雅麗氏何妙齡那打素醫院
Alice Ho Miu Ling Nethersole Hospital
caring since 1887

A well-known but rare event

Speaker: Dr. CK Leung, Resident (AHNH ICU)
Supervisor: Dr. SO So, Consultant (AHNH ICU)

Case History

- F/20 years old
- Good past health
- Diagnosed of right cerebellar hemangioblastoma
- Angiographic assisted embolization in Baptist Hospital done in 9-2012
- Cx with peri-procedural SAH and IVH
- Proceeded to crainotomy with tumor excision

Case History

- Because of expected delayed neurological recovery and financial reasons
- Patient was transfer to PWH ICU
- Poor neurological recovery with absent corneal, gag or cough reflexes
- Pupils 3mm sluggish response
- Some spontaneous triggering but entirely ventilator dependence

Case History

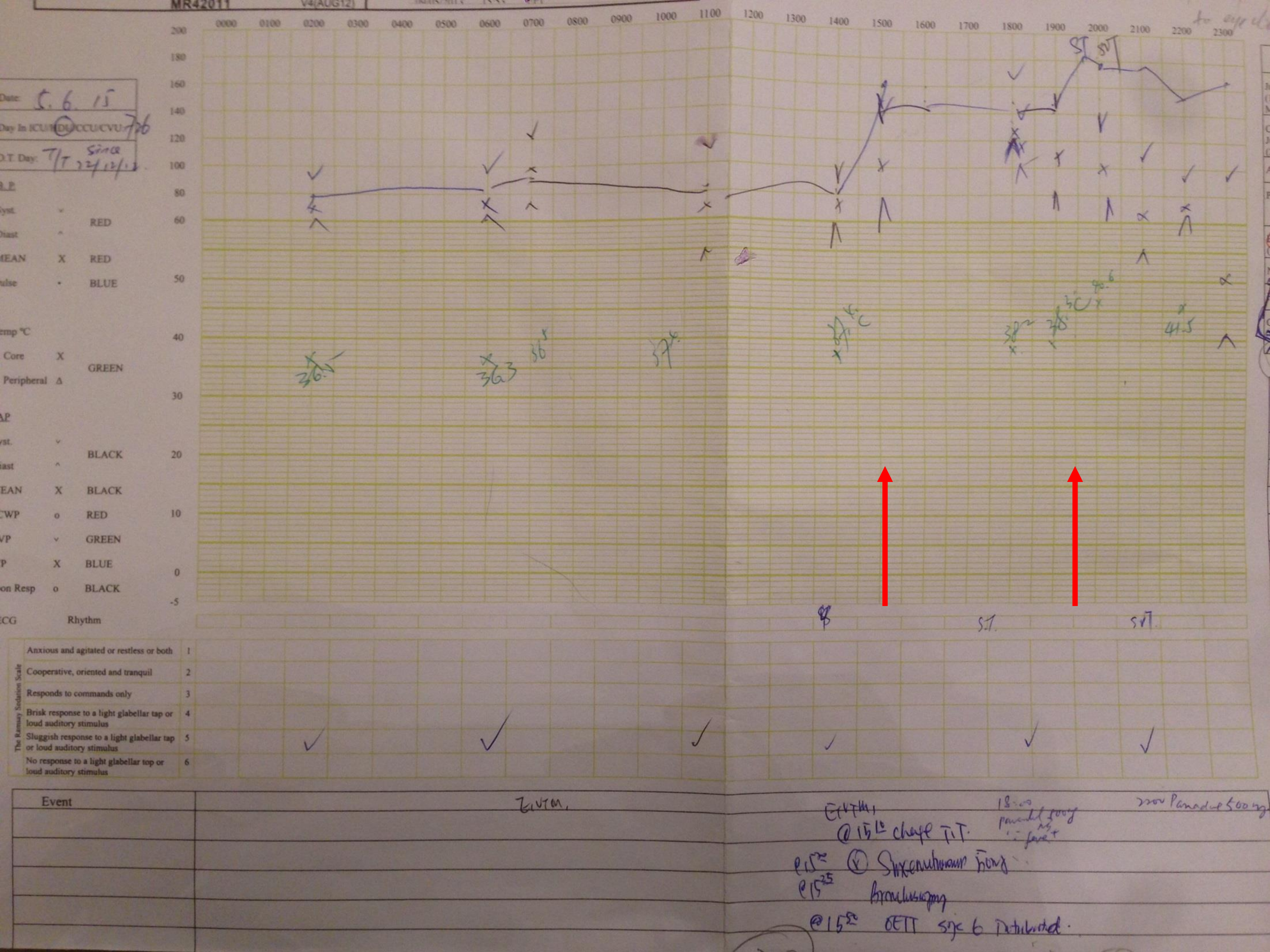
- Trachaeostomized and transferred to AHNH HDU for long-term care since 5/2013
- Despite repeated interview, family still had unrealistic expectation about pt's clinical recovery and remained demanding at times
- Colonization of high resistance bacteria in multiple sites

Case history

- On 6/6/2015, scheduled change of tracheostomy tube
- However, failed insertion of new tube
- Decided to convert to oral endotracheal intubation
- Difficult airway
  - Small mouth/chin, stiff jaw
  - Copious amount of oral secretion
- Failed for a few times with DL/glidescope
- Finally successful with IV suxamethonium 50mg given for facilitation

Case history

- A few hours later
- Noted high fever/shock with SVT
- Given panadol/noradrenaline
- Clinically suspected to have sepsis at that juncture
- Septic workup was repeated
- CXR: mild left lung hazziness
- Empirically put on tazocin

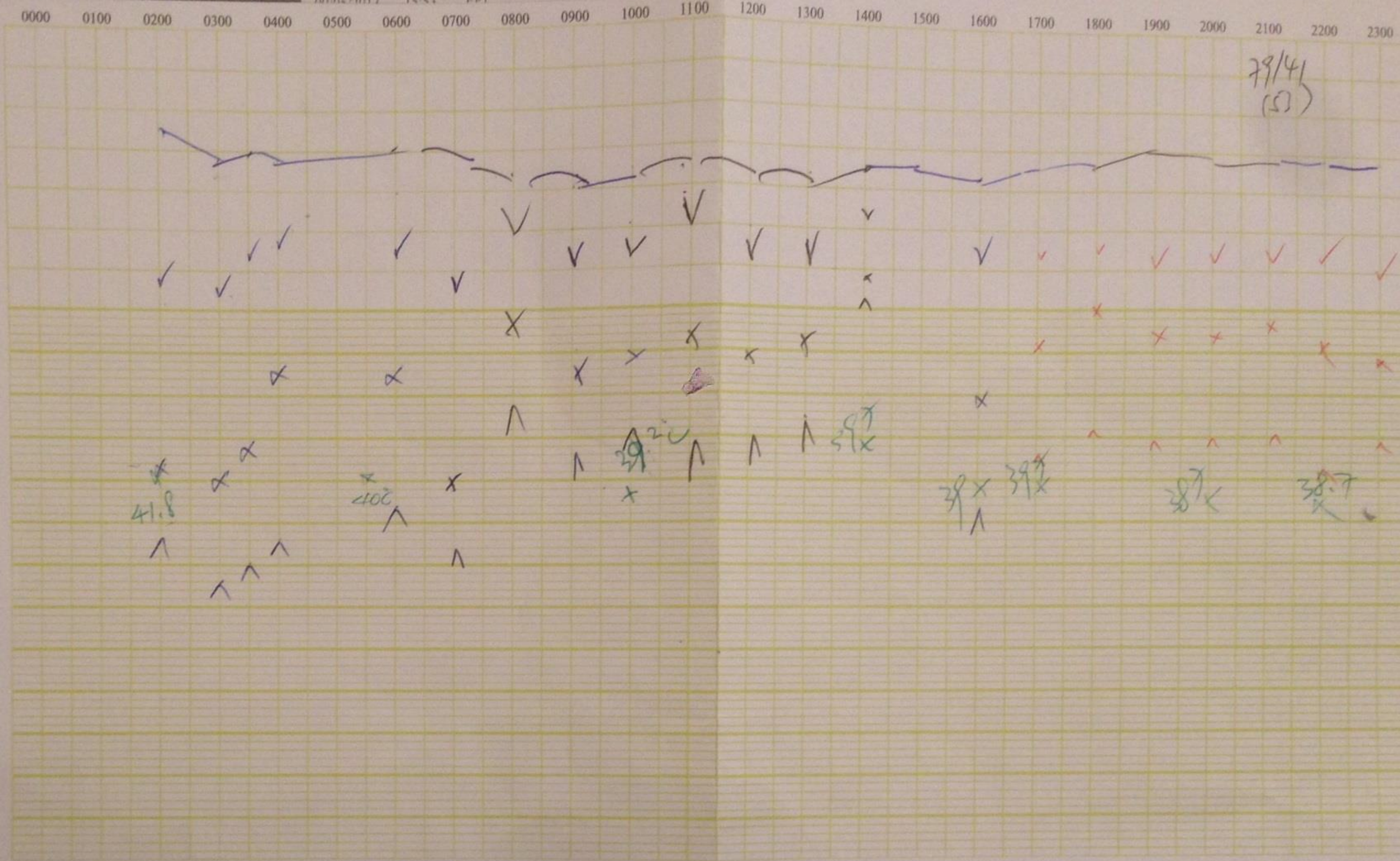

MR42011
V4(AUG12)
0000 0100 0200 0300 0400 0500 0600 0700 0800 0900 1000 1100 1200 1300 1400 1500 1600 1700 1800 1900 2000 2100 2200 2300
Date: 5.6.15
Day In ICU/HDU/CCU/CVU: 7/36
O.T. Day: T/T since 22/12/13
B.P.
Syst. ˅ RED
Diast ˄
MEAN X RED
Pulse • BLUE
Temp °C
Core X GREEN
Peripheral Δ
AP
Syst. ˅ BLACK
Diast ˄
MEAN X BLACK
PCWP o RED
CVP ˅ GREEN
IAP X BLUE
Spon Resp o BLACK
ECG Rhythm
ST
SVT
The Ramsay Sedation Scale
Anxious and agitated or restless or both 1
Cooperative, oriented and tranquil 2
Responds to commands only 3
Brisk response to a light glabellar tap or loud auditory stimulus 4
Sluggish response to a light glabellar tap or loud auditory stimulus 5
No response to a light glabellar tap or loud auditory stimulus 6
Event
@15[10] change T/T
@15[20] Suxamethonium
@15[25] Bronchoscopy
@15[50] OETT size 6 Intubated
18:00 paracetamol 500mg
2200 Panadol 500mg

Date: 6/6/2015

Day In ICU/HDU/CCU/CVU: 0 7/7

O.T. Day: 7/7 Since 27/7/2012

Parameter	Symbol	Colour
B.P.		
Syst.	v	RED
Diast	^	
MEAN	X	RED
Pulse	•	BLUE
Temp °C		
Core	X	GREEN
Peripheral	Δ	
PAP		
Syst.	v	BLACK
Diast	^	
MEAN	X	BLACK
PCWP	o	RED
CVP	v	GREEN
ICP	X	BLUE
Spon Resp	o	BLACK



ECG Rhythm: ST (0200), ST (0600), ST (0700), ST (1000), ST (1400), ST (1800), ST (2100)

The Ramsay Sedation Scale

Description	Score
Anxious and agitated or restless or both	1
Cooperative, oriented and tranquil	2
Responds to commands only	3
Brisk response to a light glabellar tap or loud auditory stimulus	4
Sluggish response to a light glabellar tap or loud auditory stimulus	5 (✓ 0200, 0600, 0700, 1000)
No response to a light glabellar tap or loud auditory stimulus	6 (✓ 1400, 1800, 2100)

Event

0200 Panadol 500mg

0600 Panadol 500mg

E4VTM1

10h Na. Panadol 500mg

1340 Syrup KCl 2g NG

E1VTM1

1400 Syr. Panadol 500mg

ⓥ D5 40ml ...x 2 ...

D5 5ml ...x1

Syr. Panadol 500mg

22h Syr. Panadol 500mg

E1VTM1

2315: KCl 20mmol in 100ml H2O

2315: Meropenem 1g

Case history

- However, condition progressively deteriorated
- Developed MOF with AKI/DIC
- Mixed metabolic and respiratory acidosis
  - pH 7.11
  - pCO2 7.6
  - HCO3 17.6/BE -12

Case history

- Family was interviewed with poor prognosis, high chance of fatal outcome discussed
- Still insisted on aggressive Tx
- Transfer to ICU for further supportive care and CRRT
  - ? Underlying severe sepsis with MODS


500
500

Progress in ICU

- Clinically evidence of rhabdomyolysis
  - CK up > 600,000
  - Urine x myoglobin +ve
  - PO4 > 5
  - CRP <16 only
  - Clinically no suspicious drugs taken
  - No evidence of soft tissue infection/compartment syndrome
  - Sepsis workup shown no pathogen identified

Emergency Laboratory

Doctor: LUN, Chung Tat

Clinical Details: sepsis

E

Collect Date :	08/06/15	08/06/15	08/06/15	08/06/15	09/06/15		
Collect Time :	05:29	09:10	14:50	23:31	05:36		
Arrive Date :	08/06/15	08/06/15	08/06/15	08/06/15	09/06/15		
Arrive Time :	05:58	10:07	15:14	23:38	06:24		
Request No. :	E7660571	C3064597	C3070624	E7662621	E7663536	Reference	
Urgency :	URGENT	URGENT	URGENT	URGENT	URGENT	Range	Units
Plasma							
Sodium	150 *	147 *	147 *	141	142	137-144	mmol/L
Potassium	8.0 *	7.9 *	7.4 *	4.6	3.8	3.5-5.0	mmol/L
Urea	13.7 *	14.9 *	14.3 *	9.5 *	8.1 *	2.6 - 6.6$	mmol/L
Creatinine	437 *	461 *	412 *	253 *	206 *	49 - 83$	umol/L
Total Protein	57 *	59 *			50 *	65 - 82$	g/L
Albumin	24 *	26 *	25 *	26 *	21 *	35 - 52	g/L
Globulin	33	33			29		g/L
Total Bili.	21 *	20 *			36 *	< 17$	umol/L
Alk Phos	47	51		57	55	33 - 84$	IU/L
ALT	3611 *	3934 *			2682 *	< 55$	IU/L
Calcium	1.21 *		1.33 *	1.75 *	1.73 *	2.15 - 2.55	mmol/L
ALB Adj. Calcium	1.44		1.55	1.95			
Phosphate	5.01 *			2.48 *	1.95 *	0.72 - 1.43$	mmol/L
Creatine Kinase		348856 *	417996 *	605172 *	624252 *	42 - 186$	IU/L
LDH (IFCC)		11943 *	13170 *	13289 *	10864 *	103 - 199$	IU/L

Comment:

15E7660571 WATCH OUT for clinical signs of hyperkalaemia. CONSIDER POSSIBLE ARTEFACTUAL CAUSES: haemolysis, fluoride / EDTA / citrate contamination, storing sample overnight or in refrigerator. Please correlate clinically and send another sample if necessary.

Test repeated

Dx: suspected Malignant hyperthermia due to succinylcholine

Progress in ICU

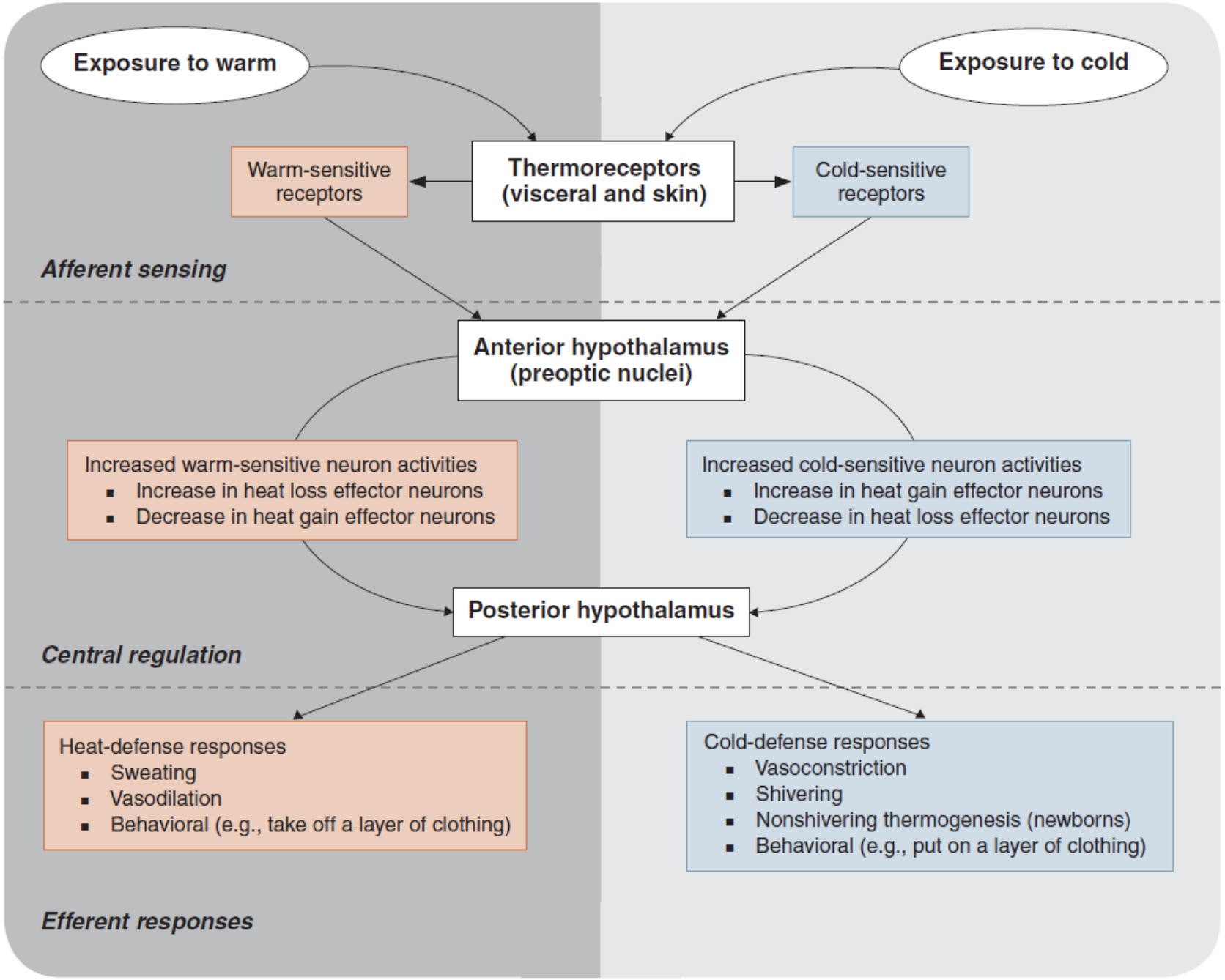
- Supportive care, external cooling
- CPK gradually came down, intermittent HD for established oliguric renal failure
- Concerning patient's past anaesthetic Hx
  - Failed to trace back those private hospital, but probably uneventful
- No known adverse event or MH within patient's family members

Discussion

Malignant Hyperthermia (MH)

Discussion

- Disorder in thermoregulation is common in ICU
- Prevalence ranges from 26% to 70%
- IDSA defined fever as core body temperature >=38.3 ◦C
- Classified as fever v.s. hyperthermia


Exposure to warm
Exposure to cold
Warm-sensitive receptors
Thermoreceptors (visceral and skin)
Cold-sensitive receptors
Afferent sensing
Anterior hypothalamus (preoptic nuclei)
Increased warm-sensitive neuron activities
Increase in heat loss effector neurons
Decrease in heat gain effector neurons
Increased cold-sensitive neuron activities
Increase in heat gain effector neurons
Decrease in heat loss effector neurons
Posterior hypothalamus
Central regulation
Heat-defense responses
Sweating
Vasodilation
Behavioral (e.g., take off a layer of clothing)
Cold-defense responses
Vasoconstriction
Shivering
Nonshivering thermogenesis (newborns)
Behavioral (e.g., put on a layer of clothing)
Efferent responses

Classifications

- Fever
  - Infectious cause
  - Pyrogens (bacterial lipopolysaccharide, TNF, IL-1)
  - ↑ prostaglandin E2
  - ↑ set-point in anterior hypothalamus
  - ↑heat production and conservaation

Classifications

- Hyperthermia
  - Non-infectious
  - Uncontrolled heat production
  - Impaired heat dissipation
  - No adjustment of the hypothalamic set-point

Epidemiology

- Infectious and non-infectious are equally represented
- Associated with young age/male/septic shock/trauma/emergent surgery/neurocritical illness
- Prolonged (>5 days) and high fever (>=39.3°C) likely infectious cause
- Surgical cases, usually happened at post-op Day 1

Circiumaru B , Baldock G , Cohen J
A prospective study of fever in the intensive care unit
Intensive Care Med . 1999 ; 25 (7): 668 - 673 .

Barie PS , Hydo LJ , Eachempati SR
Causes and consequences of fever complicating critical surgical illness
Surg Infect (Larchmt) . 2004 ; 5 (2): 145 - 159

Epidemiology

- Fever associated with increased ICU length of stay
- Prolonged fever/high fever associated with significant increased risk of death

Laupland KB , Shahpori R , Kirkpatrick AW , Ross T , Gregson DB , Stelfox HT
Occurrence and outcome of fever in critically ill adults
Crit Care Med . 2008 ; 36 (5): 1531 – 1535

Laupland KB , Zahar JR , Adrie C , et al
Determinants of temperature abnormalities and infl uence on outcome of critical illness
Crit Care Med . 2012 ; 40 (1): 145 - 151

Table 1—*Common Causes of Persistent Fever in the ICU: A Head-to-Toe Approach to Differential Diagnosis*

Site	Infectious	Noninfectious
Head and neck	Meningitis	Cerebrovascular accident
	Otitis media	Seizure disorder
	Sinusitis	Traumatic brain injury
	CVC-related blood stream infection	
Chest	Infective endocarditis	Myocardial infarction
	Ventilator-associated tracheobronchitis	Pericarditis
	Ventilator-associated pneumonia	Pulmonary embolism
	Empyema	ARDS
Abdomen and pelvis	Intraabdominal infections (eg, SBP, abscesses)	Pancreatitis
	Clostridium difficile infection	Acalculous cholecystitis
	Pyelonephritis	Ischemic colitis
	Catheter-related UTI	
	Perineal or perianal abscess	
Extremities	Femoral line/PICC-related blood stream infection	Gout
	Septic arthritis	DVT
Skin and back	Cellulitis	Drug eruptions
	Infected pressure ulcer	
	Surgical site infection	
Miscellaneous	…	Drugs
		Transfusion reactions
		Endocrine disorders (eg, thyrotoxicosis, adrenal insufficiency)
		Malignancy
		Inflammatory disorders (eg, SLE)

CVC = central venous catheter; PICC = peripherally inserted central catheter; SBP = spontaneous bacterial peritonitis; SLE = systemic lupus erythematosus; UTI = urinary tract infection.

Hyperthermic syndrome

- Can clinically mimic fever
  - Enviromental heat-related illness
  - Malignant hyperthermia (MH)
  - Serotonin syndrome
  - Neuroleptic malignant syndrome (NMS)
  - Recreational drug use (sympathomimetic drugs)
  - Withdrawal (alcohol, benzodiazepines, opiates)

Table 1. Common features of hyperthermia syndromes

	Adrenergic Fever	Anticholinergic Fever	Antidopaminergic Fever (NMS)	Serotonin Syndrome	Malignant Hyperthermia
Receptor involvement	α, β, sometimes serotonin	Cholinergic	Dopamine	Serotonin	Ryanodine 1
Onset	Variable	Variable	Variable-commonly days after exposure	Variable, typically minutes to hours after exposure(s)	Immediate to hours after initiation of anesthesia
Hyperthermia	Yes	Yes	Yes	Yes	Yes
Mental status changes	Variable	Yes	Yes	Yes	N/A, usually occurs while patient is anesthetized
Muscular	Agitation	Tremor, agitation	Progressive generalized rigidity	Akathisia, clonus, rigidity > lower extremities, hyperreflexia	Fulminant muscle rigidity
Autonomic instability	Yes	Tachycardia	Yes	Yes	Tachycardia
Common offending agents	Amphetamines, MDMA, cocaine, MAOIs, theophylline, thyroxine	Neuroleptics, antispamodics, antihistamines, anti-parkinsonsian drugs, atropine, scopolamine, herbals containing belladonna alkaloids, mushrooms	Withdrawal of dopamine agonists, initiation of antipsychotic medications, including atypicals, metoclopramide, droperidol	SSRI, medications with serotonin activity	Succinylcholine, inhaled anesthetic agents
Duration of pharmacologic treatment	Variable	Variable	Variable	Unknown	48–72 hrs after symptoms resolve
Pharmacotherapy	Sympatholytics, including benzodiazepines	Sedatives, physostigmine (controversial)	Possibly effective-bromocriptine, dantrolene	Benzodiazepines, cyproheptadine > chlorpromazine	Dantrolene
Rechallenge	Variable	Variable	Cautiously	Not recommended	Not recommended

Malignant hyperthermia (MH)

- An uncommon autosomal dominant pharmacogenetic disorder of skeletal muscle
- Results in an extreme form of hypermetabolic crisis
- Susceptible patients expose to potent volatile anaesthetics or depolarizing muscle relaxant
- Rare in stressful conditions like vigorous exercise or heat

Malignant hyperthermia (MH)

- Overall incidence
  - 1 per 250,000 anesthetic procedures
  - Incidence is higher 1 per 62,000 when combination of potent inhaled anesthetic agents and succinylcholine
  - First three decades of life
  - ~half in patients younger than 15 years of age
  - Male>female (2-4 times more)

Malignant hyperthermia (MH)

- First recognized in 1960s, mortality rate was as high as 70-80%
- Dramatically decrease to 5-10% after introduction of dantrolene/early detection of MH
- Mortality remains high 34.8%
  - Renal dysfunction (97.3%)
  - Conscious level (9.8%)
  - Cardiac dysfunction (9.4%)
  - DIC (7.2%)

Larach MG, Gronert GA, Allen GC, Brandom BW, Lehman EB
Clinical presentation, treatment, and complications of malignant hyperthermia in North America from 1987 to 2006
Anesth Analg 2010; 110: 498-507

Triggers
(must be avoided)

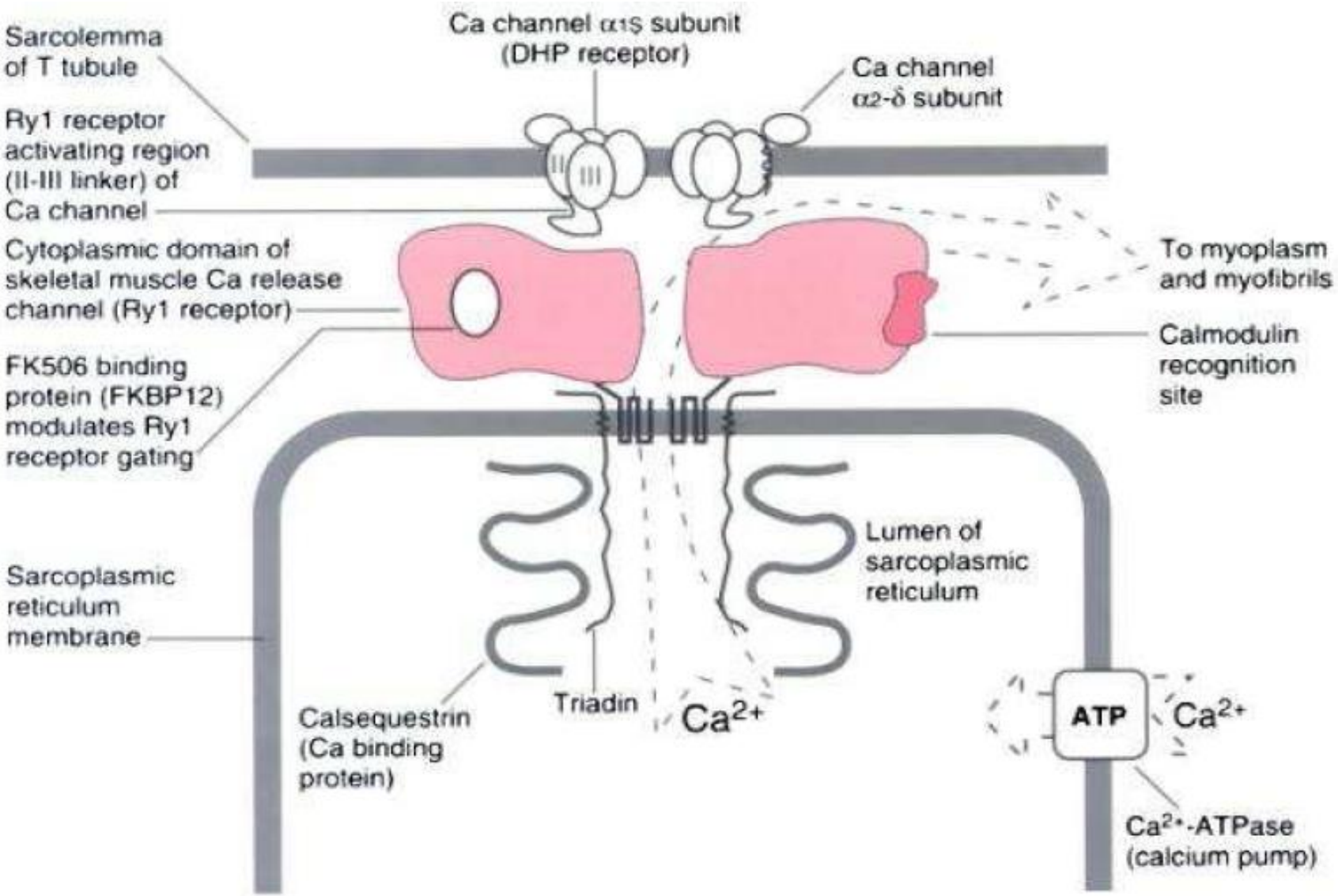
Halothane
Enflurane
Isoflurane
Sevoflurane
Desflurane
Succinylcholine

Safe drugs

All intravenous anaesthetics including ketamine
All benzodiazepines
All non-depolarising neuromuscular blocking drugs
All local anaesthetics,including preparations containing vasoconstrictors
All analgesics,including opioids
Neostigmine
Atropine
Glycopyrrolate
Metoclopramide
Droperidol

Pathophysiology

- Exact mechanism not fully elucidated
- Related to uncontrolled release of calcium from skeletal muscle sarcoplasmic reticulum
- ↑iCa stimulate metabolism in 2 ways:
  - Directly through activation of phosphorylase to ↑glycolysis
  - Indirectly as demand for ATP production
    - ATPase important components of myofilament relaxation + Ca sequestration pumps of SR and sacrolemma



Excitation-contraction coupling

Pathophysiology

- Uncontrolled release of Ca to myoplasma
- Sustained muscle contraction
- Rapid depletion of ATP
- Increase in glucose metabolism/O2 consumption/CO2 and heat production
- Failure of membrane integrity
- Leakage of cell contents (e.g. electrolytes, myoglobin, CK)

Pathophysiology

- Defective in Ryanodine 1 receptor (RYR1)
  - ~70% of families with MHS having mutations in RYR1
  - At least 4 chromosomal locations identified:
    - Most common one: chromosome 19q3
    - Also in 17, 7 and 3
- Defective in dihydropyridine receptor (DHPR) or FK 506 binding protein
  - Mutations in CACNA1s in chromosome 1q32

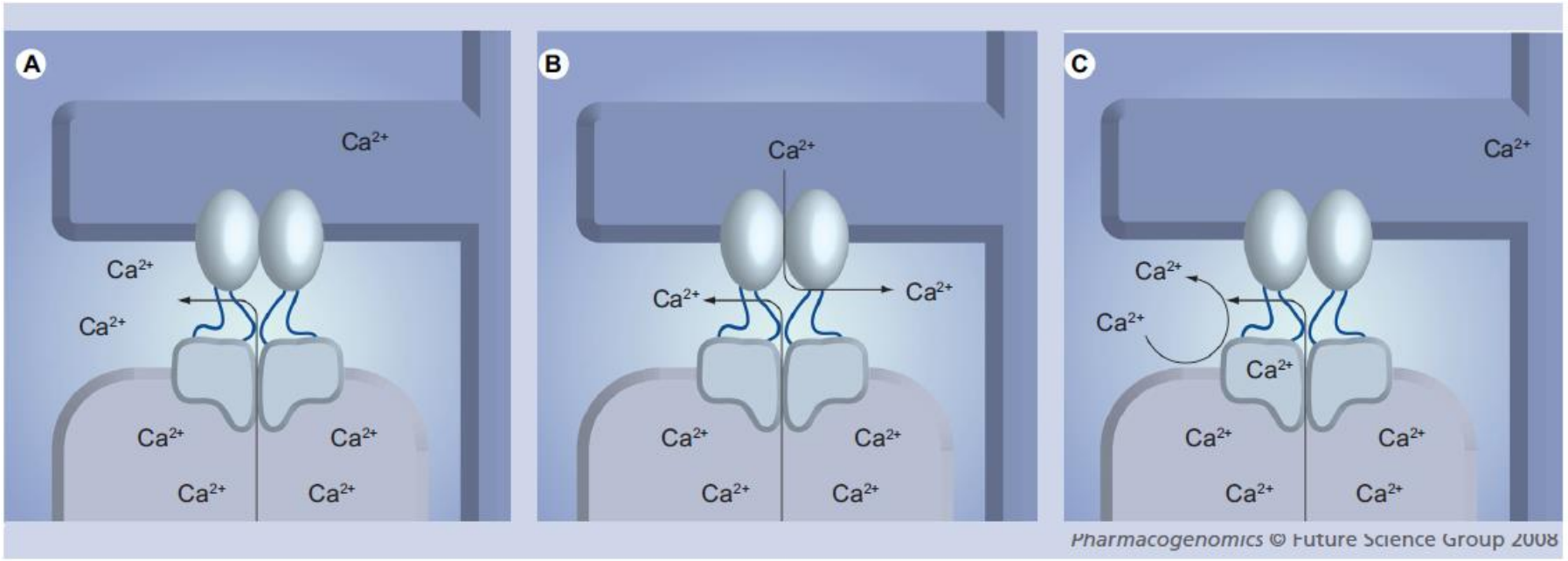
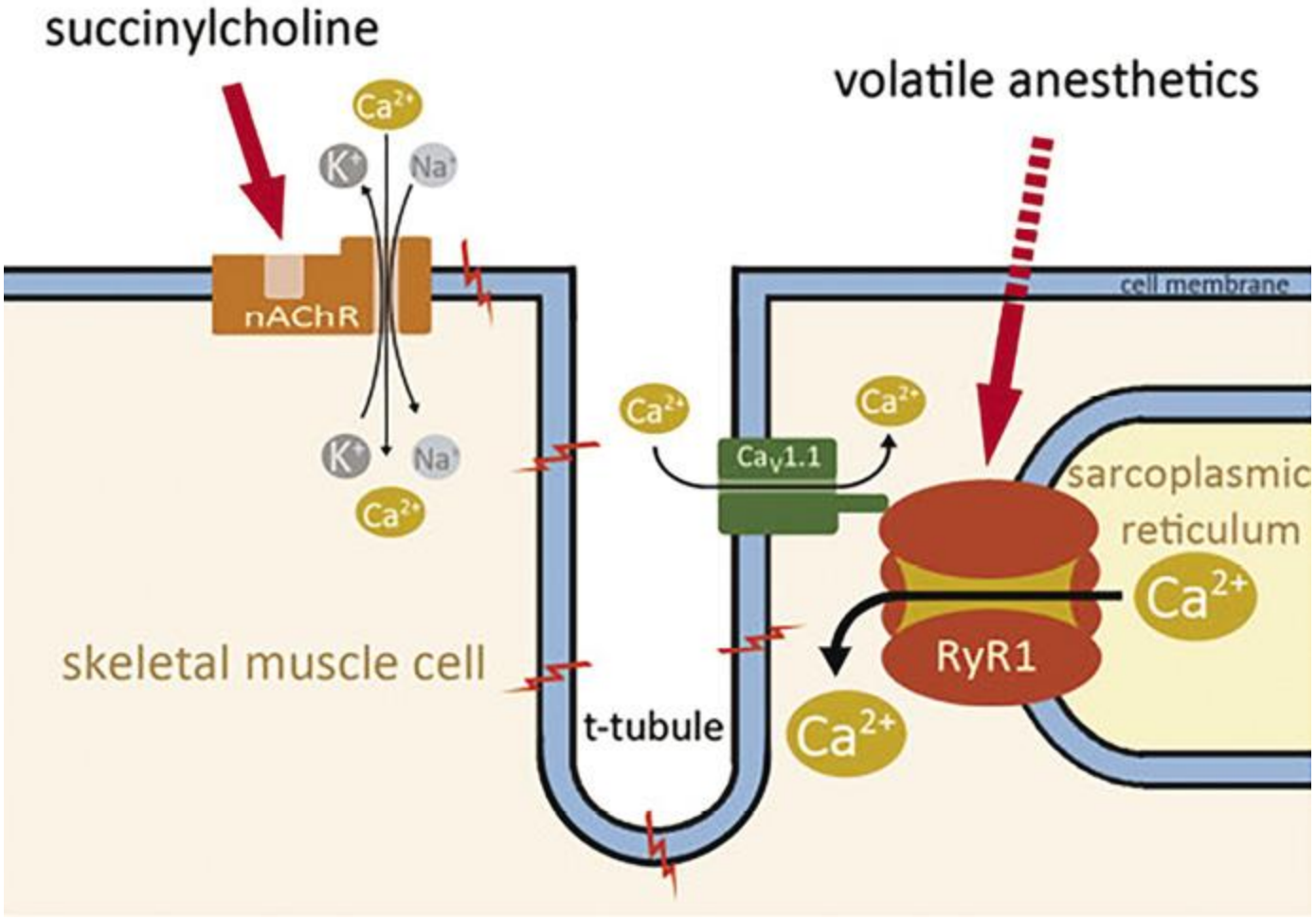


Figure 2. Models that may account for the hypersensitivity of the RyR1 channel with malignant hyperthermia mutations. **(A)** Leakage from the sarcoplasmic reticulum stores. **(B)** Increased influx through the plasma membrane. **(C)** Increased efflux from stores as a result of increased calcium-induced calcium release.


succinylcholine
volatile anesthetics
Ca2+
K+
Na+
nAChR
cell membrane
Ca2+
Ca2+
CaV1.1
sarcoplasmic
reticulum
Ca2+
RyR1
skeletal muscle cell
t-tubule
Ca2+

Clinical presentation

Table 3. Clinical Signs Associated with MH (Adapted from Glagn et al. [70])

Early signs

Metabolic

- Inappropriately elevated CO_2 production (raised end-tidal CO_2 on capnography, tachypnoea if breathing spontaneously).
- Increased O_2 consumption.
- Mixed metabolic and respiratory acidosis.
- Profuse sweating.
- Mottling of skin.

Cardiovascular

- Inappropriate tachycardia.
- Cardiac arrhythmias (especially ectopic ventricular beats and ventricular bigemini).
- Unstable arterial pressure.

Muscle

- Masseter spasm if succinylcholine has been used.
- Generalized muscle rigidity.

Later signs

- Hyperkalaemia.
- Rapid increase in core body temperature.
- Grossly elevated blood creatine phosphokinase levels.
- Grossly elevated blood myoglobin levels.
- Dark-colored urine due to myoglobinuria.
- Severe cardiac arrhythmias and cardiac arrest.
- Disseminated intravascular coagulation.

Clinical Grading Scale for MH

Process	Clinical Criteria	Points
Muscle rigidity	Generalized rigidity	15
	Masseter muscle rigidity	15
Muscle breakdown	Creatine kinase > 10,000 units/l	15
	Cola-colored urine	5
	Excess myoglobin in urine or serum	3
	K+ > 6 mEq/l	
Respiratory acidosis	End-tidal CO_2 > 55 mmHg; $PaCO_2$ > 60 mmHg	15
	Inappropriate tachypnea	10
Temperature increase	Rapidly increasing temperature	15
	Inappropriate temperature > 38.8°C	10
Cardiac involvement	Unexplained sinus tachycardia, ventricular tachycardiac, or ventricular fibrillation	3
Family history	MH history in first-degree relative	15
	MH history in family, not first-degree relative	5

Raw score range	MH rank	Description of likelihood
0	1	Almost never
3–9	2	Unlikely
10–19	3	Somewhat less than likely
20–34	4	Somewhat greater than likely
35–49	5	Very likely
> 50	6	Almost certain

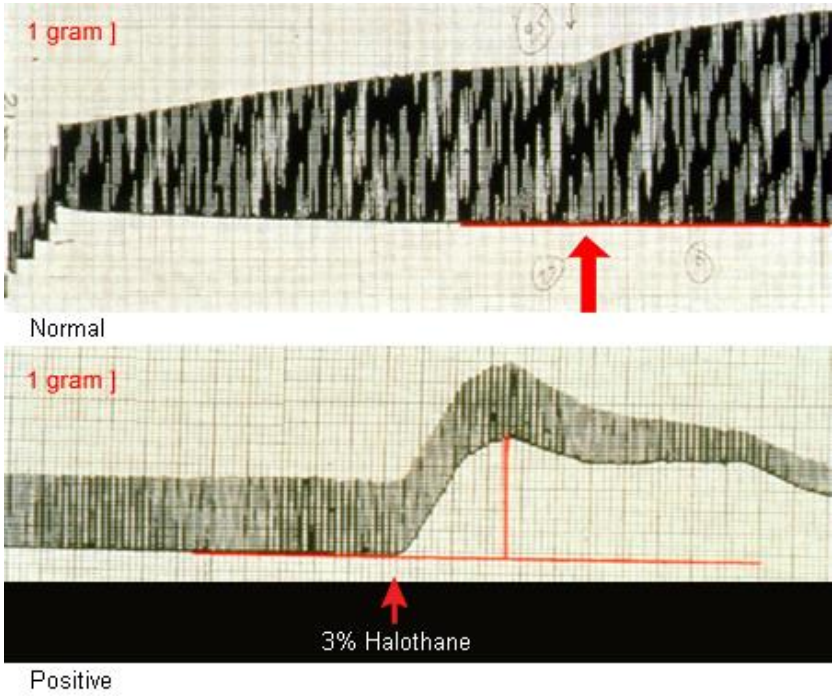
Larach MG, Localio AR, Allen GC, Denborough MA, Ellis FR, Gronert GA, et al
A clinical grading scale to predict malignant hyperthermia susceptibility
Anesthesiology 1994; 80: 771-9

Laboratory diagnostic tests

- Confirmation diagnostic test is important in pt survived from a suspected MH episode
  - Prevent future exposure to triggering agents, ↓anaesthetic risk
  - Other family members may be affected by this dominantly inherited disorder

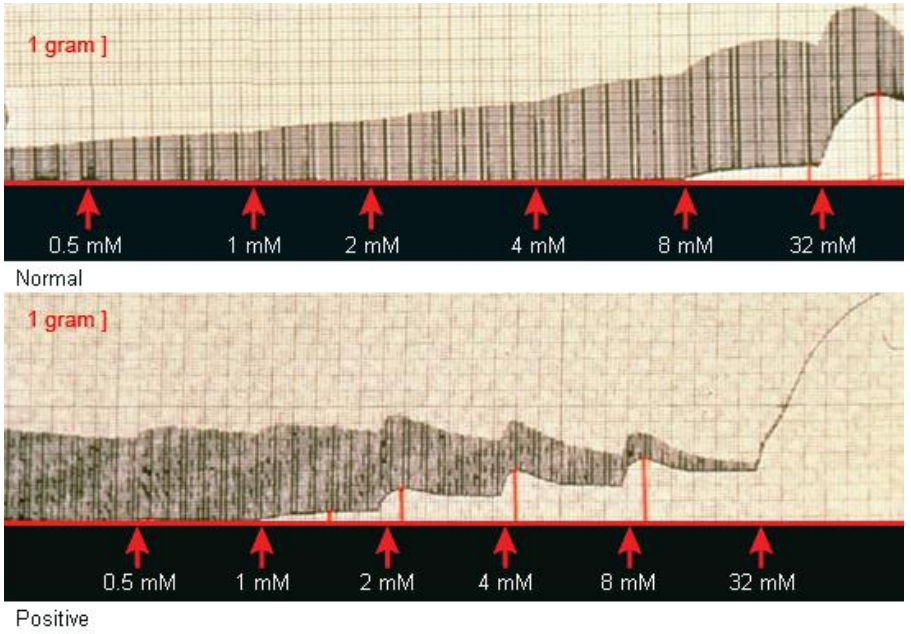
Laboratory diagnostic tests

- Muscle biopsy contracture test
  - Gold standard
  - Excised muscle, usually from vastus lateralis, mounted in baths with caffeine & halothane alone or in combination
  - Contracture responses are measured and interpreted to standardized values
  - Only available in specialized centres
  - Fresh specimen mandates patient travel to the centre for testing



The mechanical response of normal and MH-susceptible muscle (positive) to direct stimulation in the presence of a bolus of 3% halothane

The mechanical response of normal and MH-susceptible muscle (positive) to direct stimulation in the presence of incremental administration of caffeine



Laboratory diagnostic tests

- Muscle biopsy contracture test (MCT)
  - 2 different protocols
    - In vitro contracture test (IVCT)
      - European Malignant Hyperthermia Group (EMHG)
    - Caffeine-halothane contracture test (CHCT)
      - North American Malignant Hyperthermia Group (NAMHG)
  - Classify patients into 3 groups
    - Normal (MHN)
    - Equivocal (MHE)
    - Susceptible (MHS)

Laboratory diagnostic tests

- Molecular genetic testing
  - Positive MCT, strong FH, clinical episode of suspected MH
  - Discordance between phenotype and genotype
    - >300 MHS associated mutations in RYR1
    - Only ~34 confirmed functionally with muscle contracture test
  - Variable penetrance and expressivity within the susceptible family
  - Only can be diagnostic in one situation
    - When an MH case confirmed with MCT and causative mutation loci identified
    - Family member can be Dx MHS directly once genetic test shown presence of same mutation

Table 1 – Conditions that Mimic MH

Fever (without rigidity)	Fever and/or muscle symptoms	Increased End-Tidal CO_2
Thyrotoxicosis	NMS (psych meds)	Faulty equipment
Sepsis	Hypoxic encephalopathy	Tourniquet (children)
Pheochromocytoma	CSF ionic contract agents	Laparoscopic insufflation
Iatrogenic overheating	Cocaine, amphetamine, ecstasy	
Anticholinergic syndrome	Dystrophinopathy	
	Myotonic syndromes	
	Rhabdomyolysis	

Treatment of acute MH

- Decide whether the surgical procedure should be postponed (emergency v.s. elective)
- Stop volatile anesthetics and succinylcholine
  - Use non-triggering agents if surgical procedure must be continued
- Hyperventilate with 100% O2
- Cooling techniques, e.g. external or internal
  - No less than 38°C to avoid Cx from hypothermia
- Dantrolene sodium
- Correction of hyperK/metabolic acidosis
- Watch out rhabdomyolysis/DIC

Dantrolene sodium

- Specific ryanodine receptor antagonist
- Blocking Ca release from SR
- Neuromuscular transmission + electrical properties of skeletal muscle membrane unaffected
- No effect on smooth/cardiac muscle

Dantrolene sodium

- Each vial:
  - lyophilized orange powder 20mg + mannitol 3g (isotonicity) + NaOH (increase solutbility)
  - diluted with 60ml H2O for injection (incompatible with acidic solutions)

- Pharmacokinetics
  - IV or oral form (20% bioavailability)
  - Metabolized by liver by hydroxylation to weakly active metabolite and excrete in urine/bile
  - T1/2 ~12 hrs

Dantrolene sodium

- Recommended initial dosage is 2.5mg/kg IV every 15 mins until 10mg/kg or symptoms resolved
- 1mg/hr every 4-6 hrs or 0.25mg/kg/hr continuous infusion for next 24 hrs to prevent recurrence

Dantrolene sodium

- S/E of dantrolene
  - From formulation:
    - Mannitol – diuresis may occur (? Good in case of rhabdomyolysis)
    - NaOH – thromboplebitis
  - Side effect related to dantrolene itself
    - Skeletal muscle weakness – usually does not affect respiration / coughing
    - GI upset – nausea / vomiting / diarrhea
    - Uterine atony
    - Hepatitis + pleural effusion in chronic PO uses

Dantrolene sodium

- Drug interaction
  - Avoid concurrent use of Ca channel blocker (e.g. verapamil, diltiazem) for control of cardiac arrhythmias
  - Cause hyperkalemia, VF, profound myocardial depression, cardiac arrest
  - Also retriggers MH

Dantrolene in the Treatment of Refractory Hyperthermic Conditions in Critical Care: A Multicenter Retrospective Study

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Open Journal of Anesthesiology, **2015**, 63-71.

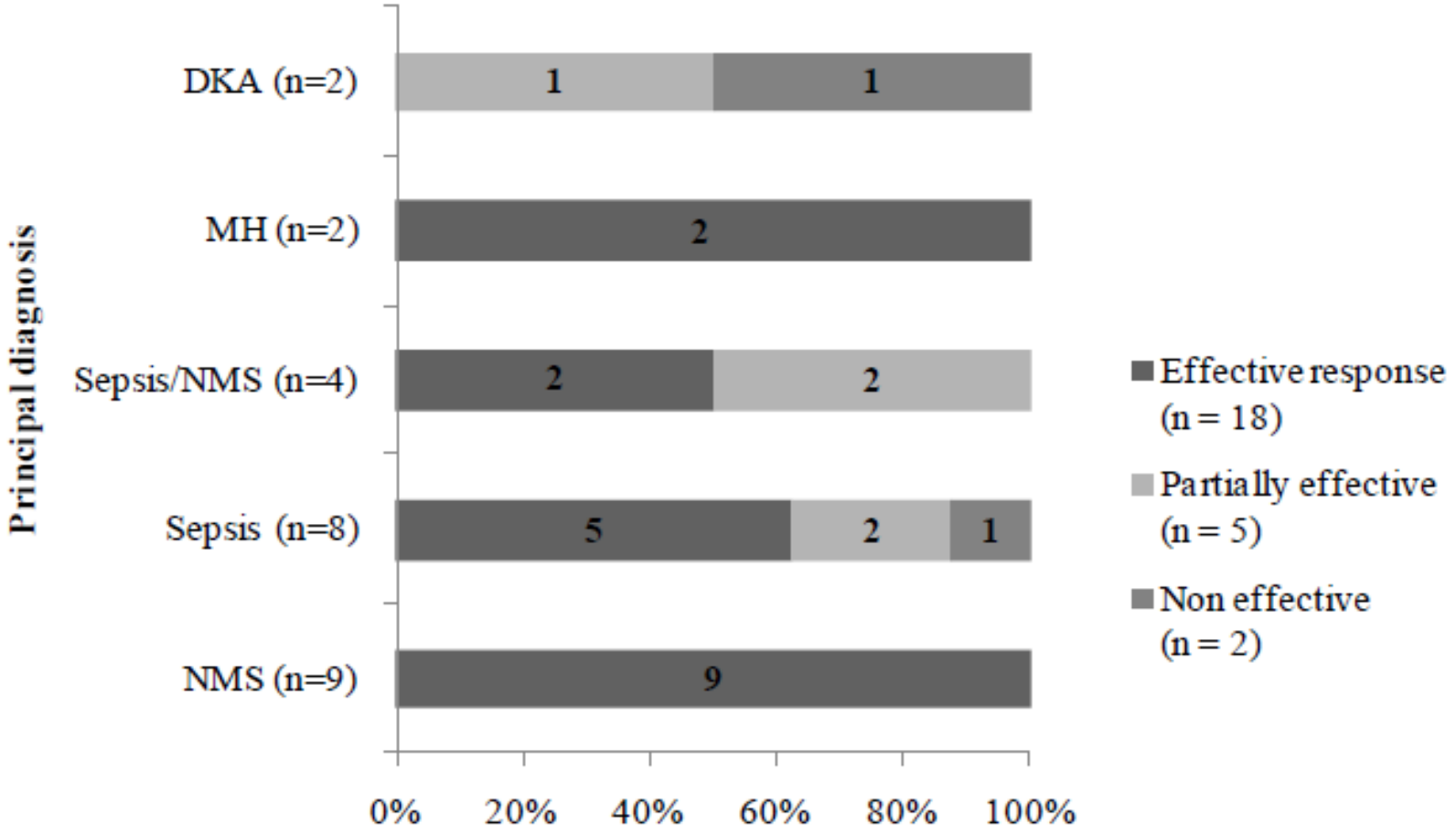
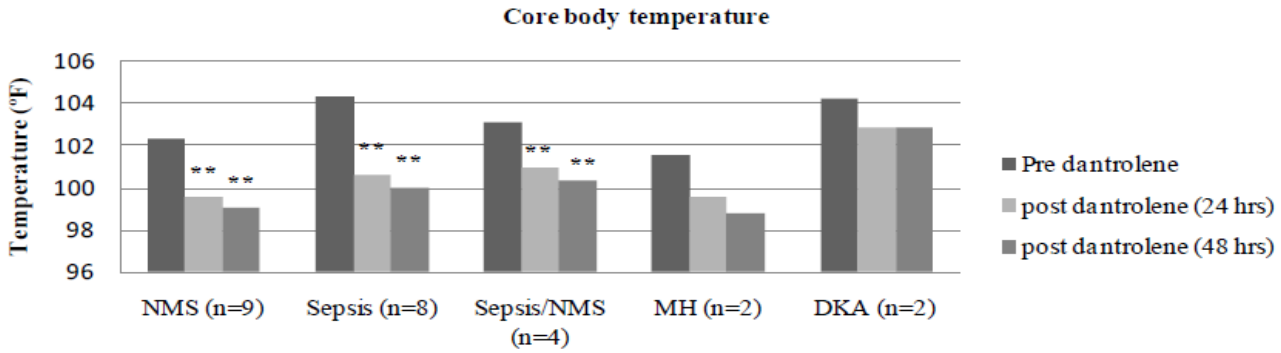


Figure 2. Overall response to dantrolene administration in 25 patients. Abbreviations: Data is presented as number of patients (n); NMS, neuroleptic malignant syndrome; DKA, diabetic ketoacidosis; MH, malignant hyperthermia; Effective response, dantrolene use was associated with a lowered temperature, lowered creatine kinase levels and lowered degree of rigidity; Partially effective, lowered either 1 or 2 of the 3 clinical parameters; Non effective, no effect.


Core body temperature
Temperature (°F)
106
104
102
100
98
96
**
**
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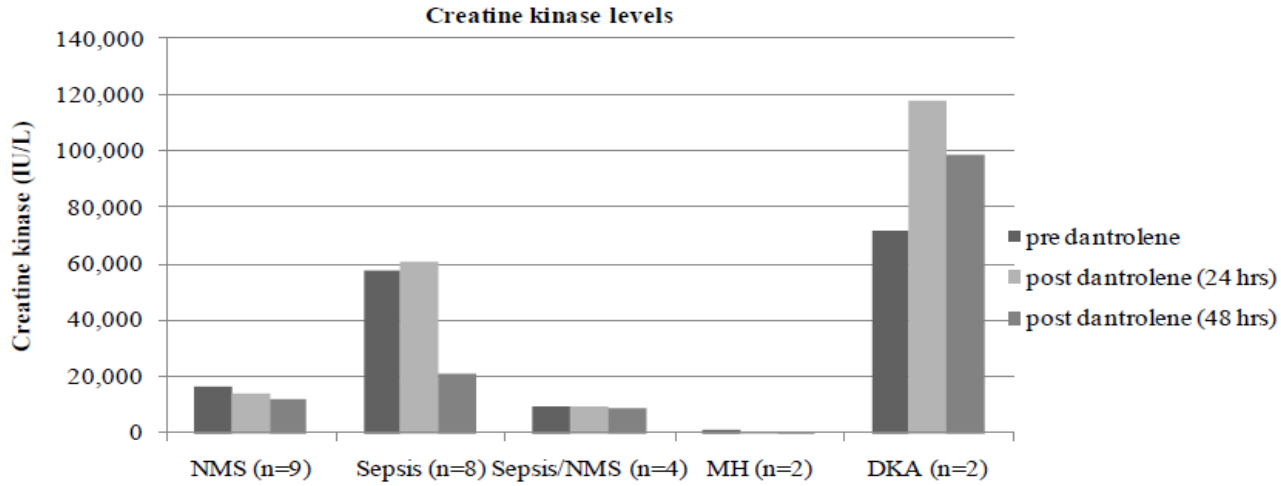
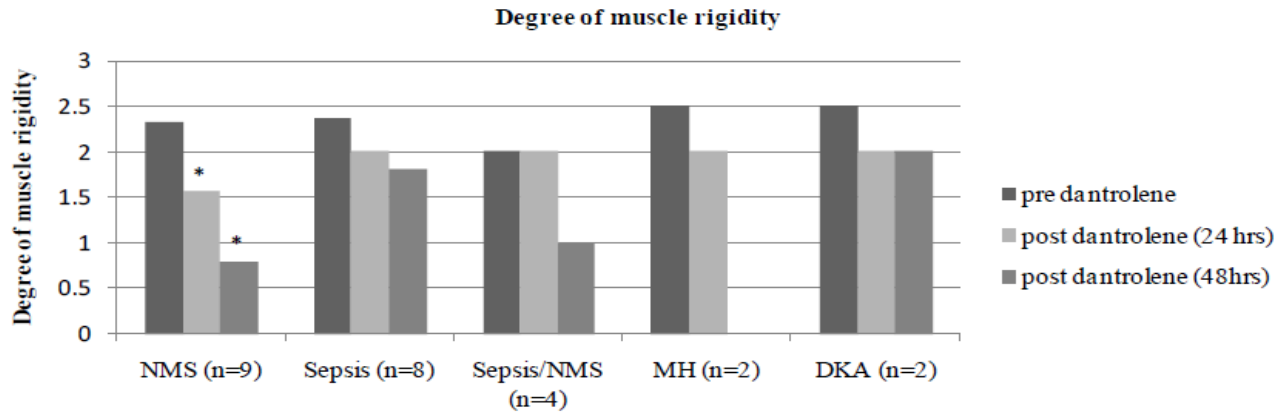
**
**
**
NMS (n=9)
Sepsis (n=8)
Sepsis/NMS (n=4)
MH (n=2)
DKA (n=2)
Pre dantrolene
post dantrolene (24 hrs)
post dantrolene (48 hrs)

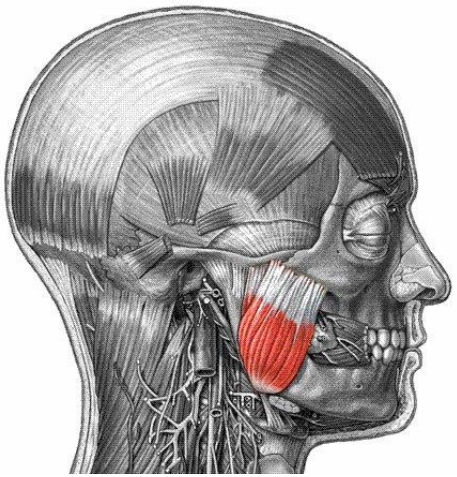

Creatine kinase levels
Creatine kinase (IU/L)
140,000
120,000
100,000
80,000
60,000
40,000
20,000
0
NMS (n=9)
Sepsis (n=8)
Sepsis/NMS (n=4)
MH (n=2)
DKA (n=2)
pre dantrolene
post dantrolene (24 hrs)
post dantrolene (48 hrs)


Degree of muscle rigidity
Degree of muscle rigidity
3
2.5
2
1.5
1
0.5
0
*
*
NMS (n=9)
Sepsis (n=8)
Sepsis/NMS (n=4)
MH (n=2)
DKA (n=2)
pre dantrolene
post dantrolene (24 hrs)
post dantrolene (48hrs)

Masseter muscle rigidity (MMR)

- Sustained contraction of masseter muscles causing marked stiffness of jaw barely allows any opening of mouth

- Associated with use of succinylcholine



Succinylcholine

- Mild increase in masseter muscle resting tension following use of succinylcholine with limb faccidity is a normal response

- MMR
  - Occur more frequently in children, with or without inhalation agents
  - Presage of MH, ~10% will progress to generalized rigidity
  - MH may follow immediately or delayed for several minutes
  - Dantrolene is not recommend unless clinical signs of MH
  - Likelihood to develop rhabdomyolysis, regular monitor CK level
  - Discuss muscle biopsy afterwards (50% cases found to be MHS)

Hong Kong Journal of Emergency Medicine

Succinylcholine-induced masseter muscle rigidity in an emergency department: a case report

琥珀膽鹼引起之咀嚼肌僵硬：一個在急症室內的個案報告

KW Suen 孫健榮, HY Lee 李凱揚, HF Ho 何曉輝

Succinylcholine is the most popular muscle relaxant employed in local accident and emergency departments because of its effectiveness and short-acting half life. Significant adverse reaction is rare and it increases the success rate of intubation. We describe a case of masseter muscle rigidity after administration of succinylcholine alone in the emergency room. The patient subsequently required nasotracheal intubation for ventilation. (*Hong Kong j.emerg.med.* 2010;17:281-284)

琥珀膽鹼是本地急症室內最常用的肌肉鬆弛劑，因它半衰期短及有效提升氣管內插管的成功率，而嚴重的副作用則罕見。本文描述一個在急症室內，只注射了琥珀膽鹼後咀嚼肌僵硬的個案，病人其後需經鼻氣管插管以換氣。

Keywords: Intratracheal intubation, malignant hyperthermia, spasm, trismus

The END