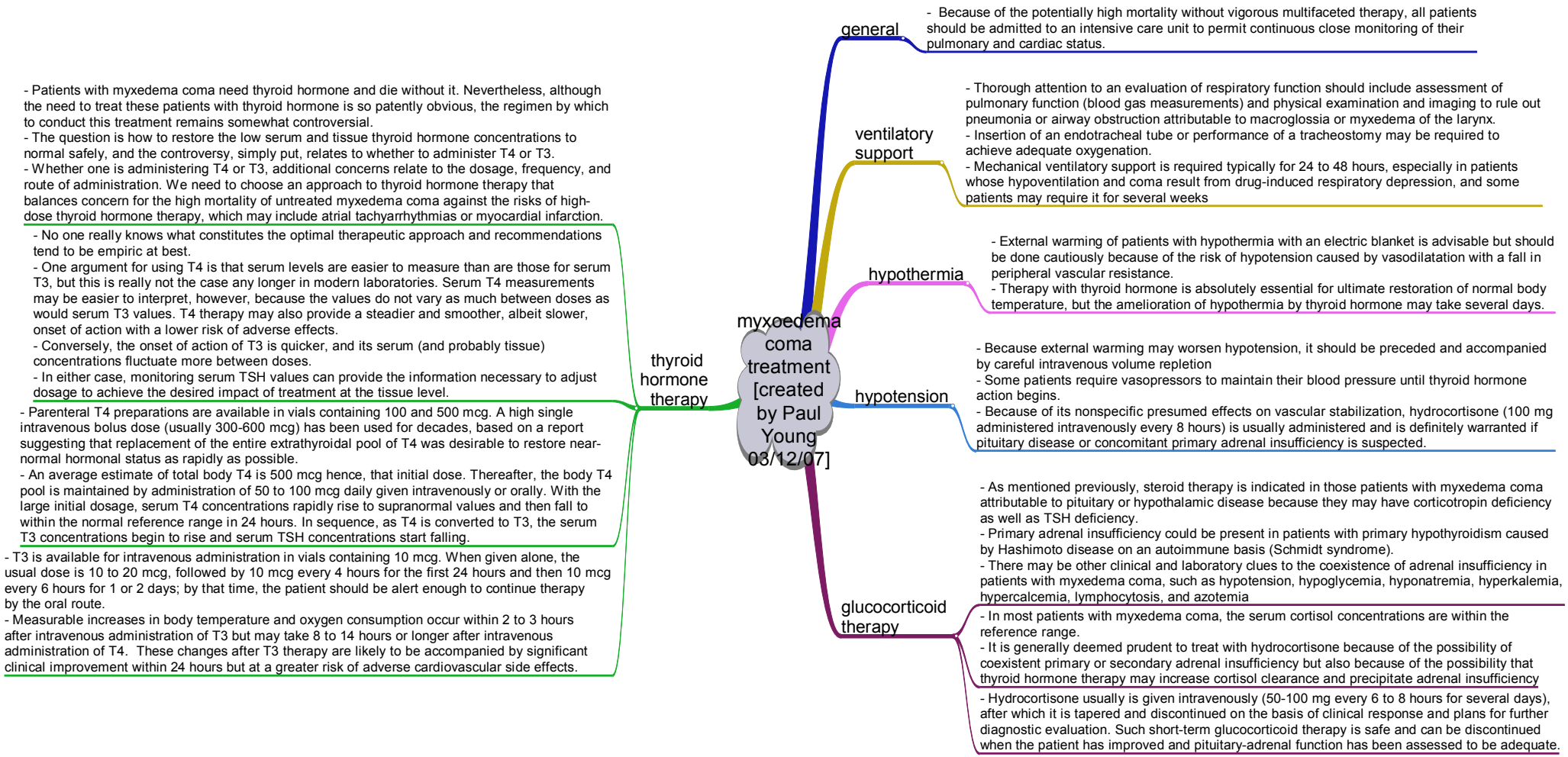
myxoedema coma treatment [created by Paul Young 03/12/07]
general
- Because of the potentially high mortality without vigorous multifaceted therapy, all patients should be admitted to an intensive care unit to permit continuous close monitoring of their pulmonary and cardiac status.
ventilatory support
- Thorough attention to an evaluation of respiratory function should include assessment of pulmonary function (blood gas measurements) and physical examination and imaging to rule out pneumonia or airway obstruction attributable to macroglossia or myxedema of the larynx.
- Insertion of an endotracheal tube or performance of a tracheostomy may be required to achieve adequate oxygenation.
- Mechanical ventilatory support is required typically for 24 to 48 hours, especially in patients whose hypoventilation and coma result from drug-induced respiratory depression, and some patients may require it for several weeks
hypothermia
- External warming of patients with hypothermia with an electric blanket is advisable but should be done cautiously because of the risk of hypotension caused by vasodilatation with a fall in peripheral vascular resistance.
- Therapy with thyroid hormone is absolutely essential for ultimate restoration of normal body temperature, but the amelioration of hypothermia by thyroid hormone may take several days.
hypotension
- Because external warming may worsen hypotension, it should be preceded and accompanied by careful intravenous volume repletion
- Some patients require vasopressors to maintain their blood pressure until thyroid hormone action begins.
- Because of its nonspecific presumed effects on vascular stabilization, hydrocortisone (100 mg administered intravenously every 8 hours) is usually administered and is definitely warranted if pituitary disease or concomitant primary adrenal insufficiency is suspected.
glucocorticoid therapy
- As mentioned previously, steroid therapy is indicated in those patients with myxedema coma attributable to pituitary or hypothalamic disease because they may have corticotropin deficiency as well as TSH deficiency.
- Primary adrenal insufficiency could be present in patients with primary hypothyroidism caused by Hashimoto disease on an autoimmune basis (Schmidt syndrome).
- There may be other clinical and laboratory clues to the coexistence of adrenal insufficiency in patients with myxedema coma, such as hypotension, hypoglycemia, hyponatremia, hyperkalemia, hypercalcemia, lymphocytosis, and azotemia
- In most patients with myxedema coma, the serum cortisol concentrations are within the reference range.
- It is generally deemed prudent to treat with hydrocortisone because of the possibility of coexistent primary or secondary adrenal insufficiency but also because of the possibility that thyroid hormone therapy may increase cortisol clearance and precipitate adrenal insufficiency
- Hydrocortisone usually is given intravenously (50-100 mg every 6 to 8 hours for several days), after which it is tapered and discontinued on the basis of clinical response and plans for further diagnostic evaluation. Such short-term glucocorticoid therapy is safe and can be discontinued when the patient has improved and pituitary-adrenal function has been assessed to be adequate.
thyroid hormone therapy
- Patients with myxedema coma need thyroid hormone and die without it. Nevertheless, although the need to treat these patients with thyroid hormone is so patently obvious, the regimen by which to conduct this treatment remains somewhat controversial.
- The question is how to restore the low serum and tissue thyroid hormone concentrations to normal safely, and the controversy, simply put, relates to whether to administer T4 or T3.
- Whether one is administering T4 or T3, additional concerns relate to the dosage, frequency, and route of administration. We need to choose an approach to thyroid hormone therapy that balances concern for the high mortality of untreated myxedema coma against the risks of high-dose thyroid hormone therapy, which may include atrial tachyarrhythmias or myocardial infarction.
- No one really knows what constitutes the optimal therapeutic approach and recommendations tend to be empiric at best.
- One argument for using T4 is that serum levels are easier to measure than are those for serum T3, but this is really not the case any longer in modern laboratories. Serum T4 measurements may be easier to interpret, however, because the values do not vary as much between doses as would serum T3 values. T4 therapy may also provide a steadier and smoother, albeit slower, onset of action with a lower risk of adverse effects.
- Conversely, the onset of action of T3 is quicker, and its serum (and probably tissue) concentrations fluctuate more between doses.
- In either case, monitoring serum TSH values can provide the information necessary to adjust dosage to achieve the desired impact of treatment at the tissue level.
- Parenteral T4 preparations are available in vials containing 100 and 500 mcg. A high single intravenous bolus dose (usually 300-600 mcg) has been used for decades, based on a report suggesting that replacement of the entire extrathyroidal pool of T4 was desirable to restore near-normal hormonal status as rapidly as possible.
- An average estimate of total body T4 is 500 mcg hence, that initial dose. Thereafter, the body T4 pool is maintained by administration of 50 to 100 mcg daily given intravenously or orally. With the large initial dosage, serum T4 concentrations rapidly rise to supranormal values and then fall to within the normal reference range in 24 hours. In sequence, as T4 is converted to T3, the serum T3 concentrations begin to rise and serum TSH concentrations start falling.
- T3 is available for intravenous administration in vials containing 10 mcg. When given alone, the usual dose is 10 to 20 mcg, followed by 10 mcg every 4 hours for the first 24 hours and then 10 mcg every 6 hours for 1 or 2 days; by that time, the patient should be alert enough to continue therapy by the oral route.
- Measurable increases in body temperature and oxygen consumption occur within 2 to 3 hours after intravenous administration of T3 but may take 8 to 14 hours or longer after intravenous administration of T4. These changes after T3 therapy are likely to be accompanied by significant clinical improvement within 24 hours but at a greater risk of adverse cardiovascular side effects.