H2Noooooo!!! Water-related injury

June 2019

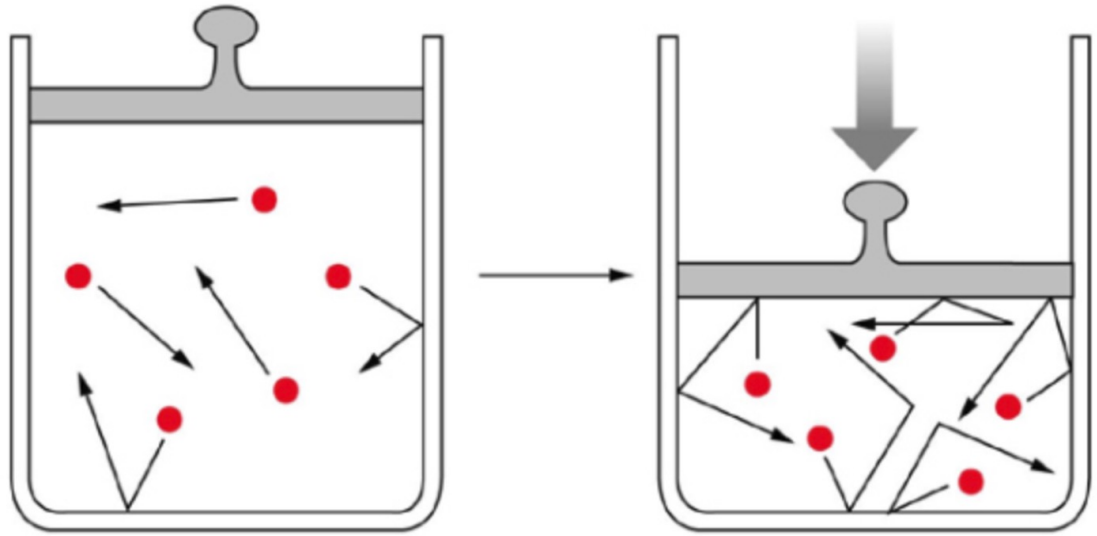
Dr Stephanie Valent

- 47 year old male
- Carried into triage by friends, wet, unconscious
- Has been scuba diving in lake Pupuke
- Dive buddy lost site of him
- 10 mins later found floating on surface

Decompression Illness

- Decompression sickness
  - bubbles growing in tissue and causing local damage
- Arterial Gas Embolus (AGE)
  - bubbles entering the lung circulation, traveling through the arteries and causing tissue damage at a distance by blocking blood flow at the small vessel level

Decreasing volume increases collisions and increases pressure.

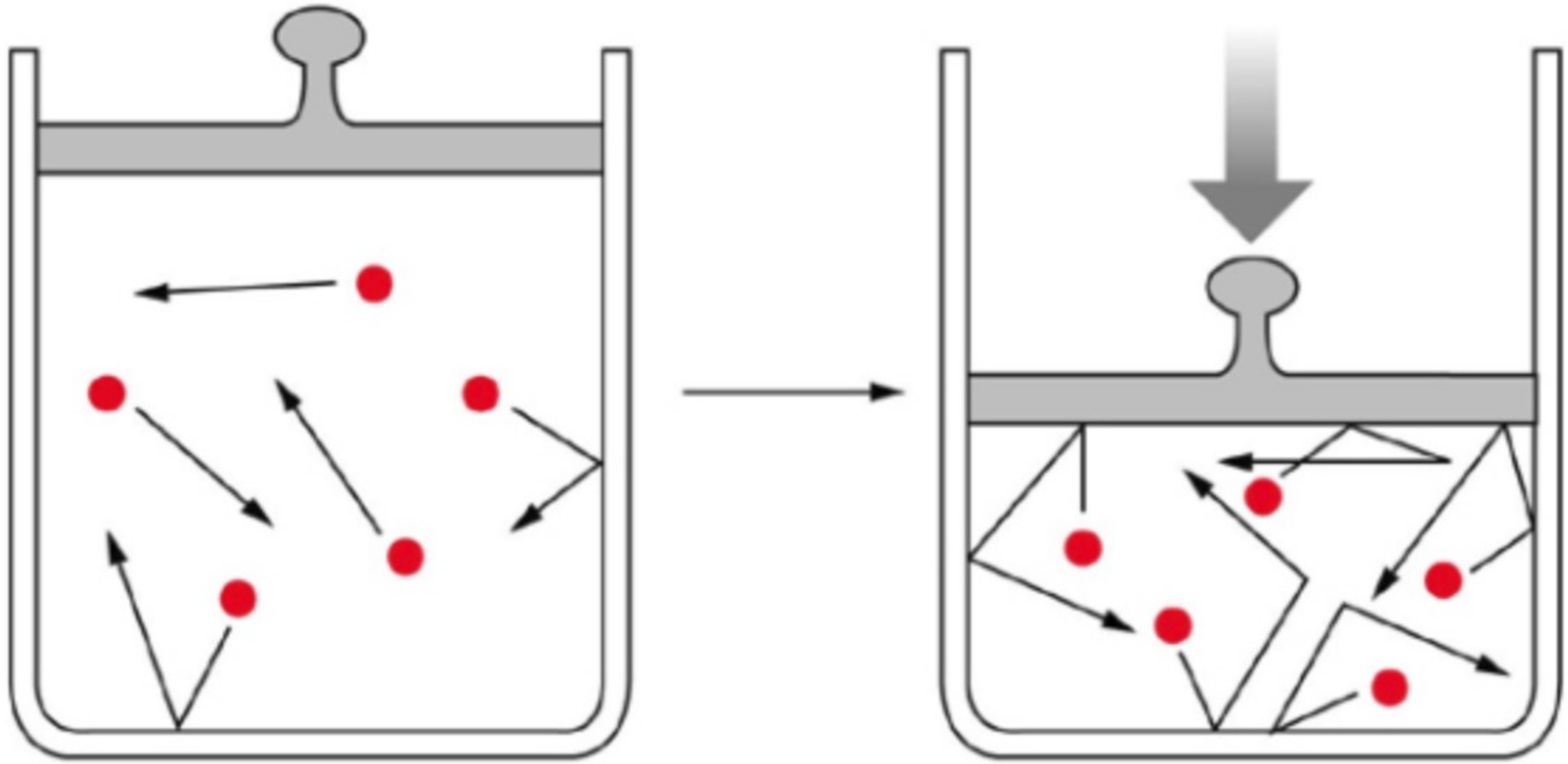


$V_1 = 1.0\,L$
$P_1 = 100$ mm Hg

$V_2 = 0.5\,L$
$P_2 = 200$ mm Hg

Boyle's Law: $P_1V_1 = P_2V_2$

Decreasing volume increases collisions and increases pressure.



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Decompression Illness

- **Risk factors**
  - deep / long dives
  - cold water
  - hard exercise at depth
  - rapid ascents

  - obesity
  - dehydration
  - hard exercise immediately after surfacing
  - pulmonary disease

Decompression Illness

- Decompression sickness
- Arterial gas embolism

Decompression Sickness

- During a dive body tissues absorb nitrogen from SCUBA in proportion to the surrounding pressure
- As long as the diver remains at pressure, the gas presents no problem.
- If the pressure is reduced too quickly, nitrogen comes out of solution and forms bubbles
- Can occur even if dive tables are adhered to

Decompression Sickness

- S&S usually appear within 15 minutes to 12 hours after surfacing
- Severe cases may appear before surfacing or immediately afterwards
- Delayed occurrence of symptoms is rare
  - Air travel

Decompression Sickness: Symptoms

- **DENIAL!!!**
- **Pain** in joints (classical ‘bends’ and / or muscles of the arms, legs or torso ->osteonecrosis
- **Numbness, tingling** and paralysis
- Unusual fatigue
- Skin itch
- Dizziness, vertigo, ringing in the ears
- Shortness of breath (chokes)
- Difficulty urinating

Decompression Sickness: Signs

- Blotchy rash
- Paralysis, muscle weakness
- Confusion, personality changes, bizarre behaviour
- Amnesia, tremors
- Ataxia
- Bloody, frothy sputum
- Collapse or unconsciousness

Decompression Sickness

- Long term sequelae
  - Symptomatic
  - Asymptomatic

AGE - Symptoms

- Numbness
- Weakness
- Tingling
- Dizziness
- Blurred vision
- Chest pain
- Personality change or difficulty thinking
- Bloody sputum
- Paralysis or seizures
- Loss of consciousness

Decompression Illness Assessment

- Depth
- Duration
- Surface Intervals…or bring along dive computer
- Number of Dives
- Any significant events during the dive such as equipment failures, uncontrolled ascents, multiple ascents
- Altitude ascent post diving
- Past history of DCI
- The experience of the diver
- Comorbidities, drugs/alcohol/medications

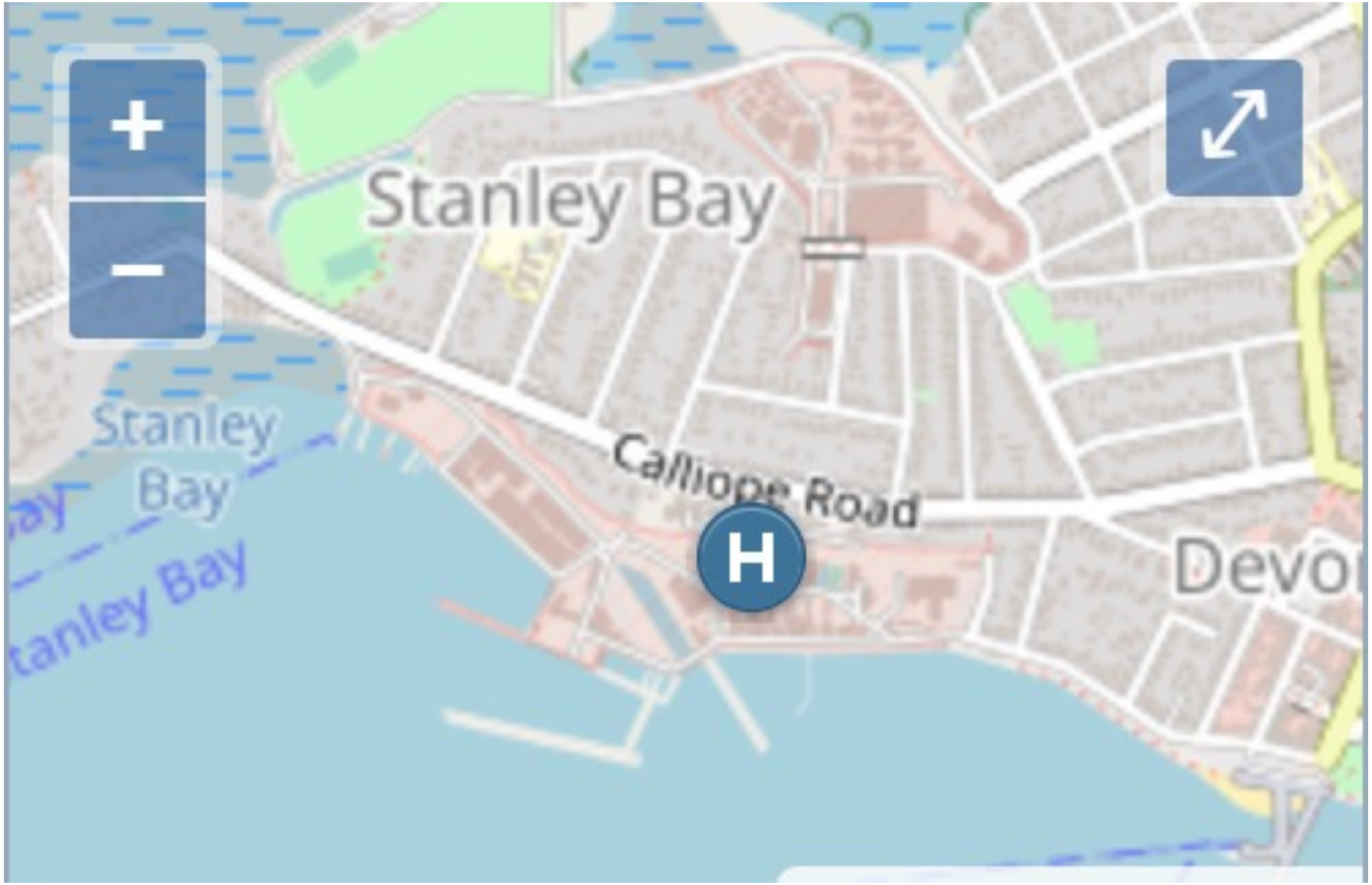
Decompression Illness Assessment

- Bloods
- Radiology – Plain xray, CT brain

Decompression Illness Treatment

- Same for AGE and DCS
- Recompression ASAP
- 02
  - may reduce symptoms substantially
  - should not change the treatment plan

- IV fluid – cautious with pulmonary involvement
- Cabin pressure should be maintained near sea level and not exceed 800 feet / 244 meters
- Prognosis


Stanley Bay
Stanley
Bay
Calliope Road
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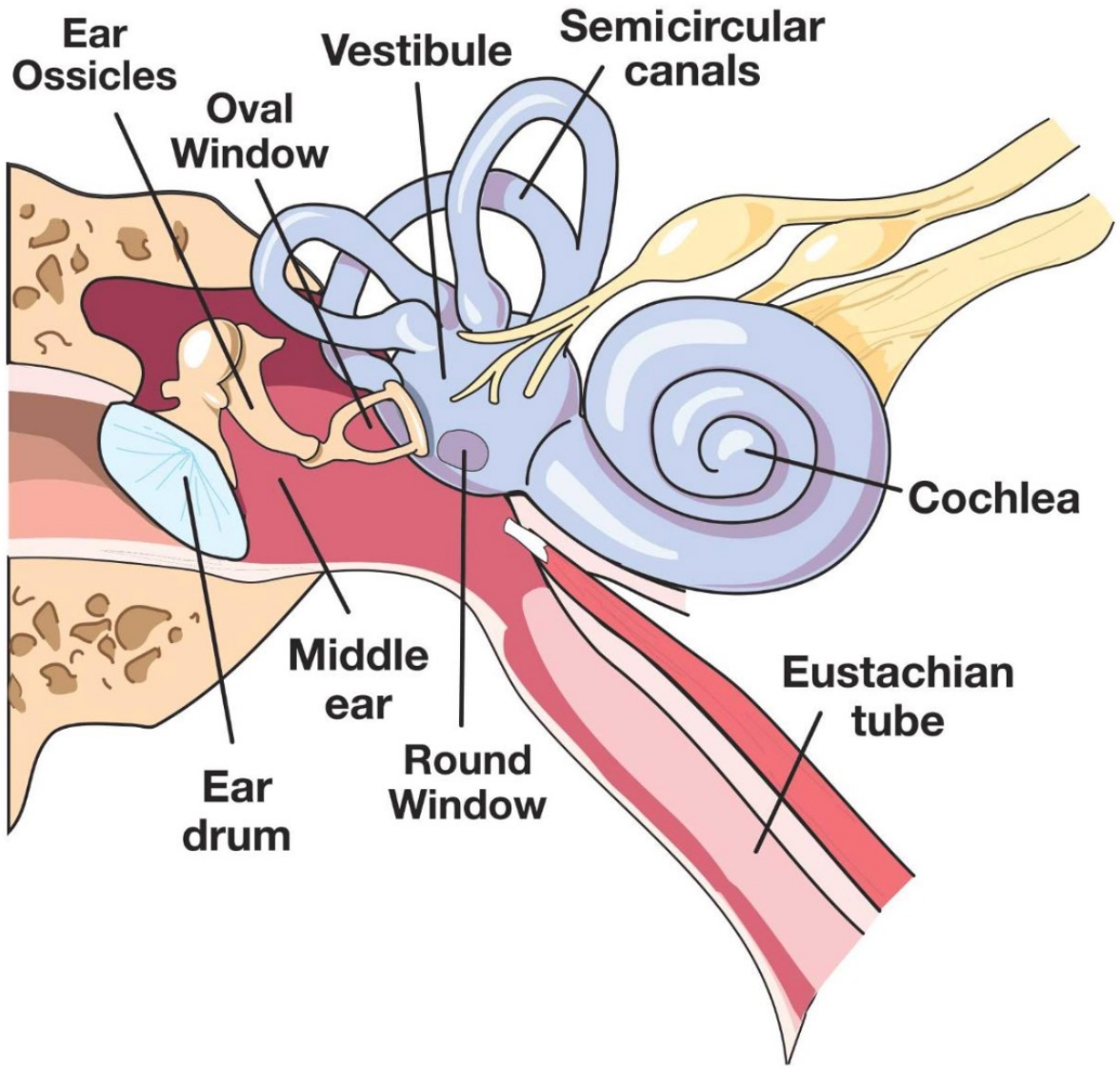
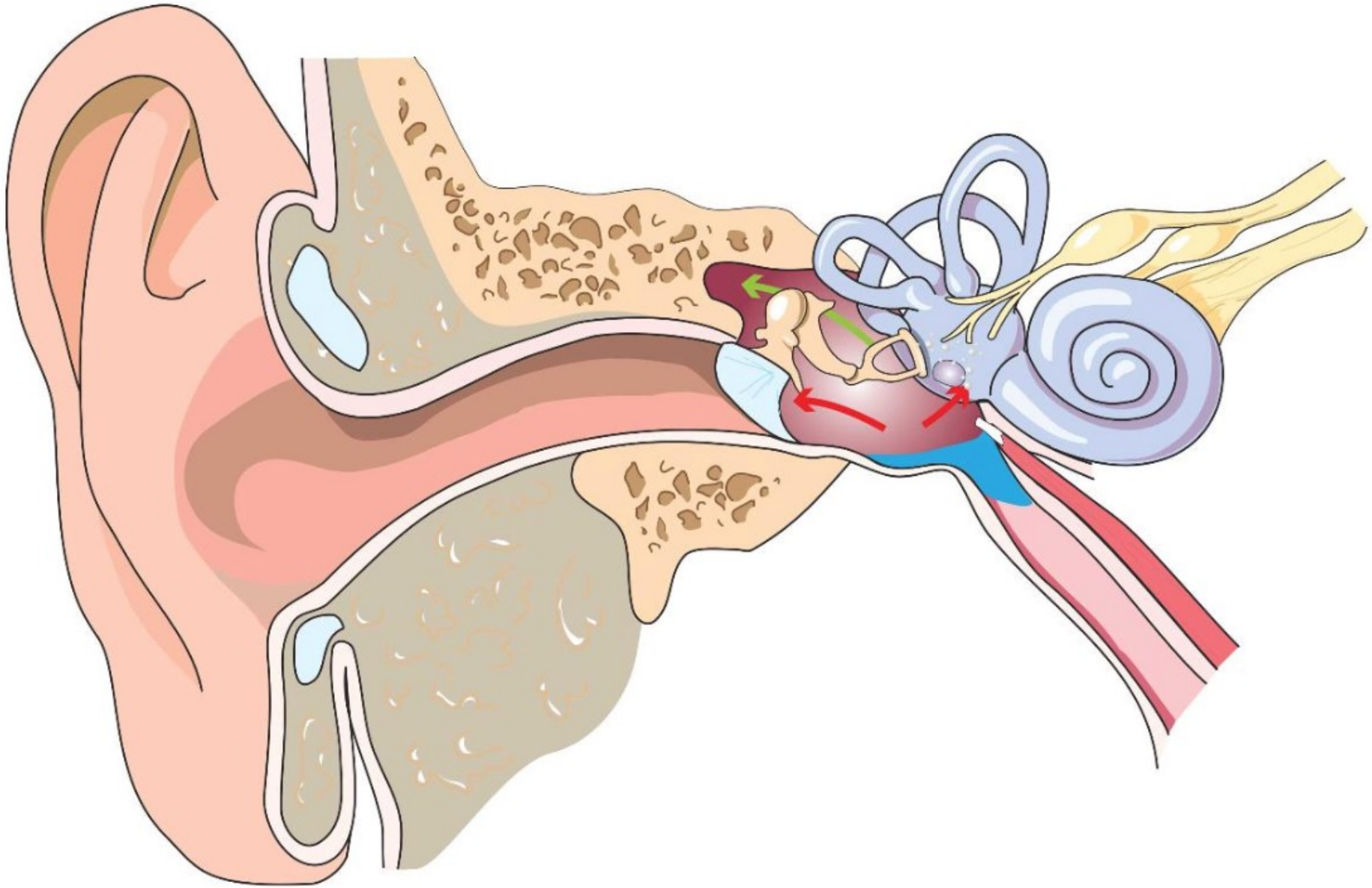
Perspective

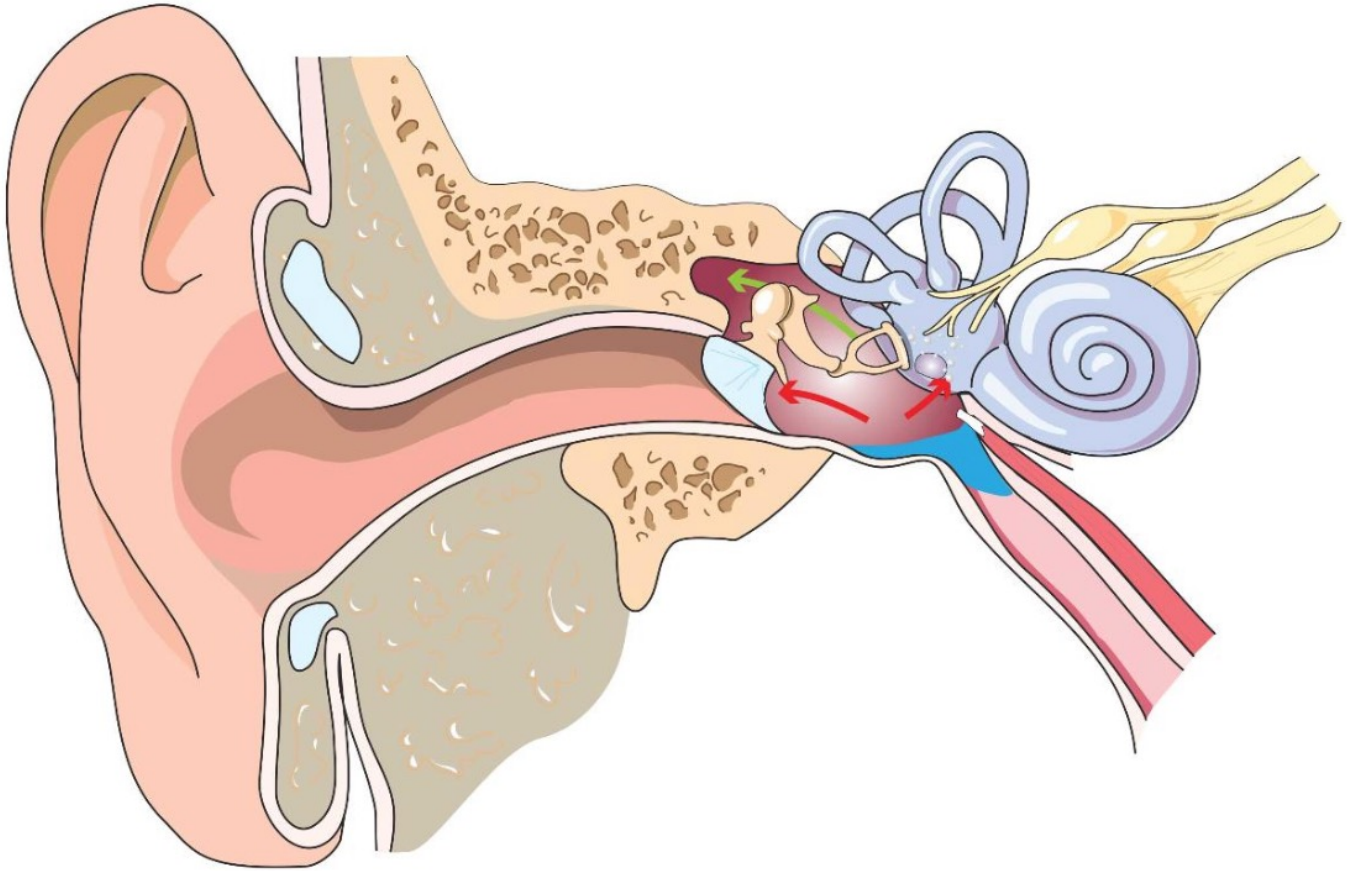
- 3-4 cases of dive injuries reported for every 10,000 dives - approximately 1,000 cases per year (US)
- Majority of the DCI cases are minor and treatment results in complete resolution with no impact on future diving activity

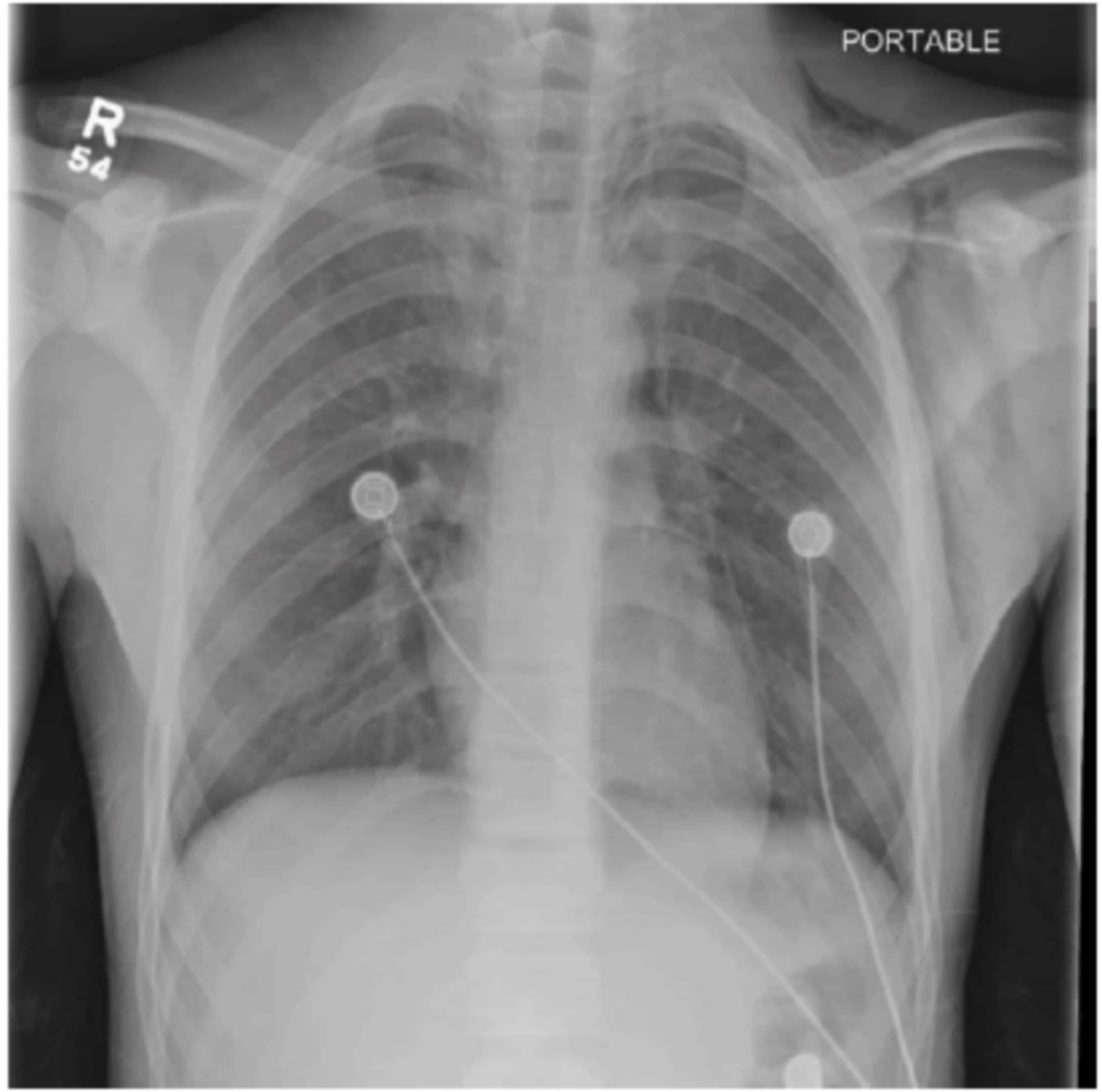
Barotrauma


Ear Ossicles
Oval Window
Vestibule
Semicircular canals
Cochlea
Middle ear
Eustachian tube
Round Window
Ear drum



A reverse squeeze is barotrauma due to an inability to release pressure from the middle ear on ascent.




PORTABLE
R
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TM Rupture

- Ear pain during descent that stops suddenly
- Clear or bloody drainage from ear
- Hearing loss
- Ringing in the ear (tinnitus)
- Spinning sensation (vertigo)
- Nausea or vomiting that can result from vertigo
- Mx: no diving until healed
  - Spontaneous healing, patch or tympanoplasty

Inner ear barotrauma

- Excessive TM pressure -> oval or round window tears ->inner-ear fluid (perilymph) leaks into the middle ear (perilymph fistula)
- If the leak is not stopped -> permanent hearing loss may occur

- Severe vertigo
- Hearing loss
- Ears roaring/ringing (tinnitus)
- Nystagmus
- Fullness of the affected ear

Inner ear barotrauma - mx

Avoid exertion, middle-ear equalization, altitude or diving exposure, sneezing or nose blowing, aspirin, nicotinic acid (vitamins), other vasodilators or anticoagulants

Conservative treatment includes bed rest in a sitting position and avoiding any strains that can increase intracranial or middle-ear pressure

If symptoms do not improve, surgery may be necessary. Healing of the tear (fistula) usually occurs within a week or two.

Inner-Ear Barotrauma or Inner-Ear Decompression Sickness?

- Treatment differs
- Symptoms are similar in both conditions

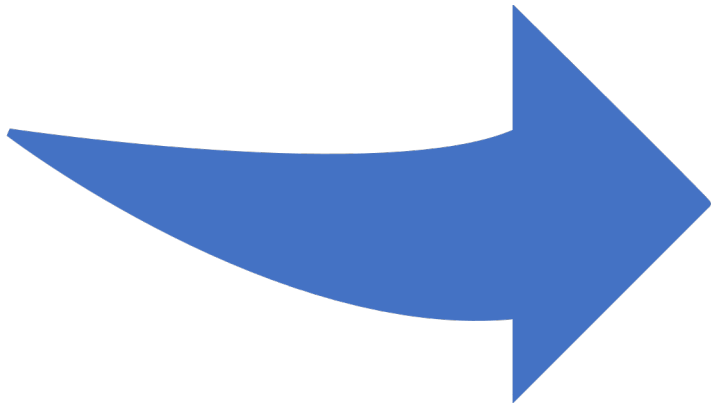
- Barotrauma is preceded by failed equalization of middle-ear pressure
  - usually occurs at the beginning of dive
- DCS occurs due to failed decompression at the end of the dive.

Alternobaric vertigo

- Disorientation, nausea and vomiting
- During ascent
- Air in the middle-ear space expands ->relative pressure increases -> Eustachian tubes open passively->gas escapes through the eustachian tubes into nasopharynx
- Unilateral blocked eustachian tube -> asymmetrical obstruction of air flow
- If pressure difference is greater than 60 centimeters of water -> vertigo (stimulation of vestibular system
- Relieved by further ascent
- Contributing factors: middle-ear barotrauma during descent, allergies, upper respiratory infections (congestion) and smoking.

Facial baroparesis
= reversible paralysis of the facial nerve due to increased pressure in the middle ear when ascending in an airplane or from scuba diving

- Overpressure in the middle ear equal or greater to capillary pressure ->circulation to facial nerve stops ->loses function

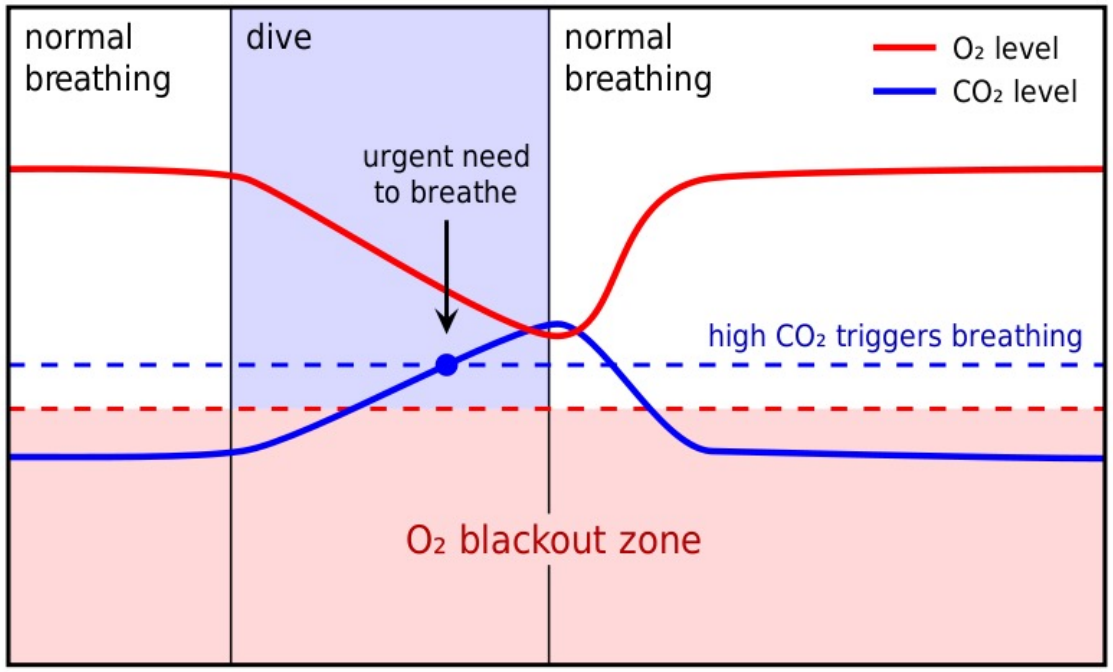


Barotrauma

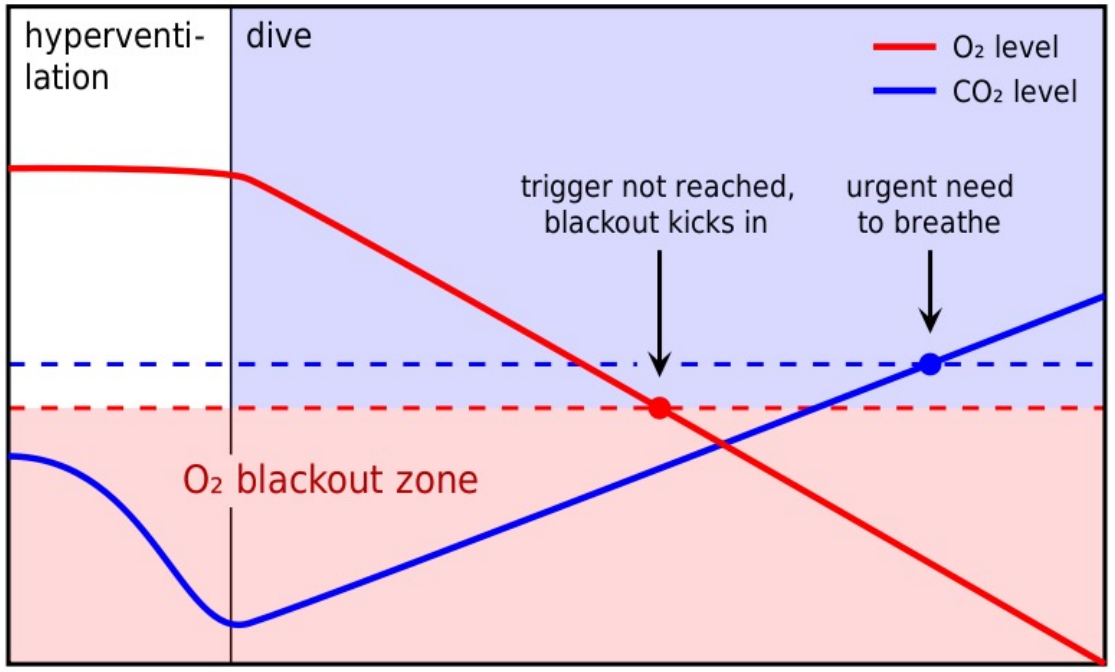
- Pneumothorax/pneumomediastinum
- Middle ear barotrauma
  - Perforated tympanic membrane
- Inner ear barotrauma
  - Perilymph fistula
- Alternobaric Vertigo
- Reverse squeeze
- Facial baroparesis

Freediving Injuries

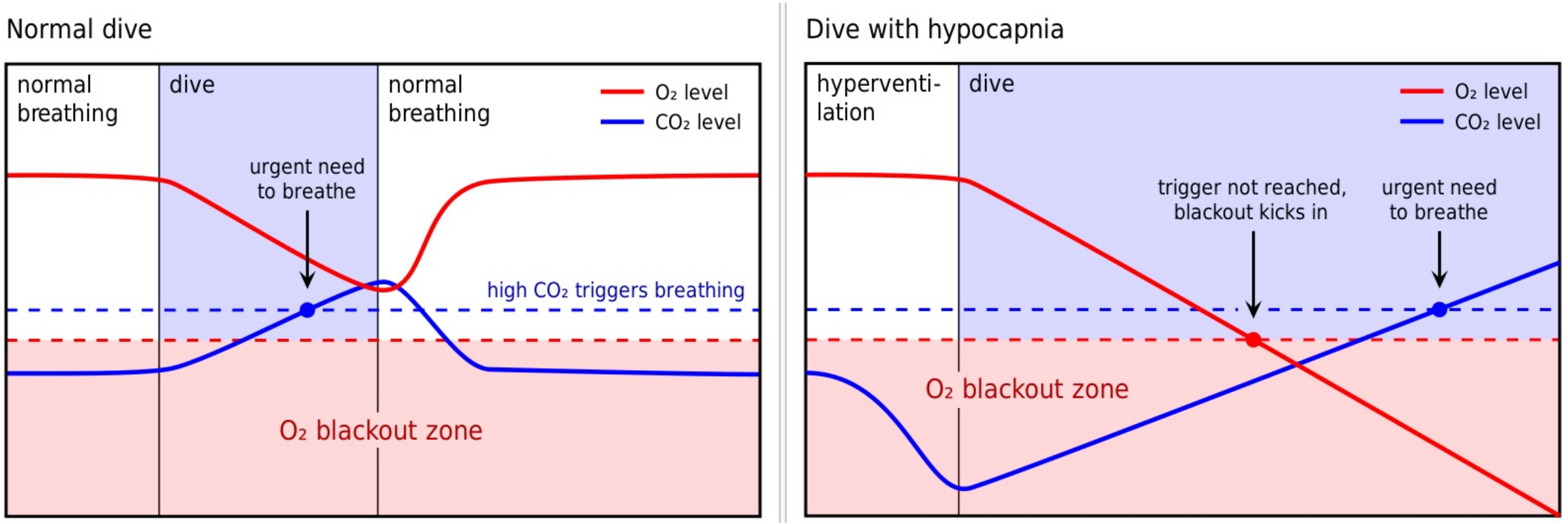
Normal dive



Dive with hypocapnia



Shallow water blackout




gettyimages
Josh Humbert
501897153

Taravana Syndrome
To fall crazily

- 40 to 60 dives/day
- Depths of 30 to 42m
- Total dive time of about 100 seconds
- Surface recovery time of 4 to 6 mins

- CNS: Vertigo, vomiting, paresthesia, muscular weakness and paralysis, impaired concentration, lethargy, speech disturbances, altered level of consciousness, visual changes and hearing loss.
- Sometimes fatal
- Many survivors suffer permanent brain and spinal cord injuries

Drowning

Drowning

"near drowning," "dry or wet drowning," "secondary drowning," "active and passive drowning," and "delayed onset of respiratory distress" are no longer used

non-fatal drowning: the drowning process is interrupted and the person survives

fatal drowning: the person dies during the drowning process (at any stage)

Mechanism

- water enters the mouth when no longer able to be kept clear (submersion or immersion)
- voluntarily spat out or swallowed at first
- next conscious response is to hold one's breath, lasts ~ 1 minute, until inspiratory drive is too high to resist
- water is aspirated into the airways ->coughing
  - laryngospasm may occur (terminated by brain hypoxia)
- continued aspiration -> hypoxemia -> loss of consciousness and apnea
- Final mode of death involves cardiac dysrhythmia: tachycardia -> bradycardia -> PEA -> asystole

- whole process, from submersion or immersion to cardiac arrest = seconds to a few minutes (>1 hour in unusual cases of hypothermia)

Non fatal drowning

- Hypoxic episode interrupted with ROSC
- End organ damage with
  - ARDS (often delayed)
  - Hypoxic ischaemic encephalophy
  - Renal failure (ATN)
  - Pancreatic necrosis
  - DIC
  - Cardiac dysrhythmias
  - Electrolyte imbalance

In hospital care of drowned patient

NEURO: head up, low normal CO_2, MAP of 80mmHg (no need for ICP monitoring), benzodiazepines for seizures, TTM

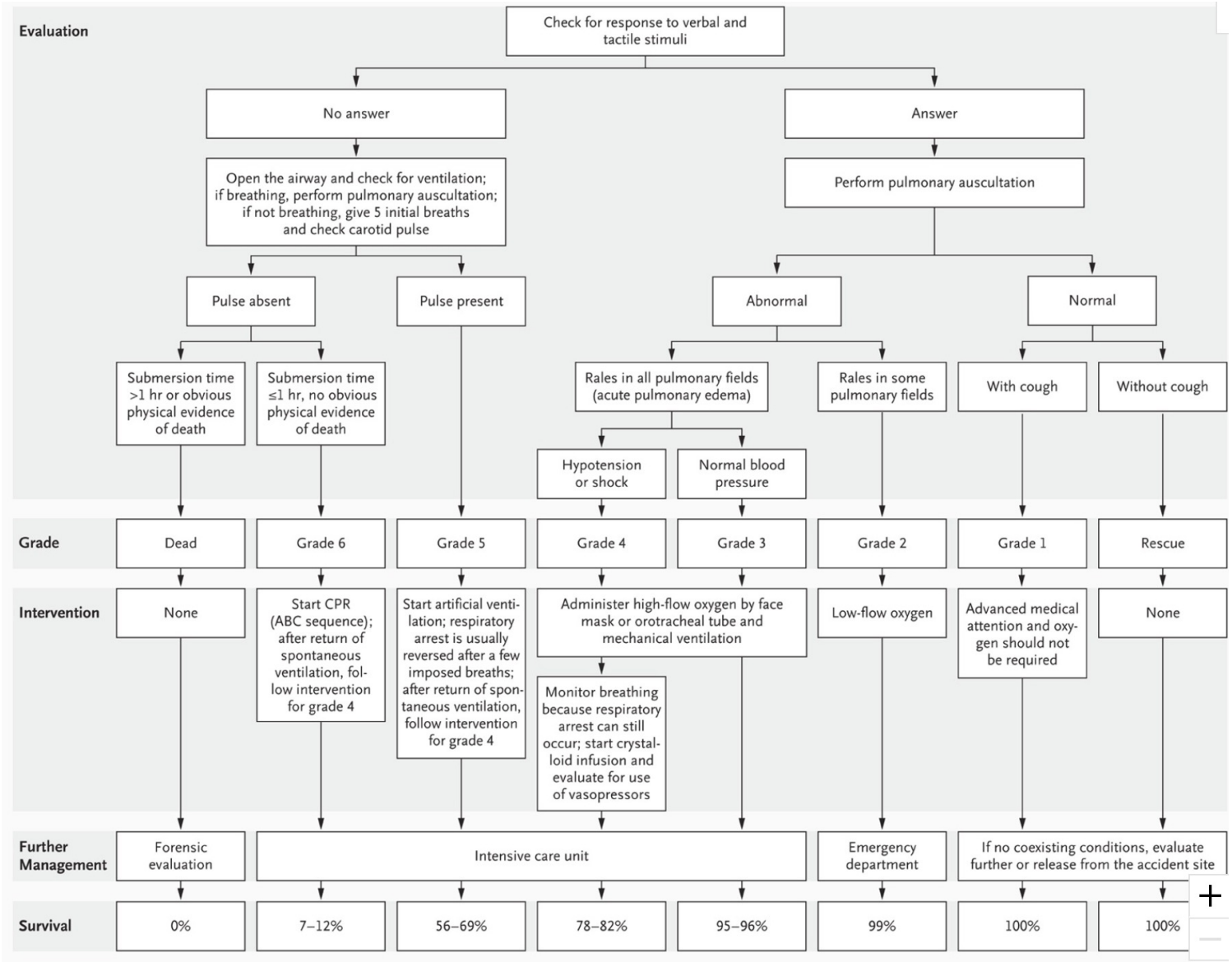
- prevent secondary brain injury.

RESP: ALI and ARDS -> protective lung ventilation, bronchodilation, iNO, prone, ECMO

METABOLIC: severe metabolic acidosis from lactate, in vivo PaO_2 in cold patient is much lower than the measured value as it is warmed to 37 C, rhabdomyolysis

CARDIOVASCULAR: below 28 C VF is common, extravasation of systemic and pulmonary capillaries + cold diuresis -> hypovolaemia, SIRS post resuscitation, often require cardiac output monitoring

INFECTION: consider antibiotics if patient submerged in grossly contaminated water


Evaluation
Check for response to verbal and tactile stimuli
No answer
Answer
Open the airway and check for ventilation; if breathing, perform pulmonary auscultation; if not breathing, give 5 initial breaths and check carotid pulse
Perform pulmonary auscultation
Pulse absent
Pulse present
Abnormal
Normal
Submersion time >1 hr or obvious physical evidence of death
Submersion time ≤1 hr, no obvious physical evidence of death
Rales in all pulmonary fields (acute pulmonary edema)
Rales in some pulmonary fields
With cough
Without cough
Hypotension or shock
Normal blood pressure
Grade
Dead
Grade 6
Grade 5
Grade 4
Grade 3
Grade 2
Grade 1
Rescue
Intervention
None
Start CPR (ABC sequence); after return of spontaneous ventilation, follow intervention for grade 4
Start artificial ventilation; respiratory arrest is usually reversed after a few imposed breaths; after return of spontaneous ventilation, follow intervention for grade 4
Administer high-flow oxygen by face mask or orotracheal tube and mechanical ventilation
Monitor breathing because respiratory arrest can still occur; start crystalloid infusion and evaluate for use of vasopressors
Low-flow oxygen
Advanced medical attention and oxygen should not be required
None
Further Management
Forensic evaluation
Intensive care unit
Emergency department
If no coexisting conditions, evaluate further or release from the accident site
Survival
0%
7–12%
56–69%
78–82%
95–96%
99%
100%
100%

Drowning - Pathophysiology

- In survivors the longterm morbidity reflects the severity and duration of cerebral anoxia experienced
- surfactant dysfunction and washout
- osmotic gradient damages alveolar–capillary membrane: disrupts the integrity of the membrane, increases its permeability, and exacerbates fluid, plasma, and electrolyte shifts
- often massive bloodstained pulmonary edema
- results in decreased lung compliance,V/Q mismatch, atelectasis and bronchospasm

CPAP for drowned patient??

Drowning – sea water vs fresh water

no significant differences in electrolytes abnormalities or degree of lung injury despite differences in osmotic gradient

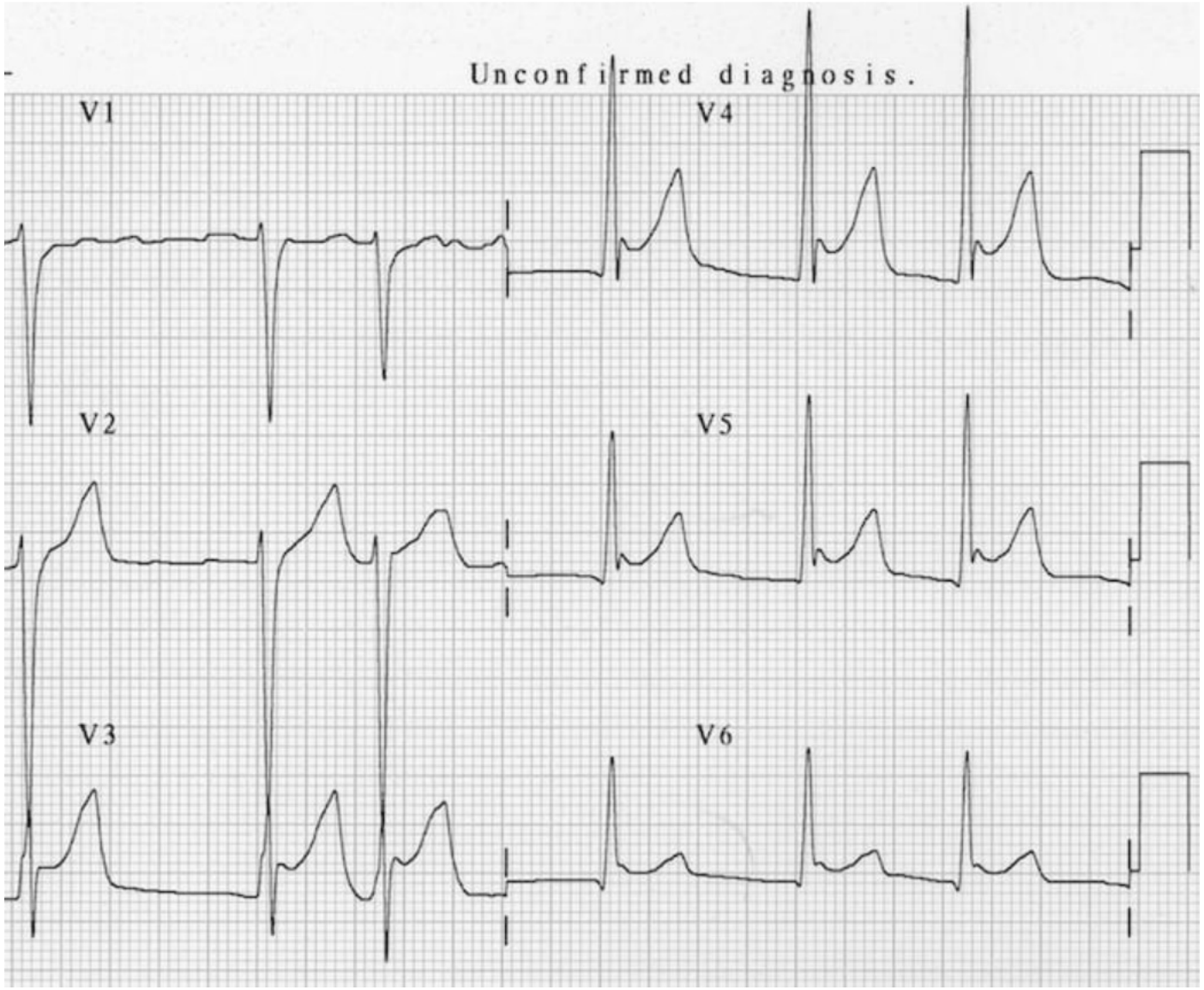
bacterial burden is greater in fresh water (gram negatives, anaerobes, Staphylococci, fungi, algae, protozoans, Aeromonas)

“dry drowning”

- Laryngospasm due to water inhalation

“secondary drowning”

- Delayed pulmonary oedema due to water inhalation
- Can occur up to 24 hours
- rare


Unconfirmed diagnosis.
V1
V4
V2
V5
V3
V6

Hypothermia

hypothermia associated with drowning can provide a protective mechanism that allows persons to survive prolonged submersion episodes

rate of cerebral oxygen consumption is reduced by ~ 5% for each reduction of 1°C in temperature within the range of 37°C to 20°C

Prognosis: Prehospital

- immersion > 10 min
- delay to CPR (e.g. no bystander CPR, unwitnessed)
- time to first breath
- water temperature (drop in brain temperature 10C doubles time that brain can survive)
- presence of cardiac arrest (pulseless or absence of respiratory effort upon rescue)
- identifiable precipitants; e.g. did the person have a cardiac arrest secondary to an AMI while in the swimming pool?

Prognosis: ED

- asystole
- CPR > 25 minutes
- dilated, non-reactive pupils and pH <7.0
- dilated, non-reactive pupils and GCS < 5
- lactate

Delayed Drowning?

- ?observe for 24 hrs
  - based on largely unsubstantiated fear of “secondary drowning” or delayed respiratory deterioration secondary to surfactant disruption
- Normal exam and normal O2 sats at 4-6 hrs post injury
- Low threshold for admitting patients overnight if there is any suggestion of respiratory compromise however minor
  - or if they have had symptoms which have required salbutamol for resolution

Happy diving!!