Anaesthesia in the elderly

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Abstract

Older people are undergoing increasingly complex surgery with much greater mortality and morbidity than the younger adult population. In this article, we discuss the physiological changes that take place in the older patient, and how these may affect anaesthetic technique. Perioperative risk in the elderly is discussed, with focus on emergency surgery and frailty.

Keywords Anaesthesia; elderly; frailty; perioperative; surgery

Royal College of Anaesthetists CPD Matrix: 1A01, 1A02, 2A03

Learning objectives

After reading this article, you should be able to:

- recall the physiological changes that occur with ageing
- describe the modifications in technique necessary when anaesthetizing older people
- list the risk factors for the development of postoperative delirium
- list the factors associated with an increase in perioperative morbidity and mortality

The demographics of the UK population are changing, placing additional pressure on finite NHS resources: 1.4 million residents are now aged over 85 years, compared to only 200,000 when the NHS was founded in 1948. The number of older people undergoing surgery is increasing: in 2014–5, the over-75s accounted for 22% of all surgical procedures carried out in England. Older people are being considered for increasingly complex surgical procedures, and surgery in older patients (particularly emergency surgery) carries a much greater risk of mortality and morbidity than that of the younger adult population.

Pathophysiology of ageing

The pathophysiology of ageing at a cellular level is still a matter of much research and debate. In summary, ageing is thought to occur through a combination of genetically pre-programmed cell death (the telomere theory) and cellular damage occurring throughout life (the free radical and somatic mutation theories). Overall, there is a reduction in the number of cells within an organ, and a decline in function of the remaining cells.

At a macroscopic level, the decline in organ function begins in early adult life, but often does not become clinically evident until almost all organ reserve is lost. Organ failure occurs either when the organ function declines to a point where it can no longer support life, or when the organ cannot increase its function sufficiently to mount a physiological response to a stressor, for example acute illness, anaesthesia and surgery. Functional decline in the cardiovascular, respiratory, renal, central nervous, haematological and musculoskeletal systems are of greatest concern perioperatively, and may influence outcome from elective or emergency surgery. Anaesthetists must therefore be familiar with the normal physiology of ageing, of which there is considerable individual variability, as well as the consequences of accumulated co-morbidities, cognitive impairment, frailty and polypharmacy.

Respiratory

Physiology:

The thoracic cage becomes more rigid due to calcification of the costal cartilages, leading to reduced thoracic wall compliance. The elastic fibres of the alveolar septae undergo degeneration, leading to:

- loss of support for alveoli and small airways, resulting in airway collapse during normal tidal breathing. This is a major cause of ventilation–perfusion (V/Q) mismatch, and is the main contributor to the increased alveolar–arterial (A–a) gradient seen in the elderly.
- an increase in lung compliance, which partially offsets the reduced thoracic wall compliance.

The architecture of the alveolae also changes with advancing age. Total alveolar surface area decreases and there is thickening of the delicate alveolar-capillary membrane, both of which reduce lung diffusing capacity and contribute to the raised A–a gradient.

The result of these changes is a clinical picture very much like that of mild chronic obstructive pulmonary disease (COPD), that is, an increase in residual volume and functional residual capacity (FRC). FRC is the point at which the inward lung elastic forces match the outward force generated by the thoracic cage. In older people, FRC occurs at a higher lung volume: anterior–posterior thoracic cage diameter is therefore increased, with flattening of the hemi-diaphragms. Kyphosis may also contribute to adverse lung mechanics. Similar to patients with COPD, these mechanical changes make inspiration less efficient, with an increase in energy expended during inspiration. These changes, along with intercostal and diaphragmatic muscle atrophy, make the elderly more susceptible to respiratory fatigue.

Anaesthetic considerations:

- Edentulous patients may be difficult to ventilate with bag and mask, but are generally easier to intubate.
- The upper airway is prone to collapse during recovery from anaesthesia, resulting in snoring and hypoxaemia.
- There is a progressive decrease in protective laryngeal reflexes with ageing, which increases the risk of aspiration.

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- The decrease in respiratory compliance, changes in lung mechanics and inspiratory muscle atrophy makes older patients more prone to postoperative respiratory failure.

Cardiovascular

Physiology:

Arteries become less elastic with age as the tunica media undergoes fibrosis, which leads an increase in systolic blood pressure. Systolic hypertension has two major consequences:

- The left ventricle must generate a greater pressure to eject blood into a stiffened aorta.
- The velocity of the arterial pressure wave is increased. Normally, the reflected pressure wave reaches the heart in early diastole, and is responsible for the bump after the dicrotic notch in the arterial pressure waveform. However when arteries are stiffened, the reflected arterial pressure wave reaches the heart in late systole, which further increases the afterload against which the left ventricle must pump.

In response to raised afterload, the left ventricle undergoes hypertrophy. Ventricular hypertrophy impairs diastolic relaxation, and the left ventricle becomes increasingly dependent on atrial contraction for filling. This physiological diastolic dysfunction can be exacerbated by co-morbid disease, for example systemic hypertension and ischaemic cardiomyopathy.

There is a reduced ability to increase cardiac output in response to hypovolaemia, due to:

- a reduction in β-receptor responsiveness, which limits the ability of the left ventricle to increase contractility.
- fibrosis of the carotid sinus, which impairs the ability of the baroreceptors to rapidly increase heart rate in response to a decrease in blood pressure.
- a decrease in venous compliance, which impairs the venous buffering mechanism.

In addition, the elderly are more prone to supraventricular tachyarrhythmias due to fibrosis of the sino-atrial node and a large reduction in the number of pacemaker cells. The onset of atrial fibrillation can therefore seriously impair the filling of the left ventricle, with a consequent reduction in cardiac output.

Anaesthetic considerations:

- Prolonged arm–brain circulation time delays onset of anaesthesia when using intravenous induction agents, but in theory increases the speed of onset of a gas induction. In reality, however, the duration of inhalational induction is usually prolonged due to the reduced alveolar diffusion and the increased *V/Q* mismatch that occurs with advancing age.
- The elderly are more prone to intraoperative hypotension, especially at induction of anaesthesia, and are less able to adequately compensate.
- The reduced cardiac responsiveness to β-agonists means that drugs such as ephedrine (a mixed α- and β-agonist) become less effective with advancing age.
- Use of oesophageal Doppler cardiac output monitoring may be less accurate in the elderly, as the aorta is poorly compliant.
- The combination of changes to cardiovascular and respiratory physiology with ageing result in a decreased oxygen uptake and oxygen delivery, and thus a greater perioperative risk of myocardial and cerebral ischaemia.

Neurological

Physiology:

There is a progressive decline in brain mass from approximately 50 years of age. In older people, this gives the appearance of cerebral atrophy on CT. Though there is a decrease in both grey and white matter, the resulting decrease in cognitive function is usually modest with short-term memory most often affected. Cerebral blood flow (CBF) decreases by 10–20% which, as CBF is tightly coupled to brain metabolic rate, is a result of the reduction in brain mass. Synthesis of some neurotransmitters (e.g. dopamine) is reduced, as are the number of receptors and binding sites. Loss of dopaminergic neurons results in Parkinson's disease, whilst loss of cholinergic neurons is implicated in the development of Alzheimer's disease. Indeed, cognitive impairment becomes increasingly common with advancing age, affecting 20% of patients aged over 80 years old. Sensory impairment is also common: deafness is very common in older people, and visual impairment affects around one-fifth of those aged over 75.

Older people are more likely to experience neurological dysfunction following surgery:

- Delirium is defined as a disturbance of consciousness that is accompanied by a change in cognition that cannot be better accounted for by a pre-existing or evolving dementia. Postoperative delirium (POD) occurs in over 20% of those aged over 65, usually develops in the first few postoperative days, and is usually temporary. It has a fluctuating course, with abnormal circadian rhythm, inattention, disorientation and memory deficit. POD may be sub-classified as hyperactive, hypoactive (which carries a higher mortality), or mixed variation. POD is independently associated with increased mortality, increased length of hospital stay, functional disability, and discharge to long-term care institutions.[1] Risk factors for the development of POD include type of surgery (high risk groups include cardiac surgery, emergency surgery and following hip fracture) and patient-specific factors: increasing age, pre-existing cognitive dysfunction, lower educational attainment, previous stroke.
- Postoperative cognitive dysfunction (POCD) is a subtle impairment of memory, concentration and information processing usually lasting weeks or months, that is distinct from delirium and dementia. The incidence of POCD is difficult to determine, partly because there are no formal criteria for its assessment and diagnosis, but is thought to be in the order of 10% at 3 months.[1] Recognition of POCD is important as it is associated with an increased mortality, and may result in a functional step-down in social circumstances, for example from residential care to nursing care.[1] The cause of POCD is unknown, and several hypotheses exist. Risk factors for the development of POCD are the same as those of POD, with cardiac surgery carrying the greatest risk. Whilst it is tempting to think that avoidance of general anaesthesia may prevent POCD, studies have failed to show that spinal anaesthesia has any protective effect when compared to general anaesthesia.[2]

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