Describe the respiratory response to hypoxaemia in both the awake and anaesthetised patient.

**Hypoxaemia** = reduced partial pressure of oxygen in arterial blood

### Awake Patients

**Sensors**

- **Peripheral chemoreceptors** (there is virtually no significant central response to hypoxaemia)
  - Located in the carotid body (bifurcation of carotid artery)
  - Have a high rate of perfusion (x10 expected for metabolic rate) – therefore low a/v PO2 difference
  - Glomus type 1 cells – synaptic contact with carotid sinus nerve endings (CN IX)
    - Thought to be O2 sensitive K channels responsible for the hypoxic response
      - Decreased O2 → inhibits activity of K channel → alters membrane potential
      - Ca channels open → influx of Ca++ → transmitter release (?ATP/Ach)
  - Respond to decreased PaO2 (NOT to decreased O2 content – therefore minimal stimulation in e.g. Anaemia, metHb/COHb)

**Triphasic time course of ventilatory response to hypoxia.** The nature of the response is dependent on the experimental conditions and CO2 (eg. Was CO2 controlled for “isocapnoea” or not controlled for “poikilocapnoea”)

**FIG. 4.7** Time course of the ventilatory response to hypoxia (SaO₂ ~ 80%). Practical problems prevent the continuous and rapid measurement of minute volume and respiratory gases for 8 h, so the curves are produced from combining the data from three studies. When arterial PCO₂ is maintained at normal levels (isocapnia) the response is triphasic. When arterial PCO₂ is not controlled (poikilocapnia) the magnitude of the response is damped because the hypoxia-induced hyperventilation reduces PCO₂ and therefore respiratory drive. See Figure 15.3 for respiratory effects of prolonged hypoxia. (After references 65, 66 and 67.)

1. **Acute hypoxic response**
   - Over seconds (immediate) to minutes

2. **Hypoxic ventilatory decline**
   - Over 20-30 minutes, there is a somewhat decline in MV until a plateau is reached. Unclear why.

3. **Ventilatory response to sustained hypoxia**
   - 8-24 hours. Sustained rise.

**Ventilatory response and relationship to PO2**

- Once again is dependent on experimental conditions. In isocapnoea – MV is fairly stable until <60mmHg, when it rapidly increases (green line)
Central integrator
- Respiratory centre in medulla

Effectors
- Respiratory muscles (diaphragm, intercostals, scalenes, etc)

Hypoxaemia increases the ventilatory response to CO2
- Note the increased gradient of the ventilatory response curve in states of hypoxaemia vs normoxia vs hyperoxia
Anaesthetised Patients

Blunting of acute hypoxic ventilatory response
- Dose dependent
- Hypoxic drive attenuated even at low MAC 0.1
- Both inhalational and intravenous general anaesthetics cause blunting
- However – the attenuation of the response is dependent on the CO2 conditions again
- Effect is thought to be on the peripheral chemoreceptors
- There is partial attenuation by surgical stimulus

Note – The examiners report has some issues. It glosses over the controversy regarding the blunting of acute hypoxic ventilatory response with anaesthesia, rather simply stating it as fact (see Nunns). It
also indicates that marks are awarded for discussion regarding hypoxic pulmonary vasoconstriction – despite that being a respiratory response to low alveolar partial pressure of O2 (Not hypoxaemia as the question asks).