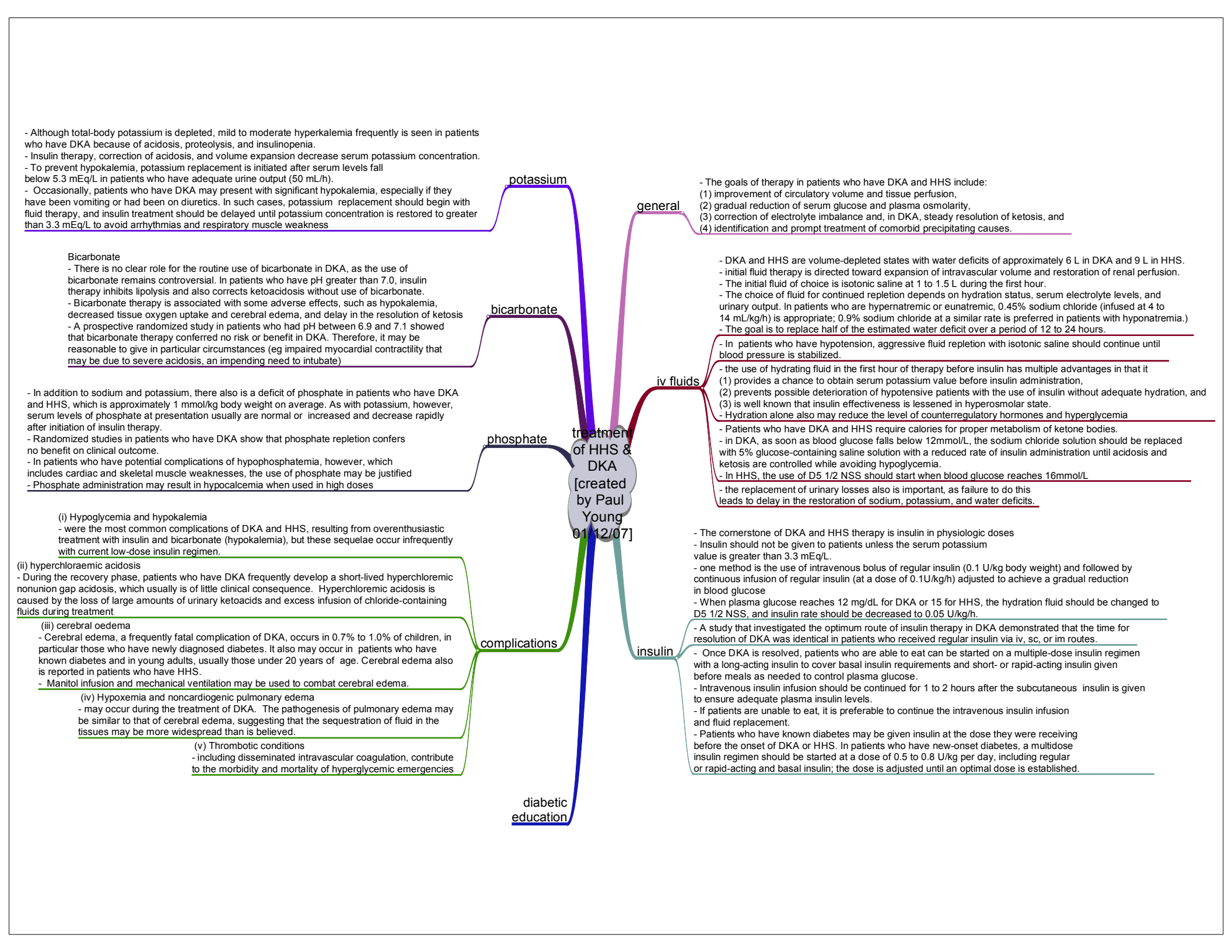
treatment of HHS & DKA [created by Paul Young 01/12/07]
potassium
- Although total-body potassium is depleted, mild to moderate hyperkalemia frequently is seen in patients who have DKA because of acidosis, proteolysis, and insulinopenia.
- Insulin therapy, correction of acidosis, and volume expansion decrease serum potassium concentration.
- To prevent hypokalemia, potassium replacement is initiated after serum levels fall below 5.3 mEq/L in patients who have adequate urine output (50 mL/h).
- Occasionally, patients who have DKA may present with significant hypokalemia, especially if they have been vomiting or had been on diuretics. In such cases, potassium replacement should begin with fluid therapy, and insulin treatment should be delayed until potassium concentration is restored to greater than 3.3 mEq/L to avoid arrhythmias and respiratory muscle weakness
bicarbonate
Bicarbonate
- There is no clear role for the routine use of bicarbonate in DKA, as the use of bicarbonate remains controversial. In patients who have pH greater than 7.0, insulin therapy inhibits lipolysis and also corrects ketoacidosis without use of bicarbonate.
- Bicarbonate therapy is associated with some adverse effects, such as hypokalemia, decreased tissue oxygen uptake and cerebral edema, and delay in the resolution of ketosis
- A prospective randomized study in patients who had pH between 6.9 and 7.1 showed that bicarbonate therapy conferred no risk or benefit in DKA. Therefore, it may be reasonable to give in particular circumstances (eg impaired myocardial contractility that may be due to severe acidosis, an impending need to intubate)
phosphate
- In addition to sodium and potassium, there also is a deficit of phosphate in patients who have DKA and HHS, which is approximately 1 mmol/kg body weight on average. As with potassium, however, serum levels of phosphate at presentation usually are normal or increased and decrease rapidly after initiation of insulin therapy.
- Randomized studies in patients who have DKA show that phosphate repletion confers no benefit on clinical outcome.
- In patients who have potential complications of hypophosphatemia, however, which includes cardiac and skeletal muscle weaknesses, the use of phosphate may be justified
- Phosphate administration may result in hypocalcemia when used in high doses
complications
(i) Hypoglycemia and hypokalemia
- were the most common complications of DKA and HHS, resulting from overenthusiastic treatment with insulin and bicarbonate (hypokalemia), but these sequelae occur infrequently with current low-dose insulin regimen.
(ii) hyperchloraemic acidosis
- During the recovery phase, patients who have DKA frequently develop a short-lived hyperchloremic nonunion gap acidosis, which usually is of little clinical consequence. Hyperchloremic acidosis is caused by the loss of large amounts of urinary ketoacids and excess infusion of chloride-containing fluids during treatment
(iii) cerebral oedema
- Cerebral edema, a frequently fatal complication of DKA, occurs in 0.7% to 1.0% of children, in particular those who have newly diagnosed diabetes. It also may occur in patients who have known diabetes and in young adults, usually those under 20 years of age. Cerebral edema also is reported in patients who have HHS.
- Manitol infusion and mechanical ventilation may be used to combat cerebral edema.
(iv) Hypoxemia and noncardiogenic pulmonary edema
- may occur during the treatment of DKA. The pathogenesis of pulmonary edema may be similar to that of cerebral edema, suggesting that the sequestration of fluid in the tissues may be more widespread than is believed.
(v) Thrombotic conditions
- including disseminated intravascular coagulation, contribute to the morbidity and mortality of hyperglycemic emergencies
diabetic education
general
- The goals of therapy in patients who have DKA and HHS include:
(1) improvement of circulatory volume and tissue perfusion,
(2) gradual reduction of serum glucose and plasma osmolarity,
(3) correction of electrolyte imbalance and, in DKA, steady resolution of ketosis, and
(4) identification and prompt treatment of comorbid precipitating causes.
iv fluids
- DKA and HHS are volume-depleted states with water deficits of approximately 6 L in DKA and 9 L in HHS.
- initial fluid therapy is directed toward expansion of intravascular volume and restoration of renal perfusion.
- The initial fluid of choice is isotonic saline at 1 to 1.5 L during the first hour.
- The choice of fluid for continued repletion depends on hydration status, serum electrolyte levels, and urinary output. In patients who are hypernatremic or eunatremic, 0.45% sodium chloride (infused at 4 to 14 mL/kg/h) is appropriate; 0.9% sodium chloride at a similar rate is preferred in patients with hyponatremia.)
- The goal is to replace half of the estimated water deficit over a period of 12 to 24 hours.
- In patients who have hypotension, aggressive fluid repletion with isotonic saline should continue until blood pressure is stabilized.
- the use of hydrating fluid in the first hour of therapy before insulin has multiple advantages in that it
(1) provides a chance to obtain serum potassium value before insulin administration,
(2) prevents possible deterioration of hypotensive patients with the use of insulin without adequate hydration, and
(3) is well known that insulin effectiveness is lessened in hyperosmolar state.
- Hydration alone also may reduce the level of counterregulatory hormones and hyperglycemia
- Patients who have DKA and HHS require calories for proper metabolism of ketone bodies.
- in DKA, as soon as blood glucose falls below 12mmol/L, the sodium chloride solution should be replaced with 5% glucose-containing saline solution with a reduced rate of insulin administration until acidosis and ketosis are controlled while avoiding hypoglycemia.
- In HHS, the use of D5 1/2 NSS should start when blood glucose reaches 16mmol/L
- the replacement of urinary losses also is important, as failure to do this leads to delay in the restoration of sodium, potassium, and water deficits.
insulin
- The cornerstone of DKA and HHS therapy is insulin in physiologic doses
- Insulin should not be given to patients unless the serum potassium value is greater than 3.3 mEq/L.
- one method is the use of intravenous bolus of regular insulin (0.1 U/kg body weight) and followed by continuous infusion of regular insulin (at a dose of 0.1U/kg/h) adjusted to achieve a gradual reduction in blood glucose
- When plasma glucose reaches 12 mg/dL for DKA or 15 for HHS, the hydration fluid should be changed to D5 1/2 NSS, and insulin rate should be decreased to 0.05 U/kg/h.
- A study that investigated the optimum route of insulin therapy in DKA demonstrated that the time for resolution of DKA was identical in patients who received regular insulin via iv, sc, or im routes.
- Once DKA is resolved, patients who are able to eat can be started on a multiple-dose insulin regimen with a long-acting insulin to cover basal insulin requirements and short- or rapid-acting insulin given before meals as needed to control plasma glucose.
- Intravenous insulin infusion should be continued for 1 to 2 hours after the subcutaneous insulin is given to ensure adequate plasma insulin levels.
- If patients are unable to eat, it is preferable to continue the intravenous insulin infusion and fluid replacement.
- Patients who have known diabetes may be given insulin at the dose they were receiving before the onset of DKA or HHS. In patients who have new-onset diabetes, a multidose insulin regimen should be started at a dose of 0.5 to 0.8 U/kg per day, including regular or rapid-acting and basal insulin; the dose is adjusted until an optimal dose is established.