POST OPERATIVE NEUROCOGNITIVE DYSFUNCTION

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POST OPERATIVE NEUROCOGNITIVE DYSFUNCTION

INTRODUCTION

BUT SHE WAS FINE WHEN WE BROUGHT HER IN...!?

‘So my family members asked physicians they knew the same question and reported back to me that a cardiologist, a neurologist, and an internist had said that my father should not be experiencing any confusion after surgery and therefore he must have dementia. This came as a complete surprise to me, as I just assumed that all health care professionals, especially those who work with older people, would know what I knew. I didn’t receive medical training, but I am a geriatric social worker. I have worked in hospitals, where I came to know first-hand that older people are commonly confused for a period of time following major surgery and anaesthesia. How could it be that these physicians were not aware of this?’[1]

The ageing brain presents with many structural and functional changes that will affect the anaesthetist. With age, the brain atrophies with neuronal loss, which is significant in the cerebral cortex but is also appreciated in the thalamus, locus coeruleus and basal ganglia. Imaging studies show an alteration in functional activation of the prefrontal cortex and hippocampus with associated reduction in executive function [2]. There is a reduction in cerebral blood flow, oxygen consumption and blood brain barrier penetration. A reduction in the production of neurotransmitters and neurotransmitter receptors, namely, dopamine, serotonin and acetylcholine, are documented. Patients also have a decreased sensory input to the brain from the body. This population experiences sleep cycle changes with increased wakefulness at night with associated lethargy during the day. Memory decline is progressive which can be associated with cognitive dysfunction.[3, 4]

It is predicted that by the year 2030, 20% of the population will be above the age of 65 years and by the year 2050, thirty-one million of the population will above the age of 80 years [3]. With the advancement of medical and surgical interventions the elderly population is fast growing with more patients presenting to theatre for surgeries. With ageing there is associated increase in perioperative morbidities and mortality. The effects of anaesthesia on the ageing brain has been topical over the past years, looking at specifically its impact on cognition. Above the age of sixty-five, patients are at a higher risk of developing postoperative cognitive dysfunction [5] this presents a challenge to an unknowing anaesthetist.

In 2018 the Nomenclature Consensus Working Group recommended the term Perioperative Neurocognitive Disorders to describe cognitive impairment in the perioperative period as shown below. [6]
Cognitive decline before surgery | Neurocognitive disorder
---|---
Any acute cognitive decline event | Post-operative delirium
Postoperative cognitive decline diagnosed up to thirty days after surgery | Delayed neurocognitive recovery
Cognitive decline up to twelve months | Post-operative neurocognitive disorder

The commonest cognitive disorders in the postoperative period are post-operative delirium and post-operative cognitive dysfunction (14). Delirium is better understood and described as a disturbance in consciousness that is accompanied by an acute change in cognition that cannot be accounted for by a pre-existing or evolving dementia [7].

Post-operative cognitive dysfunction (POCD) was first described by Bedford in the 1950’s, to date the topic is still poorly understood. POCD is defined as a change in neurocognitive function and behaviour after surgery[8] . This can be seen from seven days up to one year in 60% post cardiac surgery and in 10% after general major surgery. [9] Patients present with changes in memory, attention, concentration, executive function, decline in psychomotor speed and visuospatial ability.

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Inflammation
The protective nature of the inflammatory response, once uninhibited, can have detrimental effects to the human body and can herald neuro inflammation. Surgery induces local and systemic inflammatory response with associated immune activation. Tissue damage supports the release of interleukin 1 (IL-1) and tissue necrotic factor alpha (TNF-alpha) which promotes production of interleukin 6 (IL-6). The inflammatory marker IL6 and elevated C-reactive protein (CRP) have been shown to have a strong association with memory impairment and decline in cognitive function in both animal and human studies [11].

Systemic inflammation can cause sickness behaviour, which presents with physiological and behavioural changes such as depression, cognitive impairment and social withdrawal [11]. This is not exclusive to the post-operative period but can be found in other conditions. In surgical trauma patient’s monocyte activation leads to elevated monocyte chemoattractant protein 1, which promotes migration of monocyte derived macrophage into the brain. This is associated with neurological problems including dysfunctional cognition.

The blood brain barrier with its unique endothelial layer does not permit the circulating agents to reach the brain, thus protecting the brain from toxicity. A breach in this system promotes migration of proinflammatory markers into the brain. Gadolinium-
enhanced magnetic resonance imaging showed acute disruption in the blood brain barrier after cardiac surgery and this has been linked to neurological impairment[11].

Microglial cells are immune cells located within the brain and are responsible for remodelling and synaptic activity in the brain. When activated they secrete pro and anti-inflammatory factors which are neuroprotective unless there is dysregulation of this process. In patients with dementia, they are protective by removing amyloid deposits and promoting release of growth factors. However, they can also cause hyperphosphorylation of tau and cytokine release, which contributes to neuronal loss. Microglia also induces astrocytes, which lead to neuronal toxicity and death. They too have been implicated in POCD after liver surgery. In orthopaedic surgery, there is associated increase in complement activation in astrocytes and microglia, which has an association with hippocampus inflammation and synaptic loss. In a study by Forsberg et al, microglial activation was seen in patients with cognitive impairment at three months.[11, 12]

Oxidative Stress
The high-mobility box-1 chromatin protein (HMGB1), which is released and activated during oxidative stress and intense inflammatory response, is also associated with perpetuating long-term inflammation by promoting immune cell proliferation and maturation. A study done in mice showed a link in poor cognitive function postoperatively with increased HMGB1. An antibody administered to counteract HMGB1 showed a reduction in inflammation and cognitive decline post-surgery. In a different study, surgery and anaesthesia induced an increase in HMGB1 in the hippocampus implicating it in cognitive dysfunction.[12]

Brain hypoperfusion causes ischaemia, which subsequently increases the production of reactive oxygen species (ROS). ROS can induce and perpetuate neuro inflammation, disrupting the integrity of the blood brain barrier. The brain has low levels of antioxidants therefore prone to oxidative stress. In patients diagnosed with depression oxidative species level was associated with cognitive dysfunction. This hypothesis has led to the use of antioxidants to attenuate the symptoms of cognitive decline in the older population[12].

Anaesthesia
Centrally acting anaesthetic agents have been studied to find their effect on the ageing brain. Most studies have been carried out on animals and have not been validated in human participants. Volatile agents, especially isoflurane, are associated with microglial activation and inflammation. A suggestion is that anaesthetic drug exposure causes mitochondrial dysfunction and neuronal oxidative damage. Conflicting evidence exists regarding depth of anaesthesia with burst suppression which showed some protection. When comparing regional and general anaesthesia, there was no difference in outcomes at three months.[9, 11, 12]
POST-OPERATIVE DELIRIUM

POD is described as an acute and fluctuating alteration of mental state of reduced awareness and disturbances in attention for up to five days after surgery[13]. This must be differentiated from emergence delirium which is an abnormal state as a result of anaesthesia administration during transition from unconsciousness to complete wakefulness[14].

Diagnosis
The DSM IV diagnostic criteria for Delirium [15]:

A. A disturbance in attention and awareness.
B. The disturbance develops over a short period of time, represents a change from baseline attention and awareness and tends to fluctuate in severity during the course of the day.
C. An additional disturbance in cognition, memory deficit, disorientation, language, visuospatial ability and or perception.
D. Disturbances in A and B are not better explained by another pre-existing established or evolving neurocognitive disorder and do not occur in the context of a severely reduced level of arousal, such as a coma.
E. Evidence from history, physical examination, laboratory investigations that the disturbance is a direct physiological consequence of another medical condition, substance intoxication or withdrawal, exposure to toxins or is due to multiple aetiologies.

POD can further be divided into hypoactive, hyperactive and mixed subtypes. Patients presenting with hypoactivity can go undiagnosed leading to a delay in treatment intervention. A high level of suspicion and screening of these patients can reduce missed diagnosis[16].

<table>
<thead>
<tr>
<th>Hyperactive</th>
<th>Agitated, aggressive, combative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoactive</td>
<td>Anhedonia, reduced alertness</td>
</tr>
<tr>
<td>Mixed</td>
<td></td>
</tr>
</tbody>
</table>

Delirium is diagnosed in 62% of high-risk surgical patients and has healthcare costs of up to $150 billion in the United States [17]. Postoperative delirium occurs in 14% in the medical ward and up to 80% in the intensive care units. It is associated with prolonged hospitalisation, cognitive impairment, functional decline and increased six months mortality[18].

The highest incidence is reported in older patients and institutionalised patients. POD occurs in up to 50% of patients presenting for vascular, cardiac and hip fracture operations[16].
POD is associated with short term and long-term cognitive dysfunction, with POCD found in POD in ICU patients. POD is also associated with dementia in up to five years and post-traumatic stress disorders three months after surgery.

**Risk Factors**
Risk factors for POD are increase in age, frailty, pre-existing cognitive impairment, pre-existing uncontrolled comorbidities and sensory impairment. POD can be precipitated by substance abuse, emergency surgery, higher ASA grading, peri-operative drugs, prolonged fasting time, interrupted sleep, intraoperative blood loss and poor pain control. The impact of pre-existing cognitive dysfunction increases the vulnerability to minor insults leading to POD, where minor local infection can lead to POD whereas in previously well patients only systemic sepsis will lead to POD [16, 18, 19]. Patients with multi organ disease will be at a higher risk for POD. Disease processes with low haemoglobin, reduced ejection fraction, renal impairment and coronary artery disease place patients at a higher risk[20].

**Assessment Tool**
It is suggested that patients must be screened for POD before discharge from the recovery room. A wide variety of test are available such as the Delirium Rating Scale, Confusion Rating Scale, DSM IV, ICD 10, The Nursing Delirium Screening Scale (Nu-DESC) and the Confusion Assessment Method (CAM). The best scale to be used should have high specificity and sensitivity, be easy to use, fast and applicable to the population. The CAM and Nu-DESC have been found to have high sensitivity. The Nu-DESC had a sensitivity ranging from 32% to 95% with a specificity of 87%, the CAM had a sensitivity of 28% to 43% with a specificity of 98%[19].

The CAM is said to have lower sensitivity when used by untrained staff. Both tests were found to be time consuming and not ideal for use in recovery.
Algorithm for management of postoperative delirium in adult patients [20]

Management Approach
General care of patients at high risk include the use of non-pharmacological measures to reduce POD. The Hospital Elder Life Programme reduced the incidence of delirium by 40% and the duration of the disease by 35% (3). This was directed at treating cognitive impairment, sleep deprivation, immobility, visual and hearing impairment and dehydration. Multi-disciplinary care is advised including the geriatric team, dieticians, physiotherapy, occupational therapy and importantly family members. Patient using visual or hearing aids must have them in use very soon post operatively. Patients must be nursed in quiet wards with appropriate re-orientation, early mobilisation, minimise catheters and drains, early and maintained nutrition.

Premedication with benzodiazepines should be avoided unless in patients with severe anxiety or in patients with withdrawal syndromes. The use of melatonin and neuroleptics have been inconclusive and its use as a premedication is not recommended[20]. Alpha 2 agonists have been shown to be beneficial in patients presenting for cardiac and vascular surgery[16].

The development of POD with different anaesthetic approaches is unclear, with regional anaesthesia not showing any benefit in POD. What affects the outcomes is the reduction of stress response and adequate pain control. A study suggested that the use of infusions to administer analgesia e.g. Remifentanil infusion compared to
bolus analgesia e.g. fentanyl may reduce the incidence of POC. Ketamine reduces opioid consumption and inflammatory markers post operatively, however has had variable results when used in POD. A small group showed reduction of POD in cardiac patients with a larger group showing no statistically significant difference in POD but higher incidence of nightmares and hallucination with higher doses\cite{16, 18}.

Intraoperative neuromonitoring should be used to avoid burst suppression in the elderly\cite{16}. Hypotension has been associated with poor outcomes, one study showed that blood pressure swings when compared to hypotension were more predictive of POD \cite{20}.

Postoperative use of opioids increases the incidence of POD, however patient controlled analgesia is said to be a better mode of delivery of the drug. Post-operative hypoxia, though unclear if it is an independent predictor of POD, should be avoided\cite{20}.

### Table 8 Evidence-based and consensus-based statements regarding prevention and treatment

<table>
<thead>
<tr>
<th>Statement</th>
<th>LoE</th>
<th>Age group (inclusion criteria)</th>
<th>GoR</th>
</tr>
</thead>
<tbody>
<tr>
<td>We suggest implementing fast-track surgery to prevent POD</td>
<td>[198], 1b; [199], 2b; [198], 2b; [193], 2b</td>
<td>[198,199]</td>
<td>B</td>
</tr>
<tr>
<td>We suggest avoiding routine premedication with benzodiazepines except for patients with severe anxiety</td>
<td>[10], 2b; [106], 5; [198], 2b; [196], 3b; [197], NR; [198], 2b; [204], 2b</td>
<td>[10], Inc. none. POD+, 67.7 years, POD-, 50 years; [105,195,199], &gt;60% were ≥65 years; [197]; [199] mean age 66.8 years and range 43–87 years</td>
<td>B</td>
</tr>
<tr>
<td>We recommend monitoring depth of anaesthesia</td>
<td>[105], 5; [199], 1b; [200], 1b; [201], 1b; [202], 1b</td>
<td>[199–201]</td>
<td>A</td>
</tr>
<tr>
<td>We recommend adequate pain assessment and treatment</td>
<td>[103], 2b; [153], 1b; [197], NR; [203], 4; [205], 2b; [206], (SR)</td>
<td>[202]</td>
<td>A</td>
</tr>
<tr>
<td>We suggest using a continuous intraoperative analgesia regimen (e.g. with remifentanil)</td>
<td>[13], 2b; [207], 2b</td>
<td>[13,207]</td>
<td>B</td>
</tr>
<tr>
<td>We recommend promptly diagnosing POD, establishing a differential diagnosis, and instituting treatment</td>
<td>[37], 2b; [38], 2b; [178], 2b; [206], Consensus review</td>
<td>[37]</td>
<td>A</td>
</tr>
<tr>
<td>We suggest using low-dose haloperidol or low-dose atypical neuroleptics to treat POD</td>
<td>[206], 5; [209], SR; [210], 2b; [211], 2b</td>
<td>[206,209]</td>
<td>B</td>
</tr>
</tbody>
</table>

Data presented as reference number, LoE, Incl. inclusion criteria; obsvd., observed; LoE, level of evidence; GoR, grade of recommendation (strong = A, conditional = B); POD, postoperative delirium. *Low-dose haloperidol means 0.25 mg stepwise titrated up to maximum of 3.5 mg. An excessive dose of haloperidol of more than 6 mg a day should not be used.** Long-term use in dementia patients may increase harm.
Post-operative cognitive dysfunction (POCD)

Post-operative cognitive dysfunction (POCD) is an objective measurable decline in cognitive function of varying intervals after anaesthesia and surgery, up to 3 months to 7.5 years after surgery[6]. POCD has not been described in the DSM-V nor in the International Classification of Disease but it can be related to mild neurocognitive disorder of unspecified aetiology.

The highest incidence, 60% is seen in patients undergoing cardiac surgery, with elective hip surgery at 22%[5]. Just as we risk stratify patients for systemic disease, it is important that we identify preoperatively patients at risk for POCD. This will allow for allocation of resources and intervention adequately in hospitals and it gives patients an opportunity to put measures in place for the postoperative period. [21]

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>General</th>
<th>Anaesthesia</th>
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<tbody>
<tr>
<td>Age above 65 years</td>
<td>Hyperventilation</td>
<td></td>
</tr>
<tr>
<td>Pre-existing cognitive impairment</td>
<td>Hypoxia</td>
<td></td>
</tr>
<tr>
<td>Low level of education</td>
<td>Hypotension</td>
<td></td>
</tr>
<tr>
<td>Invasive surgery</td>
<td>Hypoglycaemia</td>
<td></td>
</tr>
<tr>
<td>Low socioeconomic background</td>
<td>Hyperthermia</td>
<td></td>
</tr>
<tr>
<td>Genetic – Reduced Apoprotein E</td>
<td>Depth of Anaesthesia</td>
<td></td>
</tr>
<tr>
<td>Alcohol Abuse</td>
<td>Anticholinergic medication</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Poor pain control</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Blood Transfusion</td>
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</table>

Seven out of twenty-four studies identified age as the common risk factor, with patients over the age of sixty-five years having the highest incidence. The level of education plays a key role, patients with low level of education are prone to POCD when compared to their counterparts with higher learning level. POD can progress to POCD but patients who have not had POD can still develop POCD. A relation to progression to dementia has been suggested [22] but there is no clear evidence, patients who were followed up to ten years found no association with dementia and previous diagnosis of POCD[5].

Other risk factors include, previous cerebrovascular accidents, lacunae on brain imaging, depression post-operative infection, post-operative pulmonary complications and time spent with the BIS of less than 40. Patients going for major thoracic, intra-abdominal and orthopaedic surgery have a higher risk of developing early POCD. Evidence of hypotension and hypoxia has not shown any association with POCD. Hypoperfusion measured by near infrared spectroscopy does indicate an association with POCD.
Assessment Tool

Patients are diagnosed using neuropsychological testing preoperatively and postoperatively to ascertain cognitive decline[5]. Ideally these tests are done one week before the proposed surgery and patient follow up to one-year post surgery. The use of biomarkers and radiological testing is theoretical and still being investigated. Neuropsychological testing can be affected by multiple factors, making the timing of the test an important consideration. Confounders to assessment on the day of surgery include pain, anxiety and acute drugs administration. Suggestions to avoid this include testing at pre-anaesthetic clinics, memory clinics and surgical clinics, however time factor may hinder this process.
Multiple tests are available, most lack sensitivity and specificity and are not validated for POCD but for mild cognitive impairment. The test must be administered by trained personnel, results must be matched against age, sex, level of education and the population background. The Montreal Cognitive Assessment (MoCA) tool was studied in neurosurgical, vascular and general surgery patients and has a sensitivity of 90% and specificity of 87% in assessing mild cognitive impairment (MCI). The Addenbrooke’s Cognitive Exam (ACE111) has high sensitivity for detection of dementia but has not been validated in surgical patients. The Quick MCI is a development of the AB cognitive screen and has an improved ability to discern normal cognition, mild cognitive impairment and dementia.[5]

Management
A multidisciplinary approach was advocated by the Royal College of Anaesthetists to manage these patients. The cost implication of perioperative management can be outweighed by the burden of expense that POCD will have on the health system. Appropriate management and control of comorbidities plays a crucial role. Preoperative optimisation of electrolytes such as magnesium, which is associated with memory impairment, has been shown to have some benefit. Anaemia is not directly related to POCD; however, optimisation has some indirect benefits. The use of benzodiazepine must be done with caution, however in patients on chronic psychoactive medications, it is not advised to discontinue perioperatively as it increases the risk of withdrawal which is associated with dementia.

Informed consent must explicitly include the possibility of POCD. This allows the patient the opportunity to put in place processes for future unexpected occurrence[21], such as medical interventions, financial planning and a will. It must be ensured that this is done voluntarily by the patient and under no pressure. Informing the family of the potential risk of POCD allows room for adjustment and transparent level of expectation with the road to recovery.

To date there is no evidence which shows superior results with any anaesthesia choice[5]. In cardiac surgery neurological outcomes were believed to be from micro emboli, a study done showed no significant risk reduction in patient on pump and off pump surgery[22]. Intravenous lignocaine given to older patients in spine surgery showed a higher mini mental state exam, this is from the reduced release of IL6 and other proinflammatory markers. Cerebral oxygenation using NIRS and monitoring of depth of anaesthesia with BIS showed a reduction in the incidence of POCD. Of note, amongst its other short comings, the BIS has not been validated in older patients and a better assessor would be an EEG. A suggestion is to use age adjusted end tidal MAC fraction and aim to maintain cerebral perfusion.[21]

The use of steroids to counter the inflammatory response showed no benefit[23].

Avoiding post-operative complications such as wound infection and respiratory complications can reduce the incidence of the disease[8].

As one of the predictors of POCD is preoperative cognitive function, brain training has been shown in animal studies and in some low-quality human studies to be beneficial.
It is suggested that placing patients in stimulating environment promotes neurogenesis[12] this can be applied preoperatively to patients at risk.

### Novel Therapies for POCD[12]

<table>
<thead>
<tr>
<th>Anti-Inflammatories</th>
<th>Non-steroidal anti-inflammatories, statins, aprotinin, heparin, steroids, lignocaine, ketamine, minocycline, N-Acetylcysteine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neuroprotection</td>
<td>Barbiturates, Dexmedetomidine, Xenon</td>
</tr>
</tbody>
</table>

**Table 3. Medications Commonly Given by Anesthesiologists That Should Be Avoided or Used With Caution In Patients Over 65 Years of Age**

<table>
<thead>
<tr>
<th>Medication or Class of Medication</th>
<th>Examples</th>
<th>Rationale for Avoiding</th>
</tr>
</thead>
<tbody>
<tr>
<td>First-generation antihistamines</td>
<td>Diphenhydramine</td>
<td>Central anticholinergic effects</td>
</tr>
<tr>
<td>Phenothiazine-type antihistamines</td>
<td>Promethazine</td>
<td>Central anticholinergic effects</td>
</tr>
<tr>
<td>Antispasmodics/anticholinergics</td>
<td>Atropine, scopolamine</td>
<td>Central anticholinergic effects</td>
</tr>
<tr>
<td>Antipsychotics (first and second generation)</td>
<td>Haloperidol</td>
<td>Risk of cognitive impairment, delirium, neuroleptic malignant syndrome, tardive dyskinesia</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>Midazolam, diazepam</td>
<td>Risk of cognitive impairment, delirium, psychosis</td>
</tr>
<tr>
<td>Corticosteroids</td>
<td>Hydrocortisone, methylprednisolone</td>
<td>Risk of cognitive impairment, delirium</td>
</tr>
<tr>
<td>H2 receptor antagonists</td>
<td>Ranitidine</td>
<td>Extrapyramidal effects</td>
</tr>
<tr>
<td>Metoclopramide</td>
<td></td>
<td>Anticholinergic effects</td>
</tr>
<tr>
<td>Neostigmine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skeletal muscle relaxants</td>
<td>Cyclobenzapine</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviation: H2, histamine 2 receptor.

High risk patients
- Age > 65y
- Pre-existing cognitive impairment
- Suspected cognitive impairment
- Previous stroke
- Poor functional status
- Major surgery
- Predicted anaesthetic time > 1.5 h
- Risk of postoperative respiratory complications

Identification

Optimise chronic disease

Hearing/visual/other functional aids

Prehabilitation?

Alcohol/smoking cessation

Correct anaemia & electrolytes

Optimisation

Information & counselling

Surgical options

Consent

Depth of anaesthesia monitoring

Adequate analgesia

Maintain blood pressure & oxygenation

Continue pertinent medications

Avoid high risk medications

Prevention
Beyond Emergence: Understanding Postoperative Cognitive Dysfunction (POCD)

POCD has historically been limited to scientific observation and research whereas the spectrum of mild cognitive impairment (MCI) and dementia in the general (non-surgical) population has evolved into well-defined clinical, functional and prognostic constructs.

The cause of POCD is unclear but it has some consistent associations:
- Age
- Preexisting MCI
- Fewer years of education
- POCD may be related to an inflammatory process
- Susceptible patients are most commonly those with dementia and cerebrovascular disease
- Incidence of POCD after non-cardiac surgery.

POCD is most often associated with hypotension or hypoxemia
- The incidence of POCD is similar for cardiac surgery, total joint surgery and coronary angiography

It is unclear whether the duration of exposure to anesthesia matters.

A multidisciplinary international working group established in 2015 recommends incorporating the nomenclature for cognitive decline as used in other disciplines into the perioperative period. They recommend the term perioperative neurocognitive disorder.
CONCLUSION

Perioperative neurocognitive disorders are fast becoming an area of concern with a 10% to 40% risk of patients developing it. The pathophysiological process of the disease is vast, and research is still on going with primary suggestion that it is from a disinhibited inflammatory response.

No biomarkers have been validated to date and no imaging test has been shown to diagnose the disease. Multiple risk factors exist with age above sixty-five years being the highest incidence. There still are no standardised risk stratification protocols and no specific diagnostic tests. The morbidity associated with neurocognitive disorders are detrimental to patients, care givers and the health system. This mandates that we prevent and treat the progression of the disease. This will require ongoing research into the pathophysiology and possible treatment options.

Patients and family counselling need to be promoted preoperatively to alleviate the stress associated with the burden of disease and give patients the autonomy about the surgery and implications of cognitive dysfunction post-surgery.

Of note, contrary to what was previously suggested by previous research the choice of anaesthesia does not show any difference in outcomes. It is suggested that his may be a failure of resilience to perioperative stress and pre-operative cognitive reinforcement may be the direction to take to prevent and manage neurocognitive disorders.
REFERENCES


