respiratory acidosis [created by Paul Young 12/12/07]

definition

- A respiratory acidosis is a primary acid-base disorder in which arterial pCO2 rises to a level higher than expected.
- At onset, the acidosis is designated as an 'acute respiratory acidosis'. The body's initial compensatory response is limited during this phase.
- As the body's renal compensatory response increases over the next few days, the pH returns towards the normal value and the condition is now a 'chronic respiratory acidosis'.

pathophysiology

- The arterial pCO2 is normally maintained at a level of about 40 mmHg by a balance between production of CO2 by the body and its removal by alveolar ventilation. If the inspired gas contains no CO2 then this relationship can be expressed by:
paCO2 is proportional to VCO2 / VA where:
VCO2 is CO2 production by the body
VA is Alveolar ventilation
An increase in arterial pCO2 can occur by one of three possible mechanisms:
(i) Presence of excess CO2 in the inspired gas
(ii) Decreased alveolar ventilation
(iii) Increased production of CO2 by the body

- An adult at rest produces about 200mls of CO2 per minute: this is excreted via the lungs and the arterial pCO2 remains constant. An increased production of CO2 would lead to a respiratory acidosis if ventilation remained constant.
- The system controlling arterial pCO2 is very efficient (ie rapid and effective) and any increase in pCO2 very promptly results in a large increase in ventilation. The result is that increased CO2 production almost never results in respiratory acidosis.
- It is only in situations where ventilation is fixed that increased production will cause respiratory acidosis. Examples of this would be a ventilated patient who develops acute malignant hyperthermia: the arterial pCO2 will rise unless the alveolar ventilation is substantially increased.

aetiology

A: Inadequate Alveolar Ventilation
(i) Central Respiratory Depression & Other CNS Problems
(ii) Drug depression of resp. center (eg by opiates, sedatives, anaesthetics)
(iii) Nerve or Muscle Disorders
(iv) Lung or Chest Wall Defects
(v) Airway obstruction
(vi) Inadequate mechanical ventilation
B: Over-production of Carbon Dioxide
(i) Hypercatabolic Disorders
C: Increased Intake of Carbon Dioxide
(i) Rebreathing of CO2-containing expired gas
(ii) Addition of CO2 to inspired gas
(iii) Insufflation of CO2 into body cavity (eg for laparoscopic surgery)
- in clinical practice, nearly all cases are due to inadequate alveolar ventilation.

effects

- Important effects of Hypercapnia include
(i) Stimulation of ventilation via both central and peripheral chemoreceptors
(ii) Cerebral vasodilation increasing cerebral blood flow and intracranial pressure
(iii) Stimulation of the sympathetic nervous system resulting in tachycardia, peripheral vasoconstriction and sweating
(iv) Peripheral vasodilation by direct effect on vessels
(v) Central depression at very high levels of pCO2

- As CO2 rapidly and easily crosses lipid barriers, a respiratory acidosis has rapid & generally depressing effects on intracellular metabolism.

Cerebral Effects
- The cerebral effects of hypercapnia are usually the most important. These effects are:
(i) increased cerebral blood flow,
(ii) increased intracranial pressure, &
(iii) potent stimulation of ventilation.
- This can result in dyspnoea, disorientation, acute confusion, headache, mental obtundation or even focal neurologic signs.

Cardiovascular Effects
- Typically, the patient is warm, flushed, sweaty, tachycardic and has a bouncing pulse.
- The clinical picture may be modified by effects of hypoxaemia, other illnesses and the patient's medication. Arrhythmias may be present particularly if significant hypoxaemia is present or sympathomimetics have been used.
- Acutely the acidosis will cause a right shift of the oxygen dissociation curve. If the acidosis persists, a decrease in red cell 2,3 DPG occurs which shifts the curve back to the left.

Respiratory Effects
- An arterial pCO2 in excess of about 90 mmHg is not compatible with life in patients breathing room air. This is because of the obligatorily associated severe hypoxaemia. The alveolar gas equation predicts an alveolar pO2 of 37mmHg when the pCO2 is 90mmHg:
pAO2 = [0.21 x (760-47)] - 90 / 0.8 = 37 mmHg.

prevention

- Monitoring of at-risk patients with capnography is appropriate in some situations (eg in an Intensive Care Unit, intraoperatively and in the Recovery Room) and will allow earlier detection of a problem.
- The end-tidal pCO2 is typically lower than the arterial pCO2 and the difference between these values is an index of the magnitude of the alveolar dead space. So if the end-tidal pCO2 is elevated then the arterial pCO2 is usually even more elevated.

- Inadequate ventilation will also necessarily affect arterial oxygenation so steps to avoid, recognise and/or treat arterial hypoxaemia are very important. The simple measure of providing supplemental oxygen by face mask to patients can often correct or prevent hypoxaemia.

- Some particular medical situations where prevention can be utilised are:
(i) Better airway care and attention to safe positioning of cerebrally obtunded patients (ie prevent airway obstruction).
(ii) Increased care in the use of drugs (such as CNS sedatives or opiate drugs) which can depress ventilation
(iii) Increased attention to the care of patients at risk of aspiration (eg unconscious patients)
(iv) Ensuring adequate reversal of neuromuscular relaxants

assessment

- The best available quantitative index of the magnitude of a respiratory acidosis is the difference between the 'actual' pCO2 and the 'expected' pCO2
- Definition of Terms
(i) Actual pCO2 - the measured value obtained from arterial blood gas analysis.
(ii) Expected pCO2 - the value of pCO2 that we calculate would be present taking into account the presence of any metabolic acid-base disorder
Expected pCO2 = 1.5 (Actual [HCO3]) + 8 mmHg

'post hypercapnic alkalosis'

- The correction of the elevated bicarbonate (renal compensation) associated with chronic respiratory acidosis may not be rapid. Return of plasma bicarbonate to normal requires renal excretion of the excess bicarbonate. The kidney has a large capacity to excrete bicarbonate but in certain abnormal conditions this capacity is impaired and the bicarbonate level remains elevated.
- The persistence of elevated bicarbonate despite resolution of the chronic respiratory acidosis is referred to by some as 'post-hypercapnic alkalosis'.

restoration of ventilation

- The pCO2 rapidly returns to normal with restoration of adequate alveolar ventilation Treatment usually needs to be directed to correction of the primary cause if this is possible. In severe cases, intubation and mechanical ventilation will be necessary to restore alveolar ventilation.
- The patient can deteriorate following intubation and ventilation which results in a rapid fall in pCO2 especially if the respiratory acidosis has been present for some time. Rapid return of pCO2 towards normal in this situation may be accompanied by severe hypotension due to decreasing sympathetic stimulation as CO2 falls

buffering & compensation

Acute Respiratory Acidosis - Buffering only
- The compensatory response to an acute respiratory acidosis is limited to buffering. About 99% of this buffering occurs intracellularly.
- Intracellularly, proteins (including haemoglobin) and phosphates are the most important buffers involved.
- Though very important for carriage of carbon dioxide in the blood, the bicarbonate system is not responsible for any buffering of a respiratory acid-base disorder. This is basically because a system cannot buffer itself. Consider: For the bicarbonate system to 'buffer' H+ produced from the dissociation of H2CO3 would just result in the production of an equal amount of CO2.

Chronic Respiratory Acidosis - Renal Bicarbonate Retention
- With continuation of the acidosis, the kidneys respond by retaining bicarbonate.
- This response to a chronic respiratory acidosis is slower and takes 3 or 4 days to reach its maximum.
- The response occurs because increased arterial pCO2 increases intracellular pCO2 in proximal tubular cells and this causes increased H+ secretion from the PCT cells into the tubular lumen. This results in:
(i) increased HCO3 production which crosses the basolateral membrane and enters the circulation (so plasma [HCO3] increases.)
(ii) increased Na+ reabsorption in exchange for H+ and less in exchange for Cl- (so plasma [Cl-] falls)
(iii) increased 'NH3' production to 'buffer' the H+ in the tubular lumen (so urinary excretion of NH4Cl increases)