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 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 loss in exchange for CI- (so plasma [Cl-] falls)
  (iii) increased ‘NH3’ production to ‘buffer’ the H+ in the tubular lumen (so urinary excretion of NH4Cl increases)

- 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.

- 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'.

- 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 would calculate be present taking into account the presence of any metabolic acid-base disorder

Expected pCO2 = 1.5 (Actual [HCO3] ) + 8 mmHg

- 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 a useful 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 recognising 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

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 dysequia, 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.