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Postgraduate Topics in ANAESTHESIA

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58 Malignant Hyperthermia

DEFINITION

A pharmacogenetic myopathy, manifested in susceptible individuals as a life threatening syndrome of muscle hypercatabolism and rigor, accompanied by rapid and inexorably progressive rise in body temperature, in response to some general anaesthetics.

This syndrome is characterised as "malignant" due to its rapid progression to irreversibility despite withdrawal of the original stimulus. First described in 1960 by Denborough and Loveli.

INCIDENCE

Rare, but worldwide in occurrence. Incidence is 1/5,000 to 1/20,000.

PATHOGENESIS

Malignant hyperthermia (MH) is a myopathy that features an acute loss of intracellular control of calcium. This results in a release of free unbound ionised calcium from storage sites; there is a concomitant increase in both aerobic and anaerobic metabolism to provide more ATP to drive the calcium pumps that maintain calcium homeostasis, subsequently, there is muscle rigidity and hyperpyrexia.

Abnormalities

They are functional rather than structural.

SKELETAL MUSCLE

The histology shows central core, internal nuclei, target fibre, supercontracted fibres, variation in fibre diameter are the primary involvement.

MYOCARDIUM

It is controversial whether the myocardium is primarily involved or whether it is secondarily affected-by hyperthermia, acidosis, hyperkalaemia, and subsequent increased membrane permeability.

CENTRAL NERVOUS SYSTEM

Secondary involvement. There is coma, areflexia, unresponsiveness and fixed dilated pupils suggesting a/c cerebral oedema.

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SYMPATHETIC NERVOUS SYSTEM

Probably involved only secondarily.

BLOOD

Variation in ionic permeability, fragility, cholesterol content, deficiency of plasma pseudocholinesterase, DTC may occur following MH probably due to a release of tissue thromboplastin.

THEORIES OF MALIGNANT HYPERTHERMIA

- Abnormalities of sarcoplasmic reticulum proposed in 1977 by Endo.
- Raynodine receptor protein theory—this protein spans the gap between the terminal cisternae of sarcoplasmic reticulum and T- tubules and has been suggested as the site of defect in MH.

CLINICAL PRESENTATION

There are 2 types of clinical presentation of MH-rigid and nonrigid. The manifestation can range from classic cases to those with unusual presentation and mild symptomatology.

Muscle hypertonus or rigor appear in over 80% cases. This is preceded by an exaggerated myotonic response to suxamethonium. This myotonic response is evidenced by masseter muscle spasm (MMS), producing difficulty in tracheal intubation. MMS is now recognised as a reliable evidence of presence of acute MH.

During clinical anaesthesia, there is a surge of sympathetic activity—sudden unexplained tachycardia, tachyarrhythmias, cyanosis despite adequate ventilation hyperpyrexia and an excessively hot sodalime canister. Other features are marked skeletal muscle rigidity, etc.

Incidence of Various Clinical Signs

Tachycardia	96%	Rigidity	80%
Tachypnoea	85%	Profuse sweating	50%
Arrhythmia	70%	Altered BP	85%
Hyperpyrexia (41°C)	30%	Over heated CO_2 absorber	60%
Cyanosis	70%	Skin mottling	20%

CRUCIAL SIGNS

- Muscle rigidity after suxamethonium.
- Precipitous ↑ in end tidal CO_2.
- Unexplained hypertension and tachycardia even with deep plane of anaesthesia.
- Unexplained myoglobinurea in perioperative period.
- Unexplained increase in body temperature in perioperative period.

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LAB FINDINGS OF ACUTE MH

ABG	↓ pH	Serum	↑ Lactate
	↓ PO_2		↑ Pyruvate
	↓ PCO_2		↑ CPK/LDH
Electrolytes	↑ K^+		↑ AST/ALT
	↑ Ca^{++}		↑ Aldolase
	↑ Mg^{++}		↑ Myoglobin
	↓ Na^+		↑ Tissue thromboplastin

DIFFERENTIAL DIAGNOSIS

- Severe sepsis
- Thyrotoxicosis
- Pheochromocytoma
- Drug reaction
- Anaesthesia in small children in abnormal environment

MH TRIGGERING FACTORS

- Possession of MH gene
- Triggering agents
- Factors which sensitise the patient to the triggering agents.

Anaesthetic Agents

Safe

Tranquillizers in general (except chlorpromazine), opiates, barbiturates, etomidate, propofol, non-depolarising relaxants, amide and ester anaesthetics, epinephrine and norepinephrine, digitalis, calcium, dantrolene etc.

Unsafe

- Volatile anaesthetics—Halothane, enflurane, isoflurane, sevoflurane.
- Relaxants—Succinyl choline

Genetics

Mostly autosomal dominant.

Associated Disorders

- Duchenne dystrophy
- Periodic paralysis
- Myotonia congenita

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- Other myopathies
- King Denborough

Coincidental Association

- Sudden infant death syndrome (SIDS)
- Neuroleptic malignant syndrome
- Other diseases like lymphoma
- Osteogenesis imperfecta
- Glycogen storage disorders.

TREATMENT

- Specific
- Supportive

Specific

Dantrolene sodium 2-4 mg/kg is necessary for effective therapeutic plasma levels. It can be started with initial doses of 1 mg/kg and 0.5 mg to 1 mg/kg increments at 15 to 30 min intervals, until symptoms subside in a total dose of 10 mg/kg body weight is given. It is seldom that doses exceeding 4 mg/kg is necessary. Some advocate a repeat (Prophylactic) dose of 2.4 mg/kg after 10 to 12 hrs to prevent MH recurring in the postanaesthetic period. Dantrolene, for IV use is prepared as a highly alkaline solution in mannitol (3 mg per 20 mg vial).

Other drugs—Ca^{2+} channel blockers, procaine and procainamide

Supportive Therapy

Discontinuation of all anaesthetic agents and hyperventilation with 100% oxygen.

- Correction of metabolic acidosis to a pH of 7.2 to 7.3
- If possible, get another machine with a new circuit.
- Aggressive cooling.
- Treat hyperkalaemia.
- Treat tachyarrhythmias.
- Monitor urine output and induce diuresis if oliguria present.
- Provide calories with 20% or 50% dextrose.
- Monitor the patient—HR, BP, EKG, temperature, pulse oximetry, end tidal CO_2 , ABG.
- Coagulation profile—Get FFP for infusion if doubt of DIC.

The patient must be closely monitored and sedated for 24 hours. Dantrolene should be administered for at least 3 days after successful treatment of MH.

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PREANAESTHETIC DIAGNOSIS

This is essential for prevention of the syndrome, in susceptible individuals. Screening should also include near relatives.

Tests for diagnosis: These include blood tests, electrophysiological tests, biochemical tests, phosphorous—31 NMR spectroscopy, muscle histology, contracture test etc.

Contracture test: The most reliable test which is the '*in vitro* contracture test' (IVCT) with halothane and caffeine. The tests employed are:

- Caffeine contracture test
- Halothane contracture test
- Caffeine-Halothane contracture test
- Raynodine contracture test.

ANAESTHESIA IN MH PATIENTS

Four simple guidelines:

- Avoidance of triggering agents.
- Suppression of patients trigger sensitivity.
- Meticulous monitoring of patient.
- Immediate availability of dantrolene.

Avoidance of Triggering Agents

- Local/regional technique used
- GA-avoid all potent volatile inhalational agents and suxamethonium.

Suppression of patient's trigger sensitivity: Proper preanaesthetic control of apprehension and anxiety.

Dantrolene prophylaxis: (Controversial); should be administered IV in a dose of 2.4 mg/kg with induction of anaesthesia.

Precautions

- The patient should carry a warning card for identification of this syndrome.
- The family, and other relatives should be investigated for this syndrome.
- The individual with a history of MH in his family or other relatives should be treated as susceptible to MH until proved otherwise.

BIBLIOGRAPHY

1. Hopkins PM. Malignant Hyperthermia. Br J of Anaesthesia, 2000.
2. Incidence of Malignant Hypothermia in Denmark Anesth Analg, 1985.
3. Robert K Stoelting. Anaesthesia and Co-existing Disease.
4. Ronald D Miller. Anaesthesia.
5. Wylie. A Practice of Anesthesia (7th edn).

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59 Awareness During Anaesthesia

Unconsciousness is an integral part of anaesthesia and hence the patient becoming aware while under anaesthesia is highly unjustifiable. But this problem is as old as the speciality itself and more reports are coming out now a days. This may be either due to increase in reporting or due to the practice of 'Balanced Anaesthesia', whereby a conscious patient can be kept still by muscle paralysis.

George Crile was one of the earliest persons to report about awareness (1908) under N_2O anaesthesia. It was Winterbottom who reported the first case after- the introduction of relaxants. The first systematic investigation into the problem was by Hutchinson (1961).

Awareness is defined as the spontaneous recall of events occurring under GA. The term spontaneous is important, since some auditory inputs occur even in sufficiently deep anaesthesia and some meaningful images are retained. These can be brought out at a deliberate session of hypnosis later. This 'amnestic awareness' had been investigated by Levinson, Blacher and Benette separately. The general consensus is that critical news are registered more, there is no psychological trauma, but still it is dangerously close to awareness. A stimulus may evoke a response or be registered in explicit memory or only in implicit memory with deepening anaesthesia. Thus there are 3 types of true awareness:

- Conscious awareness without amnesia response to verbal command present but no memory afterwards.
- Conscious awareness with amnesia.
- Subconscious awareness with amnesia: No response to verbal command, but retention of verbal information can still occur.

Dreams are another entity which may be confused with awareness. Patients may report them from memory which may be even postoperative. But some hold the view that; awareness provokes them. Hallucination also may be misinterpreted.

Incidence reported from various studies is around 2% in general and 2-17% in obstetrics. Certain difficulties are met with in accurate estimation. A large group is needed since the incidence is very low. Deliberately subjecting the patient to awareness for research is unethical. The assessment is subjective. Many patients do not report spontaneously and clinical assessment depends on the experience of anaesthetist. Prospective studies may be manipulated. A systematic study by Hill et al demonstrated a fall in incidence over last 30 years from 1.5% to 0.2%. More than mere unpleasantness, awareness leads to considerable psychiatric morbidity. Wakefulness in a paralysed patients can lead to the syndrome of traumatic neurosis. Repetitive nightmares, irritability, anxiety, preoccupations with death and suicidal tendencies make their life miserable. There are also intra-

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operative problems, since there are haemodynamic, sudomotor, and hormonal responses to noxious stimuli which are harmful. Thus the practice of keeping the patient 'light' to avoid certain damages may be more deleterious.

Predisposing factors: Faulty techniques are the most important cause of awareness though the situation may be unpredictable and in many situations unavoidable. Drug requirement vary considerably according to age, obesity, addictions, previous exposure, systemic disease, etc. Psychiatric illness and certain personality makeups predisposes to awareness. Still some technique are particularly associated with awareness. The N_2O-O_2 anaesthesia following TPS + SC (the classical Liverpool technique) is reported to have higher incidence especially with lower proportion of N_2O and absence of volatile agents. Inadequate premedication, switching on N_2O late and putting off early, keeping O_2 flush on, early reversal all lead to awareness. Some ventilators contribute by leaks and entraining air when gas flows are inadequate. A sudden powerful surgical stimulus may provoke awareness at sufficient depth.

Detection of awareness require utmost vigilance as monitoring equipments are not fool proof. Symptoms of ANS stimulations like tachycardia, HT, pupillary dilations, increased secretions, tear and sweating etc. may point to awareness. Movements of limbs, eyelids and swallowing are also pointers. EEC and evoked potential monitoring may help detection of awareness, but are generally unavailable for clinical use. Attempts to process EEC simultaneously has lead to 'cerebral function monitor' which is the best one available at present. Development of power spectrum analysis offers the best hope for future. In isolated forearm technique (Turnstall) tourniquet applied to one U'L. prevents its paralysis, enabling the patient to indicate awareness, if explained to previously. EMG of frontalis muscle is possible even at clinical neuromuscular blockade.

Prevention of awareness involves:

- Adequate preparation of patient
- Check apparatus before anaesthesia
- Faultless technique and usage of certain adjuvants
- Preoperative discussion with the patient explaining the possibility of dreams and warning about awareness in special situations (wake up test, obstetrics) goes a long way in decreasing morbidity
- Premedication with lorazepam, hyoscine and adequate narcotics, etc. are helpful
- 'Proper' dose of induction agents rather than 'sleep' doses. Ketamine is reported to be a better induction agent in this respect. Using N_2O in adequate proportion and duration, volatile agents judiciously, IV agents in increments, etc. all contribute
- Do not rely entirely on G-O-R technique. Relaxants are better given as infusion
- If intubation is found difficult, add incremental anaesthetic
- At end of surgery maintain N_2O till relaxants are reversed. In a reported case of awareness sympathetic explanation by the anaesthetist and timely psychiatric intervention are important.

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Special Situations

- *Obstetrics:* The practice of keeping the patient under light anaesthesia till delivery to avoid neonatal depression leads to decreased uteroplacental blood flow due to the sympathetic stimulation caused by awareness. From induction till delivery a low dose volatile agent (e.g. halothane 0.5%) can help prevent awareness; this does not cause uterine relaxation and haemorrhage.
- *B' Scopy:* Apnoea oxygenation and jet ventilation are associated with high incidence
- *Paediatrics:* It is a faulty concept that small babies do not require much anaesthetics
- *Total IV anaesthesia:* Tailored doses should be given and maintained carefully - monitoring anaesthetic depth is not often possible with total intravenous anaesthesia
- During one lung anaesthesia because of $\uparrow FiO_2$.

In conclusion: In this era of Medical Litigations, awareness during anaesthesia should be avoided at all cost. But it has not only legal sequelae but medical and ethical implications also. The trend towards muscle relaxation and light GA has increased the problems. At the same time a patient should not be denied the benefits of light GA, just for the fear of awareness. A meticulous technique and careful monitoring are all that is needed.

Awareness with analgesia is regrettable, and with pain is unforgivable.

PRST score system for systemic evaluation of the autonomic signs of light anaesthesia.

Index	*Condition*			*Score*
1. Systolic pressure (mmHg)	< Control	+	15	0
	< Control	+	30	1
	> Control	+	30	2
2. Heart rate (Beats/min)	< Control	+	15	0
	< Control	+	15	1
	> Control	+	30	2
3. Sweating	Nil			0
	Skin moist to touch			1
	Visible beads of sweat			2
4. Tears	No excess of tears in open eye			0
	Excess of tears in open eye			1
	Tears overflow in open eye			2

BIBLIOGRAPHY

1. Awareness during anesthesia, what should the patient be told—Anaesthesia, 1990.
2. JF Nunn, JE Utting, BR Brown. Depth of Anaesthesia and Awareness - in General Anaesthesia.
3. Recall of surgery for major trauma—Anaesthesiology, 1984.
4. Recent Advances in Anaesthesia, Analgesia: Vols 14 and 18.
5. Ronald D Miller. Anaesthesia (3rd edn).

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60 Monitoring of Intracranial Pressure

Intracranial pressure (ICP) is the pressure within the cranium and reflects the CSF pressure. The source of this pressure is the secretory pressure of the choroid plexus. It has to be sufficient to overcome the flow resistance of the microtubules of the arachnoid villi. It is closely related to the venous pressure and oscillates with the arterial pulse and with respiration. Falls with inspiration and rises with expiration.

Normal intraventricular ICP = 5-15 mmHg

ICP greater than 20 mmHg is an indication for intracranial hypotensive therapy.

Factors which increase ICP

- *Physiological*
  - Coughing
  - Sneezing
  - Straining
- *Pathological*
  - Pressure from outside - bony tumour or craniostenosis
  - Presence of a space - occupying lesion neoplasm, abscess, haematoma
  - Hydrocephalus
  - Venous obstruction
  - Arterial dilatation secondary to hypercarbia
  - Cerebral oedema
    i. Inflammation
    ii. Neoplasm
    iii. Trauma
    iv. Hypoxia
    v. Venous obstruction
  - Head-down position.

Factors which decrease ICP

- Hypovolaemia
- Following removal of an intracranial space occupying lesion.
- Head up position -Raising the head 20 cm above horizontal reduces ICP by 20 cm H_2O.

Importance

- Cerebral perfusion pressure CPP = Mean arterial pressure (MAP) – intracranial pressure (ICP). Any rise in ICP causes a reduction in the CPP. So careful monitoring and prompt treatment of

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intracranial hypertension is the most important factor in the reduction of mortality following head trauma.

- Control of ICP prevents pressure-gradients between various compartments of intracranial cavity and prevents shifts of brain structures (Brain herniation) (Fig. 60.1).

Monitoring of ICP began way back in 1951.

Lundberg's classic work demonstrated that there are 3 waves in ICP tracing:

- **A waves** (Fig. 60.2) or plateau waves are recurring increases in intracranial pressure to values of 50 to 100 mmHg lasting 5 to 20 min which usually arise with severe decompensation of ICP control mechanism. Seen only when base line ICP is more than 20 mmHg. At 60 mmHg headache, restlessness incontinence, non purposive movements, etc. appear. At 80 mmHg loss of consciousness decerebration, respiratory arrest, etc. occur.
- **B waves** (Fig. 60.3) occur at a rate of 1 or 2 per min, are of small amplitude and may be precursors of plateau waves. Warns against impending intracranial hypertension and impairment of intracranial compliance. They were initially thought to be related to Cheyne-Stokes respiration, but can occur in patients on controlled ventilation. They are particularly prominent during REM sleep, and may occur in normal people.
- **C waves** (Fig. 60.4) occur more frequently (6/min) and are of less amplitude and of little clinical significance except that they only occur in the presence of brain pathology and imply an unstable control of cerebral blood flow.

  There are waves which do not fall into any of the categories (Fig. 60.4) and it may be that the recognized errors are not distinct entities but merely points in a spectrum.



Fig. 60.1: Brain herniations: 1. Cingulate, 2. Temporal (uncal), 3. Cerebellar, 4. Transcalvarial (post-traumatic or postoperative)

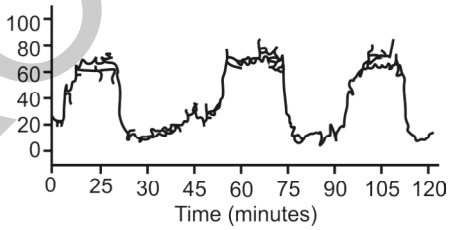

Fig. 60.2: ICP tracing—A waves

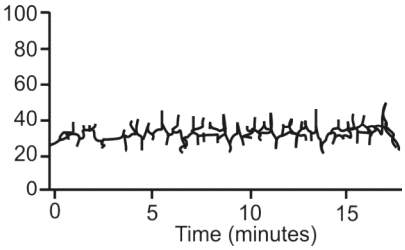

Fig. 60.3: ICP tracing—B waves

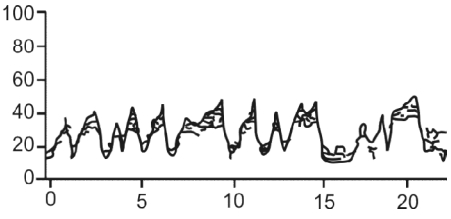

Fig. 60.4: ICP tracing—C waves

THE INTRACRANIAL PRESSURE/VOLUME CURVE

The intracranial pressure volume curve shows that as intracranial volume increases there is very little change in pressure during the phase of compensation (Fig. 60.5). With

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further increase in volume pressure begin to rise forming a 'knee'. Still further increase in volume causes marked rise in pressure during the phase of decompensation.

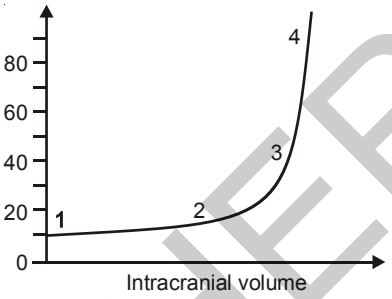

Fig. 60.5: ICP—volume (level y-axis ICP torr)

INDICATIONS FOR ICP MONITORING

- Head injury -When Glasgow Coma Scale is less than 8.
- Hepatic encephalopathy
- Reye's syndrome
- Anoxic encephalopathy
- Large intracranial space-occupying lesion
- Intraoperatively in cases where the ICP is deemed very high and likely to increase further.

USES OF ICP MONITORING

- *Diagnostic:*
  - Early detection of cerebral oedema or other causes of raised ICP.
  - Confirms that neurological deterioration is due to rising ICP and prevents inappropriate therapy.
  - Suggests the need for repeat CT scan in a patient with head injury.
- *Prognostic:* In patients with head injury.

TECHNIQUES OF ICP MONITORING

Intraventricular Catheter

The catheter is placed inside the ventricles through a twist drill hole and is exteriorised through the skin and connected to a fluid coupled system kept on patient's head or at bedside.

Advantages

- High fidelity recording
- Compliance studies possible
  Ventricular pressure response more than 4 mmHg per ml is always associated with a mass lesion.
- Permits emergency tapping of CSF for acute rise in ICP
- Cheap and simple
- Recalibration can be possible at any time.

Disadvantages

- High infection rate
- Intracerebral haemorrhage
- Difficult to insert when ventricles are small or collapsed.
- May not be suitable in head injury
- Zeroed with change in position of the head.

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Fluid Filled Surface Monitoring Devices

Balloon Devices

Fluid-filled balloons are placed subdurally or epidurally and connected to external fluid filled system.

Disdvantages

- Small balloons are too sensitive to changes in internal volume
- Large balloons are difficult to introduce
- Rupture of balloon exposes brain to the contents of the balloon.

Hollow Screw Devices

Here a hollow screw is placed through a twist drill hole and this utilises the arachnoid as the sensing membrane. It is then connected to a fluid filled system.

Advantages

- High fidelity traces
- Cheap, simple
- Recalibration possible
- Avoids cerebral trauma.

Disadvantages

- High infection rate
- Can underestimate ICP monitoring
- Produces artefact on CT scan
- Fluid drainage not possible

Cup Catheter

This is a hollow ribbon-shaped device with a cup on the flat surface of the ribbon at its end. The cup utilises the arachnoid as its sensing membrane.

Advantages

- It can be brought out through a scalp incision remote from the area of opening in the skull. So chance of infection is reduced.
- Can be inserted through a craniotomy.

Nonfluid Coupled Devices for Surface Monitoring of ICP

Mechanical (ICP Pressure Switch)

A small balloon containing electrical contacts fastened to its inner walls is introduced into the subdural or intradural space. When the balloon is fastened because of raised ICP the electrical contacts remain in contact, thereby completing a servo circuit. Servo circuit powers an infusion which pressurises the inside of the balloon up to a point which separates the electrodes and breaks the circuit. The applied pressure at this stage represents the ICP.

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