Post Resuscitative Care

Metabolic Control

Dr. Abdulhadi A. Tashkandi

MD, FRCPC

Assistant Prof. Emergency Medicine

Chairman Emergency Department - National Guard Hospital

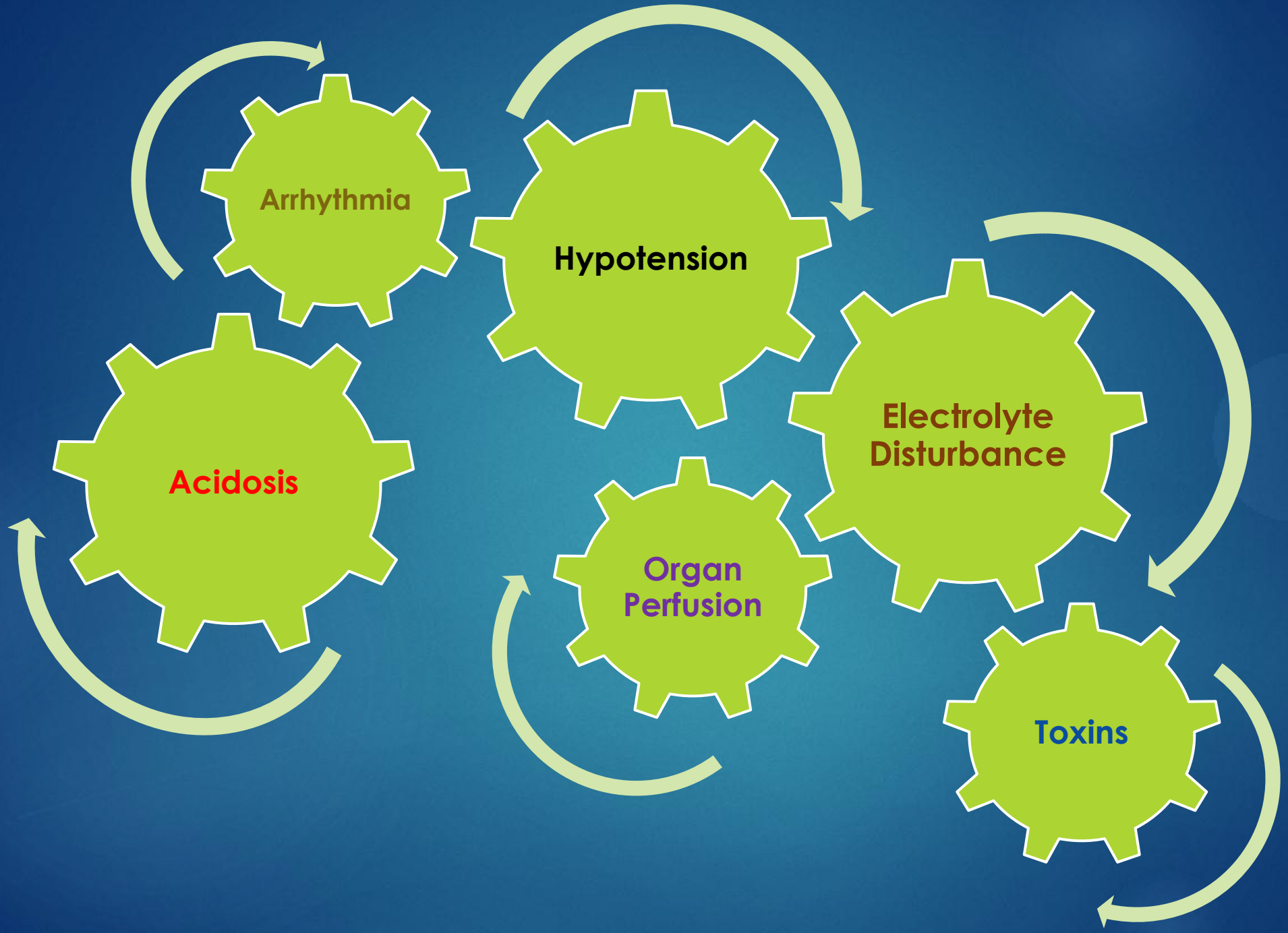
Madinah - Saudi Arabia

2nd Intercontinental Emergency Medicine Congress

EPAT Emergency Physicians Association of Turkey

GOALS OF CARE

- Must address multiple major problems simultaneously.
- **Including:**
  - Determining and treating the cause of arrest
  - Managing cardiovascular dysfunction
  - Maintain end-organ perfusion
    - Minimizing brain injury other related 2ry injuries
  - Managing problems that may arise from global ischemia, reperfusion injury & related to any intervention (Therapeutic Hypothermia)


Arrhythmia
Hypotension
Acidosis
Electrolyte Disturbance
Organ Perfusion
Toxins

Following ROSC

- Most patients require central venous access, arterial access, and potentially other invasive procedures
  - given the need for frequent arterial blood gas measurements and the common use of vasopressor and inotropic drugs.

•Reversible myocardial dysfunction in survivors of out-of-hospital cardiac arrest. J Am Coll Cardiol 2002.

Maintaining end-organ perfusion

- Determination of the etiology of cardiac arrest and the initiation of relevant treatments are performed concurrently with resuscitation efforts in order to prevent recurrent arrest and optimize outcome.
- The most immediate threat to survival during the first minutes to hours is cardiovascular collapse.

- An adequate blood pressure must be maintained in the post-cardiac arrest patient.
- Episodes of hypotension can cause secondary injury.
- Interventions to optimize blood pressure and maintain end-organ perfusion

  (IV fluid, vasopressors & inotropes)

May help prevent secondary injury from hypotension.

Maintaining end-organ perfusion

- **Goal:**
  - Mean arterial blood pressure (MAP) should be >65 mmHg to reverse the acute shock state,
    - preferably 80 -100 mmHg to optimize cerebral perfusion.
  - Maintaining CVP between 8 -12 mmHg and
  - adequate urine output (>0.5 mL/kg /hour).

- Early goal-directed hemodynamic optimization combined with therapeutic hypothermia in comatose survivors of out-of-hospital cardiac arrest. Resuscitation 2009.
- The development and implementation of cardiac arrest centers. Resuscitation 2011.

Maintaining end-organ perfusion

- **Preventing arrhythmia**
  - Determining and correcting the underlying cause of the arrhythmia (eg, electrolyte disturbance, acute myocardial ischemia, toxin ingestion) is the best intervention.
  - Antiarrhythmic drugs should be reserved for patients with recurrent or ongoing unstable arrhythmias.
    - No data support the routine or prophylactic use of antiarrhythmic drugs after the return of spontaneous circulation following cardiac arrest, even if such medications were employed during the resuscitation.

- Additional short-term of care include:
  - optimizing oxygenation and ventilation
  - correcting electrolyte abnormalities.

Post resuscitation

- **Metabolic management play very important role**
  - R/O metabolic cause as a CAUSE of arrest
  - Glycemic control
  - Monitoring any electrolyte abnormalities
  - Prognostic factor
    - Monitoring lactate clearance
  - Consequences of interventions
    - e.g. Therapeutic Hypothermia
  - 2ry organ injuries
    - Liver & Kidney

Following ROSC

- ABG measurements are obtained every 6hrs to assess acid-base status and are used to guide ventilator management (including sodium, potassium, chloride, and bicarbonate).
  - during therapeutic hypothermia (TH) and rewarming
- Serum lactate concentration is measured every 4-6hrs.

Following ROSC

- **Glycemic control**
  - Hyperglycemia is associated with worse outcomes in post-cardiac arrest patients.

- Neurologic outcome and blood glucose levels during out-of-hospital cardiopulmonary resuscitation. Neurology 1986.
- A multiple logistic regression analysis of in-hospital factors related to survival at six months in patients resuscitated from out-of-hospital ventricular fibrillation. Resuscitation 2003.

Following ROSC

- **Glycemic control**
  - Maintain serum glucose between 140 and 180 mg/dL (7.8 and 10 mmol/L) during the period following cardiac arrest, and **avoid hypoglycemic** episodes.

Following ROSC

- **Glycemic control**
  - There is no additional benefit from tight control of the serum glucose (70 to 108 mg/dL; 3.9 to 6 mmol/L) compared to more liberal management (108 to 144 mg/dL; 6 to 8.1 mmol/L) following cardiac arrest.
  - Multiple studies highlight the increased risk of hypoglycemia when lower target ranges are used.

•Strict versus moderate glucose control after resuscitation from ventricular fibrillation. Intensive Care Med 2007.
•NICE-SUGAR Study Investigators, Intensive versus conventional glucose control in critically ill patients. N Engl J Med 2009.

Following ROSC

- **K+**
  - Both high and low potassium can cause arrhythmias and must be treated immediately.
  - Rapid fluctuations in serum potassium may occur as a result of ischemia, acidosis, and catecholamine administration.
  - Hypokalemia is often accompanied by hypomagnesaemia, which should also be corrected.

Following ROSC

- **Serum lactate**
  - Initial lactate concentrations and the rate of lactate clearance correlate with survival.
    - Lactic acidosis, with serum lactate concentrations up to approximately 15 mmol/L, is common after cardiac arrest, but higher levels suggest ongoing intra-abdominal or muscle compartment ischemia.
  - Lactate should clear over time after adequate perfusion is restored.

•Effective lactate clearance is associated with improved outcome in post-cardiac arrest patients. Resuscitation 2007.

Therapeutic Hypothermia (TH)

- Evidence supports the use of therapeutic hypothermia (TH) to minimize brain injury and target body temperatures should be achieved within the first few hours following resuscitation.

Therapeutic Hypothermia (TH)

- Hyperglycemia due to insulin resistance has been noted during TH.
- Large doses of insulin may be needed in severely hyperglycemic patients.

•Therapeutic hypothermia and controlled normothermia in the intensive care unit: practical considerations, side effects, and cooling methods. Crit Care Med 2009.
•Increased blood glucose variability during therapeutic hypothermia and outcome after cardiac arrest. Crit Care Med 2011.

Therapeutic Hypothermia (TH)

- **<u>Hypothermia leads to a “cold diuresis,” which in turn can cause:</u>**
  - hypovolemia,
  - hypokalemia,
  - hypomagnesaemia, and
  - hypophosphatemia.

•Hypophosphatemia and hypomagnesemia induced by cooling in patients with severe head injury. J Neurosurg 2001.

therapeutic hypothermia (TH)

- In addition, temperature fluctuations during the induction of TH and rewarming cause potassium to move between the extracellular and intracellular compartments.
- Hypokalemia is more frequently encountered in patients maintained at 33°c.

•Hypophosphatemia and hypomagnesemia induced by cooling in patients with severe head injury. J Neurosurg 2001.
•Reversible hypophosphatemia during moderate hypothermia therapy for brain-injured patients. Crit Care Med 2001.
•Targeted temperature management at 33°C versus 36°C after cardiac arrest. N Engl J Med 2013.

Therapeutic Hypothermia (TH)

- Therefore, careful monitoring of volume status and measurement of basic electrolytes during temperature manipulation is prudent.

Therapeutic Hypothermia (TH)

- TH slows the metabolism and excretion of many drugs and thus the duration of effect may be prolonged.

•Effects of hypothermia on pharmacokinetics and pharmacodynamics: a systematic review of preclinical and clinical studies. Clin Pharmacokinet 2010.
•The effect of therapeutic hypothermia on drug metabolism and response: cellular mechanisms to organ function. Expert Opin Drug Metab Toxicol 2011.

In summery

- **Post resuscitation efforts toward:**
  - Metabolic management play very important role
    - R/O metabolic cause as a CAUSE of arrest
    - Avoid HYPER & HYPO glycemia
    - Monitor:
      - lactate clearance
      - Electrolyte disturbance as a consequences of interventions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