Medical Care

The ABC’s of Assessment and Treatment

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*Open the airway!
Mallampati Score

1. 100%
2. 50%
3. soft palate
4. hard palate

*What’s the score?
*The Difficult Airway
What’s your nightmare airway?
Look externally
- Abnormal facies
- Facial hair
- Morbid obesity
- Dentition
- Tongue
- Facial/neck trauma
Look
Evaluate
Mallampati Score
Obstructions
Neck
### What is in your toolbox?

**Pre-oxygenation for FOUR minutes**
**Section checked working & available**
**Patient Positioned? RAMP 0600SE**

**IV & DRUGS**
- IV Cannula connected to fluid & running: CHECK
- NIBP on contralateral arm and BP seen: CHECK
- Spare cannula in situ: CHECK
- INDUCTION AGENT drawn up, dose checked: CHECK
- Sux or Roc drawn up, dose checked: CHECK
- VASOPRESSORS drawn up, labelled: CHECK
- POST INTUBATION drugs drawn up & labelled: CHECK

**INTUBATION EQUIPMENT**
- BVM connected to oxygen: CHECK
- Guedel & two NPO airways available: CHECK
- Laryngoscope blade chosen, light working: CHECK
- ET tube size chosen, cuff tested: CHECK
- ETT preloaded on bougie, Kiwi Grip: CHECK
- Alternate tube size chosen & cuff tested: CHECK
- Syringe for cuff inflation: CHECK
- Stylet & Rapi-Fit Bougie connectors available: CHECK
- Geosensec, filter, inline ETCO2: CHECK
- Tube Tie & Tape available: CHECK
- Ventilator settings determined: CHECK
- Difficult airway plan’s A, B, C, D discussed: CHECK
- LMA, ILMA and Surgical Airway available: CHECK

**TEAM BRIEF**
- In-line immobilisation person briefed: CHECK
- Cricoid pressure person briefed: CHECK
- Drug giver briefed: CHECK
- Anticipated problems & post RSI care brief: CHECK
A- Initial Intubation strategy
B- Secondary Intubation strategy
C- Maintain oxygenation/ventilation
D- Rescue techniques for ‘can’t intubate, can’t ventilate’

What are your Plans?
* Position head
* Bouge
* Alternative blade
* Video laryngoscope
* LMA
* King LTd
* Combitube?
* Fiberoptic stylet

* Plan B
BVM
OPA/NPA

Plan C - Maintain oxygenation & Ventilation
* Needle cric
* Surgical cric
* Get them to the ED!

* Plan D- Can’t ventilate
*Other Difficult Airways
* Difficulty with BVM
* Usually sicker (diabetes, restrictive lung disease, obstructive sleep apnea with pulmonary hypertension, arthritis, CAD, Hypertension)
* Altered medication kinetics
* Increased metabolic demand³
* Functional residual capacity (FRC). They desaturate much more quickly (3 min compared to 6 min)

*Bariatric Patients*
* Who are Obligate Nasal Breathers, Have Large Tongues, Large Heads, Larynx, and funnel shaped Tracheas?

Next...
Pediatric Patients!
Anatomy of the Pediatric Airway

- Relatively greater proportion of soft tissue
- Larynx more superior and anterior
- Epiglottis rounder and floppier
- Smaller jaw
- Cricoid cartilage - narrowest part of the pediatric airway
- Loosely attached mucous membranes
Figure 1. Comparison Of Infant And Adult Airway

*Padding under shoulders*
Face to Face Intubation?
This can also be used with two people
Perhaps a better adjunct is that of external laryngeal manipulation. CP will NOT prevent aspiration...and indeed this is not what kills people...whereas hypoxia will.

*Cricoid Pressure - Friend or foe?
SAD NEWS

Please join me in remembering YET ANOTHER great icon of the entertainment community. The Pillsbury Dough Boy died yesterday of a yeast infection and traumatic complications from repeated pokes to the belly. He was 71. Dough Boy is survived by his wife, Play Dough, three children, John Dough, Jane Dough and Dough, plus they had one in the oven. Service were held at 3:50 for about 20 minutes
*Breathing Assessment
  * Spontaneous Breathing
  * Rate & Pattern
  * Symmetrical rise and fall
  * Increased work of breathing (Nasal flaring, retractions)
  * Accessory muscle use
  * Chest wall stability/Integrity
  * Skin Color
  * Pulse oximetry (>94%)
* Examine for life-threatening hemorrhage

* Assess perfusion (level of consciousness, skin color, pulse rate and blood pressure

* Assess the pulse manually - is it regular or irregular, what is the rate (15 seconds x 4), skin color, temperature, central and peripheral cap refill.
* The immune response that causes an allergic reaction is similar to the response that causes hay fever. Most reactions happen soon after contact with an allergen.

* Many allergic reactions are mild, while others can be severe and life-threatening. They can be confined to a small area of the body, or they may affect the entire body. The most severe being anaphylaxis or anaphylactic shock.

* First-time exposure may produce only a mild reaction. Repeated exposures may lead to more serious reactions. Once a person has had an exposure or an allergic reaction (is sensitized), even a very limited exposure to a very small amount of allergen can trigger a severe reaction.

* Most severe allergic reactions occur within seconds or minutes after exposure to the allergen. Some reactions can occur after several hours, particularly if the allergen causes a reaction after it has been eaten. In very rare cases, reactions develop after 24 hours.

* **Allergic Reactions**
* Allergen: An environmental substance that can produce a hypersensitive allergic reaction in the body but may not be intrinsically harmful.

* Anaphylactic reaction: An acute allergic response triggered by IgE-mediated antigen-stimulated mast cell activation resulting in histamine release.

* Anaphylactic shock: A severe and sometimes fatal systemic allergic reaction to a sensitizing substance, such as a drug, vaccine, specific food, serum, insect venom or chemical.

* Key Terms
* Anaphylaxis: An exaggerated, life-threatening hypersensitivity reaction to a previously encountered antigen.

* Basophil: A granulocytic white blood cell that represent 1% or less of the total white blood cell count. The relative number of basophils increases in severe allergic reactions.

* Bronchodilation: A widening of the lumen of the bronchi, allowing increased airflow to and from the lungs.

* Mast cells: A constituent of connective tissue containing large basophilic granules that contain heparin, serotonin, bradykinin and histamine.
Treatment for anaphylactic reactions includes oxygenation, airway support, and medications, such as nebulized albuterol. Additional medications, including epinephrine, diphenhydramine, dopamine and dosed fluid boluses, may also be required. Maintenance of a patient’s oxygenation levels and airway support are of primary concern. Maintaining oxygen levels is often the key to successful resuscitation efforts, although it may present one of the largest challenges.
Heat Emergencies
Despite wide variations in ambient temperatures, humans and other mammals can maintain a constant body temperature by balancing heat gain with heat loss. When heat gain overwhelms the body's mechanisms of heat loss, the body temperature rises, potentially leading to heatstroke. Excessive heat denatures proteins, destabilizes phospholipids and lipoproteins, and liquefies membrane lipids, leading to cardiovascular collapse, multi-organ failure, and, ultimately, death.

*Heat Emergencies*
The exact temperature at which cardiovascular collapse occurs varies among individuals because coexisting disease, drugs, and other factors may contribute to or delay organ dysfunction. Full recovery has been observed in patients with temperatures as high as 46°C, and death has occurred in patients with much lower temperatures. Temperatures exceeding 106°F or 41.1°C generally are catastrophic and require immediate aggressive therapy.
Heat may be acquired by a number of different mechanisms. At rest, basal metabolic processes produce approximately 100 kcal of heat per hour or 1 kcal/kg/h. These reactions can raise the body temperature by 1.1 °C/h if the heat-dissipating mechanisms are nonfunctional. Strenuous physical activity can increase heat production more than 10-fold, to levels exceeding 1000 kcal/h. Similarly, fever, shivering, tremors, convulsions, thyrotoxicosis, sepsis, sympathomimetic drugs, and many other conditions can increase heat production, thereby increasing body temperature.
The body also can acquire heat from the environment through some of the same mechanisms involved in heat dissipation, including conduction, convection, and radiation. These mechanisms occur at the level of the skin and require a properly functioning skin surface, sweat glands, and autonomic nervous system, but they also may be manipulated by behavioral responses.
Conduction refers to the transfer of heat between 2 surfaces with differing temperatures that are in direct contact. Convection refers to the transfer of heat between the body's surface and a gas or fluid with a differing temperature. Radiation refers to the transfer of heat in the form of electromagnetic waves between the body and its surroundings. The efficacy of radiation as a means of heat transfer depends on the position of the sun, the season, clouds, and other factors. For example, during summer, lying down in the sun can result in a heat gain of up to 150 kcal/h.
As the major heat-dissipating organ, the skin can transfer heat to the environment through conduction, convection, radiation, and evaporation. Radiation is the most important mechanism of heat transfer at rest in temperate climates, accounting for 65% of heat dissipation, and it can be modulated by clothing. At high ambient temperatures, conduction becomes the least important of the 4 mechanisms, while evaporation, which refers to the conversion of a liquid to a gaseous phase, becomes the most effective mechanism of heat loss.
When heat gain exceeds heat loss, the body temperature rises. Classic heatstroke occurs in individuals who lack the capacity to modulate the environment (e.g., infants, elderly individuals, individuals who are chronically ill). Furthermore, elderly persons and patients with diminished cardiovascular reserves are unable to generate and cope with the physiologic responses to heat stress and, therefore, are at risk of heatstroke. Patients with skin diseases and those taking medications that interfere with sweating also are at increased risk for heatstroke because they are unable to dissipate heat adequately. Additionally, the redistribution of blood flow to the periphery, coupled with the loss of fluids and electrolytes in sweat, place a tremendous burden on the heart, which ultimately may fail to maintain an adequate cardiac output, leading to additional morbidity and mortality.
Factors that interfere with heat dissipation include an inadequate intravascular volume, cardiovascular dysfunction, and abnormal skin. Additionally, high ambient temperatures, high ambient humidity, and many drugs can interfere with heat dissipation, resulting in a major heat illness. Similarly, hypothalamic dysfunction may alter temperature regulation and may result in an unchecked rise in temperature and heat illness.
On a cellular level, heat directly influences the body by interfering with cellular processes along with denaturing proteins and cellular membranes. In turn, an array of inflammatory cytokines, interleukins and heat shock proteins (HSPs) are produced. In particular, HSP-70 allows the cell to endure the stress of its environment. If the stress continues, the cell will succumb to the stress (apoptosis) and die.
On a microvascular level, heat stroke resembles sepsis and involves inflammation, translocation of lipopolysaccharides from the gut, and activates the coagulation cascade. Certain preexisting factors, such as age, genetic makeup, and the nonacclimatized individual, may allow progression from heat stress to heatstroke, systemic inflammatory response syndrome (SIRS), multiorgan dysfunction syndrome (MODS), and ultimately death. Progression to heatstroke may occur through thermoregulatory failure, an amplified acute-phase response, and alterations in the expression of HSPs.
Clinically, 2 forms of heatstroke are differentiated: classic, or nonexertional, heatstroke (NEHS) and exertional heatstroke (EHS). Classic heatstroke, which occurs during environmental heat waves, is more common in the very young and the elderly and should be suspected in children, elderly persons, and chronically ill individuals who present with an altered sensorium. Classic heatstroke occurs because of failure of the body's heat dissipating mechanisms.
On the other hand, EHS affects young, healthy individuals who engage in strenuous physical activity, and EHS should be suspected in all such individuals who exhibit bizarre, irrational behavior or experience syncope. EHS results from increased heat production, which overwhelms the body's ability to dissipate heat.
EHS is characterized by hyperthermia, diaphoresis, and an altered sensorium, which may manifest suddenly during extreme physical exertion in a hot environment.

A number of symptoms (eg, abdominal and muscular cramping, nausea, vomiting, diarrhea, headache, dizziness, dyspnea, weakness) commonly precede the heatstroke and may remain unrecognized. Syncope and loss of consciousness also are observed commonly before the development of EHS.
Risk factors that increase the likelihood of heat-related illnesses include a preceding viral infection, dehydration, fatigue, obesity, lack of sleep, poor physical fitness, and lack of acclimatization. Although lack of acclimatization is a risk factor for heatstroke, EHS also can occur in acclimatized individuals who are subjected to moderately intense exercise. EHS also may occur because of increased motor activity due to drug use, such as cocaine and amphetamines, and as a complication of status epilepticus.
Nonexertional heatstroke
NEHS is characterized by hyperthermia, anhidrosis, and an altered sensorium, which develop suddenly after a period of prolonged elevations in ambient temperatures (ie, heat waves). Core body temperatures greater than 41°C are diagnostic, although heatstroke may occur with lower core body temperatures.
Numerous central nervous system (CNS) symptoms, ranging from minor irritability to delusions, irrational behavior, hallucinations, and coma have been described. Other possible CNS symptoms include seizures, cranial nerve abnormalities, cerebellar dysfunction, and opisthotonos.

Anhidrosis due to cessation of sweating is a late occurrence in heatstroke and may not be present when patients are examined.

Patients with NEHS initially may exhibit a hyperdynamic circulatory state, but, in severe cases, hypodynamic states may be noted.
Classic heatstroke most commonly occurs during episodes of prolonged elevations in ambient temperatures. It affects people who are unable to control their environment and water intake (eg, infants, elderly persons, individuals who are chronically ill), people with reduced cardiovascular reserve (eg, elderly persons, patients with chronic cardiovascular illnesses), and people with impaired sweating (eg, from skin disease or ingestion of anticholinergic or psychiatric drugs). In addition, infants have an immature thermoregulatory system, and elderly persons have impaired perception of changes in body and ambient temperatures and a decreased capacity to sweat.
Vital signs
Temperature: Typically, the patient's temperature exceeds 41 °C. However, in the presence of sweating, evaporating mechanisms, and the initiation of cooling methods, body temperatures lower than 41 °C are common.

Pulse: Tachycardia to rates exceeding 130 beats per minute is common.

Blood pressure: Patients commonly are normotensive, with a wide pulse pressure; however, hypotension is common and may result from a number of factors, including vasodilation of the cutaneous vessels, pooling of the blood in the venous system, and dehydration. Hypotension also may be due to myocardial damage and may signal cardiovascular collapse. Blood pressure will usually correct with normalization of the body temperature.
Central nervous system
Symptoms of CNS dysfunction are present universally in persons with heatstroke. Symptoms may range from irritability to coma.

Patients may present with delirium, confusion, delusions, convulsions, hallucinations, ataxia, tremors, dysarthria, and other cerebellar findings, as well as cranial nerve abnormalities and tonic and dystonic contractions of the muscles. Patients also may exhibit decerebrate posturing, decorticate posturing, or they may be limp.

Coma also may be caused by electrolyte abnormalities, hypoglycemia, hepatic encephalopathy, uremic encephalopathy, and acute structural abnormalities, such as intracerebral hemorrhage due to trauma or coagulation disorders.

Cerebral edema and herniation also may occur during the course of heatstroke
The etiology of heatstroke may involve any of the following:

Increased heat production
Decreased heat loss
Reduced ability to acclimatize
Reduced behavioral responsiveness
* Increased heat production
* Increased metabolism can result from any of the following:

* Infections
* Sepsis
* Encephalitis
* Stimulant drugs
* Thyroid storm
* Drug withdrawal
Increased muscular activity may involve any of the following:

Exercise
Convulsions
Tetanus
Strychnine poisoning
Sympathomimetics
Drug withdrawal
Thyroid storm
Stimulant drugs, including cocaine and amphetamines, can generate excessive amounts of heat by increasing metabolism and motor activity through the stimulatory effects of dopamine, serotonin, and norepinephrine. The development of heatstroke in individuals intoxicated with stimulants is multifactorial and may involve a complex interaction between dopamine and serotonin in the hypothalamus and the brainstem.
* Cocaine Toxicity
* Delirium
* Delirium Tremens (DTs)
* Hepatic Encephalopathy
* Hyperthyroidism
* Meningitis
Once heatstroke is suspected, cooling must begin immediately and must be continued during the patient's resuscitation. The American College of Sports Medicine recommends that cooling be initiated at the scene, before transporting the patient to an emergency department for further evaluation and treatment. [5] Despite extensive education and training, delays are still reported due to trepidation by athletic trainers to accurately diagnose and rapidly initiate treatment for EHS. [6]
* In a review of 19 clinical trials and observational studies involving 556 patients, the conduction method of cooling was found to be more efficacious in young, active adults with EHS. Unfortunately, this review did not identify a preferred treatment found for NEHS, or a temperature endpoint to prevent overcooling.[9]

* Removal of restrictive clothing and spraying water on the body, covering the patient with ice water-soaked sheets, or placing ice packs in the axillae and groin may reduce the patient’s temperature significantly. Patients who are unable to protect their airway should be intubated. Patients who are awake and responsive should receive supplemental oxygen.
* Muscle necrosis may occur so rapidly that hyperkalemia, hypocalcemia, and hyperphosphatemia become significant enough to cause cardiac arrhythmias and require immediate therapy. In the presence of renal failure, hemodialysis may be necessary.
* In patients with heatstroke, benzodiazepines play a major role in providing sedation, controlling convulsions, and controlling shivering. Barbiturates (eg, phenobarbital) may be used to control convulsions if benzodiazepines are not effective.

* Hypotension is treated first with cooling and intravenous crystalloid fluids; dobutamine is considered if patients are hypodynamic. Treatment of rhabdomyolysis involves infusing large amounts of intravenous fluids (as much as 10 L may be required), alkalinization of urine, and mannitol infusion.
* Midazolam (Versed)

* Midazolam is a rapidly acting benzodiazepine with a short duration of action. It is ideal for sedation during short procedures and may be effective in convulsions.
Sodium bicarbonate (Neut)

Sodium bicarbonate is useful in alkalization of the urine to prevent acute myoglobinuric renal failure. It may be administered as a bolus injection or as an infusion. The ideal solution to which sodium bicarbonate is added should be hypotonic.
Mannitol (Osmitrol)

Mannitol is the drug of choice for forced diuresis in patients with rhabdomyolysis because of a number of beneficial effects on the kidneys, including an antioxidant effect.
* Adrenergic agonist agents produce vasodilation and increase the inotropic state.

* View full drug information

* Dobutamine (Dobutrex)

* Dobutamine is a synthetic compound structurally similar to catecholamines. It is the drug of choice for circulatory support in heatstroke.
* Cold Emergencies
TAC= 15:04
29 YEAR OLD MALE
(30A2) - NON-EMERGENCY, Non-recent (>=6 hrs) injuries (without priority symptoms)

PT STATES SOMEONE USED A FROZEN DILDO ON HIM 2 WEEKS AGO
911 CALL - SPRINT NEXTEL-IDEN 84
LAPD TOPANGA DIV, QUERY CALLER
ORIGINAL W911 INFO (15:01:49) -
RING AV, P: 323-9495
LON:-118.5422, LAT:34
Where do we see cold emergencies? Everywhere!
* Localized cold injury
* Subfreezing temperatures
* Vasoconstriction occurs
  * ↓ Blood flow to distal circulation (nose, ears, fingers, toes)
  * Water in tissues freezes; tissue damage occurs

*Frostbite*
* Mild (frost nip): Red, burning areas
* Superficial: White, waxy, doughy-feeling
* Deep: Dead white, hard, no sensation

**Frostbite Signs/Symptoms**
* Remove from cold
* Dry areas gently, wrap in sterile dressing
* Transport
* If transport prolonged, rewarmed rapidly in 100-105°F water
* Do NOT rub frostbite
* Do NOT allow refreezing
* Do NOT allow patient to smoke
* Generalized cooling of body
* Can occur at temperatures above freezing

Hypothermia
* Homeless
* Alcoholics
* Elderly living in poorly heated homes
* Outdoor sports participants
* ↓ LOC + Cool Environment equals Hypothermia until proven otherwise
* Support airway, breathing
* 100% O₂--warmed, if possible
* Prevent further heat loss
* Do not aggressively rewarm
Hypothermia can cause apparent absence of vital signs

Always resuscitate

You’re not dead until you’re warm and dead!
Chest Pain
Cardiovascular Compromise
Cardiac arrest is the cessation of cardiac mechanical activity resulting in the absence of circulating blood flow. Cardiac arrest stops blood from flowing to vital organs, depriving them of oxygen, and, if left untreated, results in death. Sudden cardiac arrest is the unexpected cessation of circulation within a short period of symptom onset (sometimes without warning). Sudden cardiac arrest occurs outside the hospital in about 400,000 people/yr in the US, with a > 90% mortality.
* Sudden cardiac arrest results primarily from cardiac disease (of all types, but especially coronary artery disease). In a significant percentage of patients, sudden cardiac arrest is the first manifestation of heart disease. Other causes include circulatory shock due to noncardiac disorders (especially pulmonary embolism, GI hemorrhage, or trauma), ventilatory failure, and metabolic disturbance (including drug overdose).

**Adult Cardiac Arrest**
Cardiac causes of sudden cardiac arrest are much less common (< 15 to 20%). Instead, predominant causes include trauma, poisoning, and various respiratory disorders (e.g., airway obstruction, smoke inhalation, drowning, infection, SIDS).
Global ischemia due to lack of blood flow occur at the cellular level that adversely affect organ function after resuscitation.

Direct consequences are cellular damage and edema formation.

Edema is particularly harmful in the brain, which has minimal room to expand, and often results in increased intracranial pressure and corresponding decreased cerebral perfusion post-resuscitation.

*Pathophysiology*
What happens when you suddenly stop!
Good Compressions

* Push Hard
* Push Fast
* Minimize interruptions
* Allow chest wall recoil

Good Compressions

* 2-2.4”
* 100-120 per minute
* <10 seconds every 2 min
* Change compressors every 2 minutes

So What Can We Do?
* For witnessed OHCA with a shockable rhythm, it may be reasonable for EMS systems with priority-based, multi-tiered response to delay positive-pressure ventilation by using a strategy of up to 3 cycles of 200 continuous compressions with *passive* oxygen insufflation and airway adjuncts.

*Delayed Ventilation?
* 3 cycles of passive oxygenation
* Airway adjunct insertion
* 200 continuous chest compressions with interposed shocks.

* The “Bundled” package
*Mechanical Chest Compression Devices*
*Epinephrine v. Vasopressin

*The great debate
*What’s next???
*Bystander Naloxone*
* Any Questions?