - severe IHD - global or regional dysfunction
       - recent myocardial infarction
       - ischaemia-induced valvular dysfunction
     - cardiomyopathy
     - valvular heart disease - this is argued due to validity of measurement
     - aneurysmal heart disease
     - HOCM
   - ii. aortic surgery - poor LV function
     - suprarenal clamping
   - iii. severe pulmonary disease
     - pulmonary hypertensive disease
     - multiple pulmonary emboli
   - iv. states of increased ***oedemagenesis*** - pre-eclampsia, ARDS
2. optimisation of ***perfusion*** & ***oxygen delivery***, in patients unresponsive to therapy
   - i. sepsis syndrome / SIRS / MOSF
   - ii. LV dysfunction
3. ***ancillary*** capabilities
   - i. ventricular pacing
   - ii. mixed venous S_pO_2
   - iii. diagnostic categories
     - angiography in PE
     - air embolism
     - preoperative assessment of post-pneumonectomy risk
   - iv. therapeutic - regional thrombolytic therapy
4. ***research***

- ***Guidelines For Use***

1. check position with a CXR
2. never use fluid to fill balloon & inflate the balloon slowly
3. use the minimal volume to achieve a wedge trace
4. never let balloon remain wedged
5. never withdraw the catheter across the heart with the balloon inflated
6. be aware of increased risk of PA rupture in elderly

- ***Criteria for "Wedging"***

1. blood sample = pulmonary capillary blood
   - may not be fully saturated in patients with significant intrapulmonary shunt or with excessive levels of PEEP
   - now no longer recommended
2. PA phasic contour should change to LA tracing
3. mean PAoP should be < mean PAP

- ***West Zone 3 Criteria***

1. 'a' & 'v' waves visible on PAoP trace
2. mean PAoP ~ PADP (except with large 'v' waves)
3. blood freely aspirated from distal port
4. aspirated blood has a high $P_{O2} \sim P_{aO2}$

- changes from zone 3-2-1 occur with,

a. hypovolaemia
b. high PEEP > 10 cmH_2O
c. poor catheter position
d. poor patient position

- ***Recording Methods***

1. inherent underdamping renders systolic & diastolic unrelaible (Gardner Anaes.'81)
2. most circuits do not allow damping adjustment
3. unselective, time-based electrical sampling & averaging renders "mean" unreliable
   → graphic recording ***mandatory*** to eliminate respiratory artefact

- ***PA Catheter - Clinical Aspects***

a. no absolute indications

b. essentially a poor indicator of preload in severe disease
   - trends of far greater value than isolated readings
   - derived data probably of greater benefit than PAoP

c. ***no*** improvement in ***outcome*** in CCU patients

d. ***no*** improvement in outcome in severe respiratory disease

e. some suggestive evidence for improved survival,
   i. in major postoperative and severely septic patients (Shoemaker)*
   ii. perioperative MI < 3 months (Rao, El Etr)§

f. results depend upon the ***use*** of information derived

NB: *this improvement was not necessarily related to PA catheter
§this was a none peer reviewed paper, subsequently claimed benefits withdrawn

Complications

NB: complication rate similar to ***CVC catheters***,
especially complications of ***insertion***

1. principal complication = ***misuse & misinformation***
2. minor complications common
   i. ***arrhythmias*** - **VEB's**, AEB's, persistent VEB's
      - transient RBBB, 3°HB
      - SVT
   ii. haematoma
   iii. catheter thrombosis
3. major complications rare

i.	pulmonary infarction	≤ 7%
ii.	carotid puncture	~ 1-4%
iii.	infection	~ 1-2%
iv.	thrombotic endocardial vegetation	≤ 1%
v.	pneumothorax	~ 0.5%
vi.	PA rupture	~ 0.1%
vii.	valvular damage	
viii.	papillary muscle damage	

4. catheter knotting
5. bacteraemia / sepsis
6. balloon rupture

Author	Shah *et al**	Davies, Cronin	Other
Date	1984	1,982	
Patients	6,245	220	
Carotid artery puncture	1.9 %	3.6 %	
Pneumothorax	0.5 %	-	
Arrhythmias	**72 %**	**25 %**	**17-28 %**
• **VEB's**	**67 %**	**24 %**	
• AEB's	1.3 %	-	
• persistent VEB's	3.1 %		
• transient RBBB	0.05%		
• 3°HB	0.016%		
• SVT	0.5 %		
Bacteraemia/Sepsis	~ 5 %	1.4 %	0-2 %
PA rupture	0.064%		
PE/pulmonary infarct	0.064%	0.5 %	≤ 7%
Balloon rupture		0.5 %	

* Shah, Rao, El Etr Anaesthesiology, 1984 61:271-5

Pulmonary Capillary Pressure

Def'n: the effective pulmonary capillary pressure (P_C) = the dynamic pulmonary capillary hydrostatic pressure, where,

$$\mathbf{P_C = LAP + 0.4 \times (P_{mPA} - LAP)}$$ the ***Garr Equation***

- P_C is determined by,
  a. PA pressure
  b. LAP
  c. alveolar pressure
  d. PEEP - increased LAP & PAP
     - increase in P_C ~ 0.5 x PEEP

- this is the pressure responsible for ***hydrostatic pulmonary oedema***,

$$\mathbf{PAoP \sim LAP,\ but\ PAoP \ ^1\ P_C}$$
$$\mathbf{\circledR\ PAoP < P_C}$$

- where, P_C is the "dynamic" pulmonary capillary pressure
- this can be calculated upon occlusion of the PA tracing → bi-exponential decay
- extrapolating the second phase to time zero gives an ***intercept pressure***, P_i where,

$$\mathbf{P_C \sim PAoP + P_i}$$

NB: alternatively, the pressure at the ***inflexion*** point of the decay curve ~ P_C

• by these techniques it is possible to determine the predominant site of PVR in health and disease states,

1. PAP >> P_C ~ PAoP → most PVR is ***precapillary***
2. PAP > P_C >> PAoP → most PVR is ***postcapillary***

• using this technique it has been demonstrated that,

1. most of the increase in PVR with ***histamine*** is postcapillary (ie. venous)
2. with 5HT most of the increase is precapillary

PA Catheters - Misleading Information

• the primary assumption, that **PAoP ~ LVEDP**, holds true for 90-95% of "normal" subjects
• tolerance limits are ± 0-4 mmHg
• on balloon inflation, at time = 0, the systolic component is lost and PAoP ~ PADP
• the pressure then falls away ***bi-exponentially*** to approach LAP, the rate of decay depending upon,

a. diastolic time
b. pulmonary vascular resistance* **time constant* = R x C
c. pulmonary vascular compliance*

NB: the value should be taken at ***end diastole*** and ***end expiration***

- ***Potential Problems***

1. **PAoP > P_CP** up to 11 mmHg

   i. tachycardia - inadequate time for EDP to equilibrate with LAP
   ii. PA hypertension - hypoxia, hypercarbia, acidosis
   - CAL
   - 1° PAH

   → prolongation of time constant

2. **PAoP < P_CP** up to 7 mmHg
   i. RBBB - RV systole delayed, and
   - septal movement interferes with PAEDP

   ii. hypovolaemia - increase non-zone 3 area

3. **P_CP > P_VP** (or LAP)
   i. pulmonary venous disease (fibrosis, tumour, anomalies)
   ii. PEEP > 10 cmH_2O

4. **LAP > LVEDP**
   i. mitral valve disease, prosthetic valve
   ii. atrial myxoma

5. **LVEDP [1] LVEDV**
   - accuracy with which LVEDP represents LVEDV depends upon blood volume and the compliance of the LV
   - this is ***non-linear*** in normals and displaced in disease states
   - determinants of LV compliance include,

   i. LV wall thickness and diameter
   ii. fibre stiffness
   iii. pericardial pressure* *juxtacardiac pressure
   iv. intrathoracic pressure*

   - lowered compliance occurs in: - IHD, AMI
     - IHSS
     - fibrosis
     - LVH
   - raised compliance occurs in: - dilated LA or LV

6. **LAP < LVEDP** - aortic regurgitation

7. West's zone of placement

• other problems reading PA catheters are encountered with,

a.	rapid heart rates		- difficult to judge end-diastole - insufficient time for equilibrium
b.	respiratory pattern		- rapid rate, large tidal volumes - large intrathoracic pressure swings - difficult to judge end-expiration
c.	digital readouts	→	- average pressure - where mean ≠ end-diastolic pressure
d.	underdamping		- small air bubbles < 0.25 ml
e.	overdamping		- large air bubbles - narrow, long tubing - catheter blockage

■ ***Correlation - PAoP & LAP (Sibbald, Raper)***

• generally a good correlation in postsurgical patients with no respiratory disease
• the correlation is poor with,

a. high levels of PEEP

b. hypovolaemia

c. acute respiratory failure

- ***Circumstances Where PAoP ¹ LAP***

1. incorrect catheter placement
2. non-zone 3 position
3. incorrect transducer placement
4. over/under-damping
5. respiratory pressure artefact, PEEP
6. eccentric balloon inflation
7. balloon overinflation
8. obstructive airways disease (autoPEEP)
9. valvular heart disease
10. increased pericardial pressure
11. altered myocardial compliance
12. pulmonary venous obstruction

- ***Circumstances Where LAP ¹ LVEDP***

a. altered myocardial compliance
- IHD
- IHSS
- AMI
- aneurysm
- fibrosis
- LVH
- dilated LA or LV

b. mitral valve disease

c. aortic regurgitation - falsely high PAoP

- ***Circumstances Where LAP ¹ LVEDP***

• factors which influence this include,

a. LV compliance

b. RV diastolic volume (ventricular interdependence)

c. pericardial compliance

d. intrathoracic pressures

e. normal curvilinear relationship between EDP/EDV is volume dependent
* steep vs. flat portion of the curve

- ***Correlation - Reasons why LVEDP ¹ LVEDV***

  a. myocardial fibre stiffness, ***compliance***, varies

  b. myocardial wall thickness varies

  c. alterations in juxtacardiac pressures

  NB: no animal, or human studies, have shown a consistent correlation between LVED**P** & LVED**V**,

  therefore, "PAoP must be regarded as an ***unreliable*** index of LVEDV"

• Beupre *et al.*, Anaesth.'83, assessing LVEDA and PAoP,

  1. linear regression with correlation coefficient, r = 0.3, for > 77% of measurements
  2. in > 50% of measurements, the change was in the opposite direction

• Sibbald *et al.*, Chest '83, PAoP vs LVEDV by radionuclide LVEF and thermodilution CO
• linear regression essentially a scatter diagram, but multiple errors in calculation of LVEDV
• more recent studies using TOE, PAoP versus LVEDA also show poor correlation

- ***LV Compliance = LV Pressure/Volume Curve***

• decreased compliance → left shift
• increased compliance → right shift

  a. LV preload

  b. LV mass
     - LVH decreases compliance
     - chronic dilatation increases compliance

  c. myocardial fibre stiffness
     - ischaemia
     - fibrosis, scar
     - infiltration, amyloid

  d. RVEDV
     - cor pulmonale
     - increased PVR

  e. hypoxia, temperature, osmolality, HR

  f. vasopressors, vasodilators, inotropes, adrenergic blockers

• ***ventricular interdependence*** depends upon,

  a. RV size

  b. septal shift

  c. juxtacardiac pressure change
     - tamponade
     - high PEEP
     - effusion

PAoP and PEEP

$$P_C = LAP + 0.4 \times (P_{PA} - LAP)$$ **the Garr Equation**

- P_C is determined by,
  a. PA pressure
  b. LAP
  c. alveolar pressure
  d. PEEP
     - increases LAP & PAP
     - increase in $P_C \sim 0.5$ x PEEP
     - the PAoP ~ LAP which are both less than P_C
     - thus, PEEP will affect PAoP, the important factors being,
       i. the level of PEEP
       ii. lung and chest wall compliance
       iii. airways resistance → "autoPEEP"

$$\delta P_{IP} \sim \delta P_{AW} \times C_L/(C_L + C_{CW})$$

P_{IP} - interpleural pressure
P_{AW} - airways pressure
C_L - lung compliance
C_{CW} - chest wall compliance

- in the normal physiological state, C_L & C_{CW} are approximately equal, therefore,

$\delta P_{IP} \sim \frac{1}{2} \times \delta P_{AW}$ or,

$$\delta P_C \sim \delta PCWP \sim \frac{1}{2} \times \delta PEEP$$

• in pathological lungs with decreased compliance, $C_{CW} >> C_L$, thus,

$$\delta P_{IP} \sim \delta P_{AW} \times C_L/C_{CW}$$

where, $C_L/C_{CW} << 1.0$

so, $\delta P_{IP} << \delta PEEP$

or, $\mathbf{dP_C \sim dPCWP << dPEEP}$

• that is, the "wedge pressure" is relatively protected
• the reverse occurs with either highly compliant lungs, or a pathologically stiff chest wall,

→ $C_L >> C_{CW}$

thus, $\mathbf{dPc \sim dPCWP \sim dPEEP}$

PAoP and Preload

• the correlation of CVP with LVEDP is poor when,

a. EF < 40%
b. LV dyskinaesia
c. myocardial ischaemia
d. LAP > 15 mmHg
e. right heart disease

• the correlation of PAoP and LVEDP,

a. is fair in "normal" individuals ± 4 mmHg in 95%
?? ± 1 mmHg in 90%

b. is poor where,
   i. LAP > 15 mmHg
   ii. PEEP > 10 cmH_2O
   iii. tachycardia

• the correlation of PAoP and LVEDV,

a. very poor correlation in the presence of sepsis, or cardiac disease → "scatter graph"
b. relationship between LVEDV and LVEDP is ***non-linear***
c. LV compliance is abnormal in a number of disease states

- ***Causes of Increased LV Compliance***

  a. increased EDV (low EF, volume overload)
  b. dilated cardiomyopathy
  c. vasodilators(SNP, GTN, β-blockers)

- ***Causes of Decreased LV Compliance***

  a. decreased EDV - improved EF, relief from volume overload
  b. ischaemia
  c. PEEP
  d. increased RV afterload
  e. hypotensive shock - hypovolaemia, sepsis
  f. pericardial effusion
  g. positive inotropes - β_1-agonists

- ***Factors Affecting PAOP in Critically Ill (Sibbald)***

  a. CVP and RVEDV - 80%
  b. LVEDV - 10%
  c. PVR - 10%

- ***Primary Data***

  NB: individual values are of little use, ***trends*** are more useful

  a. PAoP as an indicator of oedemagenesis
     - essentially a poor indicator of preload

  b. PA pressures indicate degree of PAH

  c. P_{vO2} indicates global O_2 supply/demand

- ***Derived Data***

  a. haemodynamic variables
     - CI, LVSWI, SVR
     - qualitative information re cardiac and vascular function
     - some quantitative information with trends

  b. DO_2 & VO_2
     - rough guide to O_2 supply and utilization
     - assessment of the effect of therapy

Cardiac Output - Thermodilution

- thermodilution introduced by Fegler 1954 in anaesthetised dogs
- injection of a known volume of cold solution in RA & detection by thermister in proximal PA
- some disagreement regarding extravascular losses of "coolth"
- however, distance between injection & detection should be as short as possible

$$CO = \frac{V_1(T_B - T_I)K_1K_2}{\int_0^\infty \Delta T_B(t)dt}$$

a. V_1 = volume of injectate

b. $T_{B/I}$ = blood / injectate temperatures

c. K_1 = ***density factor***
= [specific heat . specific gravity].Injectate / [specific heat . specific gravity].Blood

d. K_2 = ***adjustment factor***
   i. catheter dead space
   ii. heat gain by injectate
   iii. injection rate (should ≤ 2-4s)

e. demoninator corresponds to area under thermodilution curve

NB: ***recirculation peak*** should be < 4% of maximum, 5-35 seconds later

- ***Errors of Measurement***

a. injectate temperature
   - theoretically the lower the better → ↑ S:N ratio
   - multiple studies have shown little advantage & room temperature OK

   i. syringe rewarming
      - ↑ 1°C → ↑ CO ~ 2.9%
      - T_I increases ~ 1°C / 13s handling, ∴ aim at delivery within 30 secs
      - ideally, should measure T_I at entry point on catheter

   ii. loss to catheter wall - most important → K_2 ~ 0.83, assuming 17% loss (catheter specific)

b. blood temperature - lower temperature → overestimation
   - rarely a problem

   i. severe hypothermia and room temperature injectate

   ii. inspiration of cold gases & decrease PA blood temperature

   iii. rapid infusion of cold fluids

c. cardiac output
   ± 0.6% at 5.0 l/min
   ± 2.0% at 4.0 l/min
   ± 4.0% at 3.0 l/min
   ± 7.5% at 2.0 l/min
   ± 20 % at 1.0 l/min

d. volume
   - the larger the better, small volumes → overestimation
   - however, larger volumes more difficult to inject as ***bolus***

e. injectate time - K_2 allows for 2-4 secs

f. timing of injection
   - variation of CO & PA temperature with respiration and mode of ventilation
   - with IPPV, CO lower during inspiration
   - variation ~ 14% → average ≥ 3 readings

g. recirculation - only if frequent calculations, repeated over < 30 secs

h. catheter wedging → underestimation
   - also seen with catheter thrombosis

i. shunts

   i. L → R - measured CO ~ RV output >> LV

   ii. R → L - falsely ***high*** CO if injectate bypasses the thermister

j. pulmonary regurgitation - low output state
   - over & under-estimations may occur

k. diathermy - increased noise

- ***Summary - Causes of Overestimation***

a.	higher injectate temperature	- room temp. - catheter wall - handling - low CO
b.	lower blood temperature	- hypothermia - infusion of cold fluids - IPPV with cold gases
c.	low injectate volume	
d.	slow injection time	
e.	respiratory cycle	- SV → inspiration - IPPV → expiration ~ 10-15%
f.	L → R shunt R → L shunt	- overestimates effective forward LV flow - overestimates CO
g.	incorrect K_1/K_2	

- ***Summary - Causes Of Underestimation***

NB: = opposite of the above, plus

a. PA catheter wedging / thrombosis

b. rapid repetitive calculations

c. RV valve regurgitation (PI/TI)

- ***Other Complications of CO Measurement = "Complications of PA Catheter"***

a.	technical	- insertion - equipment - backup
b.	infection	- catheter - injection
c.	hypothermia	
d.	thrombosis/pulmonary infarction	
e.	haemorrhage	

Haemodynamic Measurements

1.	BSA	~	$[Ht.]^{0.725} \times [Wt.]^{0.425} \times 71.48 \times 10^{-4}$	m^2
2.	CI	=	CO/BSA	l/m^2
3.	SVI	=	CI/HR	l/m^2/beat
4.	LVSWI	=	(MAP - PAoP) x SVI x 0.0136	gm.m/m^2/beat
	W	=	$dP \times dV$	
5.	SVRI	=	(MAP - CVP)/CI x 80*	dyne.sec.cm^{-5}/m^2
	R	=	V / I	
6.	PVRI	=	(MPAP - PAoP)/CI x 80*	dyne.sec.cm^{-5}/m^2
7.	CaO_2	=	$(SaO_2 \times [Hb] \times 1.34) + (P_{aO2} \times 0.003)$	ml.O_2/100ml
8.	DO_2	=	$CaO_2 \times CI \times 10$	ml.O_2/min
9.	ERO_2	=	$(CaO_2 - Cv'O_2) / CaO_2$	
10.	Q_S/Q_T	=	$(Cc'O_2 - CaO_2)/(Cc'O_2 - CvO_2)$	

NB: *__79.98__ = δ mmHg.min/l → dyne.sec.cm^{-5}

all measurements involving output should be indexed, including resistances

Normal Cardiovascular Pressures

Right			Left		
CVP RAP	diastole ~ 0-3 systole ~ 4-8	mmHg mmHg	PAoP LAP	~ 3-7	mmHg
RV	~ 22-25 / 0	mmHg	LV	~ 120 / 0	mmHg
PA	~ 22-25 / 8	mmHg	Aortic	~ 120 / 80	mmHg
PA mean	~ 13-15	mmHg	MAP	~ 90-100	mmHg
PVRI	~ 150-250	dyne/cm^5/s/m^2	SVRI	~ 800-1800	dyne/cm^5/s/m^2

BLOOD-GAS ELECTRODES

Glass pH Electrode

- the circuit consists of,

a. a capillary tube of ***pH sensitive glass*** → $\delta V \propto pH$
   - Corning 015

b. anode - Ag/AgCl surrounded by buffer within pH sensitive glass tube

c. cathode - Hg/HgCl (Calomel) within KCl buffer

d. a "salt bridge" to allow Cl^- ion flow
   - blood sample interacts with glass & the cathode via the salt bridge

e. a surrounding water jacket at 37°C

f. a ***high impedance*** amplifier to measure gradient
   - ie. the amplifier should not interact with generated potential
   - generates potential difference

$$\rightarrow \quad E = 61.5 \times \delta pH$$

Severinghaus P_{CO2} Electrode

- measurements are based on pH, due to the dissociation of carbonic acid
- the P_{CO2} is therefore related to the $[H^+]$
- the ***Severinghaus CO_2 electrode*** provides a direct measure of P_{CO2} from the change is pH
- the circuit consists of,

a. a closed cylinder of pH sensitive glass in the centre

b. 2 electrodes, 1 inside, the other outside the cylinder

c. a surrounding solution of sodium bicarbonate

d. a thin film of bicarbonate impregnated nylon mesh covering the end of the cylinder

e. a thin, CO_2 permeable membrane covering the end of the electrode

- at the end of the electrode CO_2 diffuses from the blood sample through the membrane into the nylon mesh and by the formation of carbonic acid lowers the pH of the bicarbonate solution
- this change in pH alters the δV across the glass
- pH changes such that,

$$\delta pH \propto \delta \log_{10} P_{CO2}$$

- the output of the voltmeter can be calibrated in terms of P_{CO2}
- the electrode has an accuracy ~ 1 mmHg
- the response time ~ 2-3 mins
- as for the pH electrode, the CO_2 electrode must be kept at 37°C and regularly calibrated with known concentrations of CO_2

Clark - Polarographic O_2 Electrode

- the circuit consists of,
    a. DC voltage source ~ 0.6 V
    b. an ammeter
    c. anode Ag/AgCl → $Ag + Cl^- \longrightarrow AgCl + e^-$
    d. cathode ***platinum*** → $O_2 + 4e^- + 2H_2O \longrightarrow 4(OH^-)$
    e. an electrolyte solution (KCl, ?KOH) and O_2-permeable membrane

- separated from the sample by a gas permeable membrane
- as for any resistive circuit as the voltage is increased the current will increase proportionately
- the above circuit → a ***plateau voltage*** range over which the current does not increase with increasing voltage, however does increase with an increasing P_{O2} in the cell
- O_2 is ***consumed*** in the reaction and the current produced is proportional to the sample P_{O2}

- the platinum electrode cannot be inserted directly into the blood stream as protein deposits form an affect its accuracy
- factors apart from O_2 which affect the current generated include,
    1. the age of the membrane
    2. the condition of the buffer solution
    3. temperature

    → should be calibrated prior to use
    ~ 3% accuracy at 50% O_2

- the response time is ~ 30-60 seconds, therefore not used for breath-to-breath analysis
- some specially designed units, with electronic enhancement → 0.25s response time
- unlike fuel cells they don't deteriorate when exposed to air, however, their shelf-life is limited by the life of the membrane and the buffer solution → ~ 6 months

Other Methods of Oxygen Measurement

▪ ***Oxygen Fuel Cell***

• effectively an O_2 limited gold/lead battery, consisting of,

a. an ammeter

b. a mesh ***gold cathode*** $\rightarrow$ $O_2 + 2H_2O + 4e^- \rightarrow 4OH^-$

c. a lead anode $\rightarrow$ $Pb + 2(OH^-) \rightarrow PbO + H_2O + 2e^-$

d. a compensating thermistor

e. an electrolyte solution (KCl) and O_2-permeable membrane

• thus, current flow depends upon the uptake of oxygen at the cathode
• unlike the Clarke electrode, the fuel cells requires no external power source
• however, like other batteries, the fuel cell will eventually expire

▪ ***Paramagnetic Oxygen Analysis***

• oxygen is ***paramagnetic*** and is therefore attracted into a magnetic field
• this is due to the unpaired outer shell electrons of the oxygen molecule
• most other gases, such as N_2, are weakly ***diamagnetic*** and are repelled from a magnetic field
• actually measures oxygen ***concentration***
• problems with use include,

1. they require ***calibration*** before use with 100% N_2 and 100% O_2
2. the presence of ***water vapour*** biases the result,
   therefore gases should be dried through silica gel before analysis
3. they are not well suited to continuous analysis, ie. breath-to-breath analysis
   flows > 100 ml/min through the chamber affect accuracy
4. limited ***response time*** doesn't allow breath-to-breath analysis

• their advantages include,

1. O_2 specific
2. don't wear-out

- ***Hummel Cell***

• based upon the paramagnetic principal & used in the ***Datex*** instruments
• the sample gas and an air reference are drawn into a magnetic field through a T-piece
• each sample line is connected in parallel to a sensor chamber, where the differential pressure is measured across a diaphragm
• in the resting state both sample lines are at equal pressure, however as a magnetic field is induced across the T-piece sample set, each gas (air & sample) is "held-up" in proportion to the O_2 content
• this then results in a pressure differential within the sensor chamber
• by oscillating the magnetic field the sensor diaphragm also oscillates, in proportion to the O_2 content of the sample gas
• the diaphragm effectively acting as a microphone, with the amplitude reflecting $\%O_2$

- ***PO_2 Optode***

• based on the principle of ***photoluminescence quenching***
• when light shines on luminescent material, electrons are excited to higher energy states and on their return emit light at characteristic wavelengths
• this excited electron can also return to its original energy state by interacting with an oxygen molecule, increasing the vibrational and rotational energy of the later
• for such photoluminescent quenching dyes, the amount of oxygen present can be related to the luminescent intensity by the ***Stern-Volmer equation***,

$$I_{P_{O2}} = \frac{I_0}{1+(k.P_{O2})}$$

where, I = the luminescent intensity at a P_{O2}
I_0 = the intensity in the ***absence*** of O_2
k = the ***quenching constant*** for the dye

• the advantages of this system are its simplicity and size, which allow intra-arterial insertion and measurement
• pH-sensitive dyes are also available, therefore , a three optode sensor can measure P_{O2}, P_{CO2} and pH simultaneously

PULSE OXIMETRY

• Kramer optically measured the O_2 in arteries of animals in the early 1930's
• Karl Matthes in 1936 was the first to measure O_2 from transmission of red and blue-green light through the human ear
• the term ***oximeter*** was coined by Millikan *et al*. in the 1940's
• they developed a lightweight oximeter, a smaller version of Matthes' design, which measured SpO_2 by transillumination of the earlobe using red & green filters covering Kramer's photocells
• the signal detected from the photocell under the green filter later proved to be in the IR range
• there were two technical problems with this approach,

a. there are many non-Hb light absorbers in tissue

b. the tissues contain capillary & venous blood in addition to arterial blood

• these were overcome by first measuring the absorbance of the ear while it was compressed to remove all blood
• after this bloodless "baseline" measurement the ear was heated to "arterialise" the blood
• this device was shown to accurately predict intraoperative desaturations, however, due to the technical difficulties was never adopted on mass

■ ***Development***

• in the early 1970's, the Japanese engineer Takuo Aoyagi was working on a dye dilution method for CO, using an earpiece densitometer
• he noted that the pulsatile components of the red & IR absorbances were related to the SpO_2
• his prototype, built by Nihon Khoden, was tested clinically in 1973 and the first commercial prototype was available in 1974
• however, further refinements were required and widespread use did not eventuate until the early 1980's

■ ***Nomenclature***

1. SaO_2 = 100.(O_2 content)/(O_2 capacity)
   - arterial blood saturation measured *in vitro*
   - O_2 content ≠ 1.39 x [Hb], but the amount of O_2 which can combine with reduced Hb, ***without*** removing COHb or MetHb when they are present
   - thus, at high P_{aO2} the SaO_2 = 100%, irrespective of the [COHb + MetHb]
2. HbO_2 = ***oxyhaemoglobin concentration*** (fraction or %)
   - multiwavelength spectrometers measure all Hb species as fractions or percentages of the total [Hb] = HHb + O_2Hb + COHb + MetHb
   - this has been inappropriately termed "fractional saturation"
   - SaO_2 computed from P_{O2} and pH approximates SaO_2, not HbO_2
3. SpO_2 = ***pulse oximeter saturation***

■ ***Methodology***

• 2 wavelengths of light,

1. red = 660 nm
2. IR = 910-940 nm

• the signal is divided into two components,

a. ***ac*** = pulsatile arterial blood

b. ***dc*** = tissue + capillary blood + venous blood + non-pulsatile arterial blood

NB: all pulse oximeters assume that only the pulsatile absorbance is arterial blood

• for each wavelength, the oximeter determines the ac/dc fraction, which is ***independent*** of the incident light intensity = ***pulse added absorbance***
• then the ***ratio*** (**R**) of these is calculated,

$$R = \frac{(\text{ac absorbance/dc absorbance})_{Red}}{(\text{ac absorbance/dc absorbance})_{IR}}$$

$$= A_{660nm} / A_{940nm}$$

• this value varies from,

a. SaO_2 = 100% R = 0.4 (0.3)

b. **SaO_2 = 85%** **R = 1.0**

c. SaO_2 = 0% R = 3.4 (4.87) - Severinghaus

• being a 2 wavelength device, the pulse oximeter assumes that there are only two light absorbing Hb species in arterial blood
• the photo-detector diodes of the sensor will also register ***ambient light***
• this interference is reduced by cycling the light signal from red only → infrared only → both off
• this is repeated at 480-1000 Hz in an attempt to subtract the ambient light signal, even when this is oscillating
• this allows accurate estimation of SaO_2 at arterial pulse frequencies ~ 0.5-4 Hz (30-240 bpm)
• data is averaged over several cycles

Uses of Pulse Oximetry

- ***Monitoring Oxygenation***

1. anaesthesia & recovery
2. intensive care
   - i. non-invasive monitoring of - all ventilated patients
     - during weaning
     - respiratory / cardiac failure
   - ii. risk of oxygen toxicity - neonates
     - chemotherapy, radiotherapy
     - paraquat poisoning
   - iii. avoidance of repeated AGA's
3. emergency care & transport
   - i. labour
   - ii. premature & newborn infants
   - iii. home & hospital monitoring for SIDS
   - iv. patients in remote locations - XRay, MRI
   - v. "office" procedures - dentistry, endoscopy

- ***Monitoring Circulation***

1. systolic BP & pleth waveform appearance *inflation better than deflation
2. sympathetic blockade with central neuraxis anaesthesia
3. autonomic dysfunction with valsalva manoeuvre
4. anecdotally reported uses
   - patency of the ductus arteriosus
   - level of ischaemia in PVD
   - patency of arterial grafts
   - circulation in reimplanted digits or grafts

- ***Other***

1. optimisation of home O_2 therapy
2. sleep studies
3. research

Limitations of Pulse Oximetry

1. SpO_2 ***does not*** indicate ***oxygenation*** unless [Hb] and CO are known
2. insensitive to directional changes in P_{aO2} above 80 mmHg
3. due to automatic gain, oximetry is relatively insensitive to ***perfusion***
4. ***errors*** of saturation estimation
   - i. signal to noise ratio — - vasoconstrictors
- shock or hypothermia, low perfusion pressure
- ***automatic gain***
   - ii. motion artefact — ~ **0.5-4 Hz** range
- improved by ***coupling*** with the ECG signal
   - iii. light artefact
   - iv. dyshaemoglobins
     - **COHb** indistinguishable from HbO_2, ∴ artefactually high reading
     - **MetHb** absorbance in high at both A_{660nm} & A_{940nm} → forces R → 1.0
   - v. anaemia — ≤ 15% low error with Hb < 8.0 g/dl
   - vi. intravenous dyes — - methylene blue and indocyanine green
   - vii. pigments — - pigmented races → ↑ false high readings
- nail polish ↓ transmitted light & may result in failure
   - viii. abnormal pulses
     - venous waves — - TI, reflectance operation
     - ventilation — - a large paradox may lead to searching
   - ix. probe variability errors
   - x. probe position — → the "penumbra effect"
   - xi. electrocautery — - most unit are now immune
   - xii. MRI interference — - rare, usually probe lead distorts MRI image

e. reading failure
- Freund *et al.* ~ 1.12% failure (cumulative > 30 mins) in 11,046 anaesthetics
- Gilles *et al.* ~ 1.1% incidence (2 x 15 mins) in 1,403 anaesthetics

Patient Safety

• multiple studies showing superiority of oximetry to clinical judgement in detecting desaturation
• as yet, ***no*** published paper has shown a statistically significant reduction in ***morbidity*** and ***mortality*** resulting from the use of oximetry
• major problems relating to the detection of desaturation relate to,

1. what level of desaturation is ***unacceptable***?
2. for how long is this unacceptable?
3. in whom do these limits apply?

• SpO_2 cycling repeatedly down to 30-40% has been recorded during sleep, without detectable end-organ damage, on both,

1. chronic mountain dwellers with polycythaemia
2. obese patients with obstructive sleep apnoea syndrome

• Cote *et al.* Anesth.1988 showed that at least 50% of desaturations, $SpO_2 < 75\%$ were clinically undetected in children, hence praised use of SpO_2
• however, no ***morbidity*** was documented in any patient, in either group resulting from hypoxia
• Moller *et al.*, Anesth.1991, looking prospectively at 20,802 cases in which half were monitored by SpO_2, failed to show any reduction in morbidity or mortality, except for a decreased incidence of intraoperative myocardial ischaemia (?? this would seem contradictory)

• the ASA Closed Claims Project, in reviewing 348 "preventable" deaths or injuries, came to the conclusion that "pulse oximetry....would have been efficacious in preventing injury in 138 cases."
• using the ASA data, Caplan described 14 cases of arrest under spinal anaesthesia, 12 of whom had IV sedation/opioids without SpO_2 monitoring and hypoxia was believed to contribute
• Eichorn, Anesth.1989, looked at 1,001,000 ASA I&II patients between 1976-1988 and found that,

1. 11 major anaesthesia related incidents, of which 7-8 related to inadequate O_2
2. only 1 of these occurred after the introduction of SpO_2 in mid-'85

• this paper was accompanied by an editorial by Orchin, which pointed-out that this was not statistically significant, and this, nor any other paper had yet shown a clear cost-benefit justification for the use of pulse oximetry

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NB: Severinghaus concludes, "pulse oximetry *probably* did contribute to increasing the safety of anaesthesia...however, this change may have come through the device's educational role in promoting vigilance and awareness of inadequacies in technique"

SpO_2	P_{aO2}	Clinical example
100 %	> 250 mmHg	• FiO_2 = 40%
97.5 %	100 mmHg	• arterial, young adult
96 %	80 mmHg	• arterial, elderly • venous from skin
93 %	70 mmHg	• respiratory failure
91 %	60 mmHg	• venous, exercising muscle
85 %	50 mmHg	• ***cyanosis may be visible***
75 %	40 mmHg	• mixed venous blood • central cyanosis if [Hb] = 20
72 %		• central cyanosis if [Hb] = 18
66 %		• central cyanosis if [Hb] = 15
50 %	26 mmHg	• P_{50} of HbO_2 curve
32 %	20 mmHg	• coronary sinus blood

Cytochrome aa_3 Saturation Monitoring

- this enzyme is distal in the cytochrome oxidase chain and contains copper
- when oxidised this enzyme has an absorbance peak **~ 830 nm** in the near infrared range
- as this wavelength is absorbed by both Hb & HbO_2, simultaneous estimation of these must be carried out and ***three wavelengths*** must be used
- the device for measuring this, the ***Niros scope*** = near infrared oxygen sufficiency scope
- uses powerful laser diodes with sufficient light intensity to penetrate the skull
- effectively only measures saturation in the superficial cortical layers

CAPNOGRAPHY - $ETCO_2$

- continuously monitors $ETCO_2$ by either,
    a. in line sampling
    b. aspiration sampling

- use infrared light at a wavelength ~ **4.28 nm**
- in steady state conditions $ETCO_2 \sim P_{aCO2}$ (δ = 2-8 mmHg)
- uses include,
    a. respiratory monitoring in ICU/anaesthesia
        i. ETT position
        ii. ventilation adequacy
        iii. disconnect alarm
        iv. emboli (especially air)
    b. control of P_{aCO2} (hyperventilation in head injury)
    c. monitoring of muscle paralysis during anaesthesia
    d. adequacy of cardiac massage during CPR
    e. sleep apnoea study
    f. research

- causes of a **low $ETCO_2$** include,
    a. hyperventilation
    b. hypovolaemia, hypotension
    c. pulmonary embolus - air, fat, amniotic fluid, thrombi, etc.
    d. IPPV and PEEP
    e. anaesthesia, muscle paralysis
    f. hypothyroidism
    g. hypothermia
    h. posture change from supine - upright/lateral

- causes of a **high $ETCO_2$** include,

a.	hypoventilation	- respiratory failure - narcotics
b.	increased CO_2 production	- IV HCO_3^- - trauma - sepsis, pyrexia - MH - hyperthyroidism, storm - MOSF
c.	recovery after low CO state	- post cardiac arrest - post-hypovolaemia
d.	increased V_D with fixed V_M	- anatomical, physiological - apparatus
e.	increased inspired CO_2	

GASTRIC INTRAMUCOSAL pHi

▪ ***Tonometric Method***

• pHi is obtained ***indirectly*** from,

1. measuring P_{CO2} of the ***gut lumen*** with a silicone balloon tonometer
2. HCO_3^- level of ***arterial blood***
3. subsitiution of these into the Henderson-Hasselbach equation

$$pH_i = 6.1 + \log \frac{[HCO_3^-]}{P_{CO_2} \times 0.031}$$

• this measurement is based on the ***assumptions***,

1. superficial mucosal P_{CO2} is in equilibrium with luminal contents
   - mocosal tissue presents a definite barrier to CO_2 diffusion & gradients can exist
   - differences should be small in the most superficial layers
2. tissue $[HCO_3^-]$ is in equilibrium with arterial blood
   - residual food in the stomach will stimulate acid secretion & raise the intramucosal $[HCO_3^-]$, ∴ gastric ***acid secretion*** must be inhibited for the assumption to hold
   - validation studies by Antonsson (AJP, 1990) supported correlation under conditions of low-flow, no-flow, sepsis, anaphylaxis
3. the $pK_{A'}$ for the H-H equation is the same as for plasma

• these correlate well with microprobe samples in normally perfused animals (r = 0.945)
• however, under conditions of low flow, especially no-flow, pHi ***underestimates*** the severity of tissue acidosis
• this dissociation appears to be a ***linear function*** of the rate of decline of intramucosal pH

NB: pHi provides an accurate & reproducible measure of actual pH in the most superficial layers, but not of the submucosal space

acid secretion & generation of an ***alkaline tide*** must be inhibited for the assumption that interstitial and arterial $[HCO_3^-]$ are equal

▪ ***Determinants of pHi***

• intramucosal acidosis may theoretically result from,

1. back-diffusion of acid, CO_2 or both
2. systemic metabolic acidosis
3. local tissue acidosis
   i. ↑ VO_2
   ii. reduced mucosal perfusion / DO_2

• ***back-diffusion*** of protons appears to be clinically insignificant
• intraluminal production of CO_2 is directly proportional to the amount of acid entering the duodenum and being bufferred with pancreatic HCO_3^-
• this may be reduced by,

a. aspirating acidic gastric contents

b. administration of a H_2-receptor antagonist or proton pump inhibitor

• main sources of acid in normoxic tissues,

a. CO_2 generated from ***oxidative phosphorylation*** ~ 15,000 mmol/d

b. ADP + H^+ generated from ***ATP hydrolysis*** ~ 150,000 mmol/d
   - cf. ~ 150 mmol/d excreted by the kidney (0.1%)

• under conditions of ***no-flow***, H^+ generation is ***greater*** than ATP hydrolysis, presumable due to metabolism of other high energy phosphate compounds
• these H^+ ions are then buffered, producing the intramucosal $\uparrow P_{CO2}$ seen in hypoxia
• the increased VO_2 seen in septic patients is met by an increase in DO_2

→ supply being ***demand-dependent***

• changes in ERO_2 seen in critical illness, ***do not*** appear to contribute significantly to intramucosal P_{CO2} , as pHi bears little/no relationship to ERO_2

• in animal experiments, pHi remains in normal limits as DO_2 is decreased, either by hypoperfusion or hypoxaemia, until the ***critical point*** is reached at which ***supply-dependency*** develops
• this occurs at a higher level in septic models, and ***endotoxin*** will decrease pHi in normally perfused models
• the ***Fick principal*** would dictate that mucosal P_{CO2} should rise in proportion to decreased flow, due to failure of CO_2 removal, however this is ***not*** supported by animal models
• therefore, the dominant mechanism for P_{CO2} rise is the ***buffer principal***, that CO_2 originates from buffering of H^+ ions

NB: pHi is indicative of mucosal ***oxygenation***, and an abnormally low pHi provides an index of the inadequacy of mucosal oxygenation present

• quantification of dysoxia,

1. pH-gap pHa - pHi
2. standardized pH $7.4 - \log(P_{tCO2} / P_{aCO2})$

• others have used the tonometric P_{CO2} alone

HUMIDIFICATION - IDEAL FEATURES

a. inspired gas delivered to trachea at
   - 32-36°C
   - 90-100% humidity

b. no fluctuation of set temperature & humidity with
   - time
   - high gas flows
   - gas composition

c. simple to use, service & sterilise

d. low resistance to gas flow
   - useful for SV & IPPV
   ≤ 5 cmH_2O/l/min at 50 l/min

e. low compliance
   i. neonate < 1 ml/cmH_2O
   ii. child < 3 ml/cmH_2O
   iii. adult < 5 ml/cmH_2O

f. low dead space

g. inbuilt alarms for
   - high/low temperature
   - over/underhydration

h. protection against
   - microshock (Class A)
   - scalding, overheating
   - "rain out"
   - dehydration/overhydration
   - "drowning"

i. maintenance of sterility

- ***Complications - Dangers***

a. infection

b. drowning

c. burns

d. electrocution

Nasogastric Tubes

- ***Indications***

a. decompression of the stomach
   - air, fluid, drug ingestion, food
   - bowel obstruction, ileus, pyloric stenosis, SMA syndrome, etc
   - prior to intubation
   - to assist SV in infants (removal of air)

b. drug administration - antacids, charcoal, antibiotics

c. enteral feeding - impaired swallowing/reflexes

d. diagnostic tool - ruptured aorta
   - barium studies
   - mucosal pH

e. gastric aspiration and lavage - poisoning
   - hyperthermia

- ***Complications***

a. during insertion
   - i. incorrect placement - trachea, bronchus, mediastinum
   - ii. inability to insert
   - iii. haemorrhage
   - iv. perforation - nasal mucosa, oesophagus

b. during use
   - i. ulceration - pharynx, oesophagus, stomach
   - ii. patient discomfort
   - iii. difficulty swallowing
   - iv. infection - gastric microaspiration
     - sinusitis
   - v. macroaspiration - incompetent LOS
   - vi. extragastric "therapy" - enteral feeding etc.
     - lung, mediastinum, pleural space
   - vii. metabolic - metabolic alkalosis
     - hypokalaemia
     - fluid loss

Nerve Supply - Nose

a.	anterior ethmoidal nerve	- anterior and upper septum - anterior roof - anterior parts of middle & inferior chonchae - anterolateral wall
b.	infraorbital nerve	- vestibule
c.	anterior superior dental n.	- ant. lower septum & floor - ant. lower portion of the lateral wall
d.	pterygopalantine ganglion branches	- post. 3/4 of septum, roof, floor, and lateral wall
e.	nerve of pterygoid canal	- upper & post. roof & septum
f.	olfactory nerve	- olfactory area

Septum:	anterior ethmoidal nerve	- anteriorly
	short sphenopalantine	- superoposterior
	long sphenopalantine	- posterior
Lateral Wall:	anterior ethmoidal nerve	- anteriorly
	short sphenopalantine	- upper/middle chonchae
	greater palantine	- inferior choncha
	ant. sup. dental nerve	- inf. choncha & floor

Transoesophageal Echocardiography

- ***Advantages***

1. relatively noninvasive, low risk procedure in anaesthetised patients
2. excellent image quality
3. no interference with surgical field
4. stable continuous cardiac monitoring

- ***Clinical Uses***

1. global and regional cardiac function
2. monitoring for myocardial ischaemia
3. assessment of valvular function and integrity
4. assessment of anatomical abnormalities
   i. atrial myxoma
   ii. valvular vegetations
   iii. mural thrombi
   iv. calcific disease
5. detection of embolisation - air, fat, thrombi, other
6. assessment of aortic dissection

- ***Indications***

1. optimisation of ***LV preload*** in patients at risk of decompensation
   i. severe LV or valvular dysfunction
   ii. major vascular, thoracic or other surgery
2. monitoring for ***myocardial ischaemia***
3. monitoring and assessment of valve replacement surgery
4. monitoring for VAE, or other embolisation
5. assessment of myocardial anatomy

- ***Contraindications***

1. operator inexperience
2. oesophageal disease - tumour
   - stricture, previous surgery
   - varices