Anaesthesia, amnesia and harm

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ABSTRACT
Anaesthesia causes unconsciousness by suppressing neural mechanisms mediating arousal and awareness. It also causes amnesia by disrupting mechanisms of memory consolidation. Some patients under general anaesthesia unexpectedly become aware during surgery and form a traumatic memory of their experience. After describing the neural underpinning of phenomenal consciousness and memory, I examine the respects in which patients who experience anaesthesia awareness can be harmed by it. In cases where awareness is detected intraoperatively, I consider whether an anaesthetist would be justified in administering a drug to prevent a memory of the experience, as well as reasons for and against preoperatively informing patients of the possibility of awareness. In cases where awareness is reported postoperatively, I consider reasons for taking a drug to erase a memory of awareness against reasons for retaining the memory. A decision to take or decline such a drug would be informed by the potential harm of these memories and the potential benefits and risks of drugs intended to erase them.

INTRODUCTION
General anaesthesia causes unconsciousness by suppressing neural mechanisms mediating arousal and awareness.\(^1\) Anaesthetics also cause amnesia by disrupting mechanisms of memory consolidation.\(^2\) A patient under an anaesthetic or sedative during surgery may have some level of awareness but retain no memory of the experience. So, while anaesthetics and sedatives can induce unconsciousness and amnesia, their effects on awareness and memory are separable.

Some patients under general anaesthesia unexpectedly become aware during surgery. Awareness in these cases may involve being responsive without recall or also recalling what one experienced while responsive. Unintended intraoperative awareness is the experience and explicit recall of sensory perceptions during surgery. The incidence is estimated to be between 1 and 2 per 1000 cases.\(^3\) The harm associated with recall can be substantial. One study indicates that as many as 70% of these patients develop post-traumatic stress disorder (PTSD).\(^4\) Some who become aware can be traumatised from pain. Others experience anxiety or panic in the presence or absence of pain. In all of these cases, patients may not be able to communicate their experience to anaesthetists and surgeons. Intraoperative awareness is difficult to detect and prevent because of the complexity of the neurobiological basis of consciousness. This underscores the inadequacy of the recommendation that awareness can be prevented by more vigilant observation and use of monitoring devices. Unintended awareness may be due to underdosing of an anaesthetic or sedative, or from falling concentrations of the drugs during or near the end of surgery. Depending on the effect site concentration of the drug, some anaesthetised patients can be awakened and follow commands through the isolated forearm technique without recalling the experience.\(^5\) Persons are harmed when events or states of affairs defeat their interests and make them worse off.\(^6\) Pain and suffering experienced under general anaesthesia and recalling this experience are examples of harm since we all have an interest in avoiding them.

After describing the brain mechanisms mediating awareness and memory, I consider whether and in what respects patients who experience intraoperative awareness can be harmed by it. I then consider pharmacological interventions that could prevent or mitigate harm. The general ethical question can be broken down into more specific ethical questions. If awareness is detected intraoperatively, then would the anaesthetist be justified in administering a drug to prevent or erase a memory of the experience? Would the probability of intraoperative awareness and harm from its recall be significant enough to justify informing the patient of it preoperatively and asking him whether he would want induced amnesia if it were detected? How would any benefit to the patient from asking this question be weighed against the harm from the anxiety generated by the thought of becoming aware during surgery? If awareness was reported postoperatively, then how would the patient weigh reasons for taking a memory-erasing drug against reasons for declining the drug to retain a memory of the experience?

NEURAL MECHANISMS OF AWARENESS AND MEMORY
Neurologists Fred Plum and Jerome Posner divided consciousness into two components: wakefulness or alertness, mediated by the brainstem ascending reticular activating system (ARAS) and its projections to the thalamus; and awareness of self and environment mediated by the ARAS, its projections to the thalamus and further projections to networks in the cerebral cortex (neocortex).\(^7\) Consciousness is a graded property (p. 880).\(^1\) The neural correlates of consciousness are neither fully on nor fully off but maintain a resting potential prior to their inhibitory or excitatory action.\(^8\) Whereas general anaesthesia can quickly cause unconsciousness, emergence from unconsciousness to awareness occurs in degrees. There are competing theories about the neural mechanisms of consciousness and how they are suppressed during anaesthesia.\(^9\) For the purpose of this paper, I simply assume that anaesthetics suppress consciousness and that some patients become aware in spite of them.
Ned Block distinguishes two types of conscious awareness: phenomenal consciousness and access consciousness. He defines the first as “experience” and says that “the phenomenally conscious aspect of a state is what it is like to be in that state”. In contrast, access consciousness consists in information processing and its “availability for use in reasoning and rationally guiding speech and action”. Access consciousness is relevant to the question of whether minimally conscious patients can communicate wishes about life-sustaining treatment to families and medical practitioners. Yet what matters in intraoperative awareness is not whether a patient can process information but instead whether the patient can perceive and suffer from pain, anxiety or panic and have a memory of these phenomenal states. Thus phenomenal consciousness is the relevant type of consciousness at issue here. There can be no access consciousness without phenomenal consciousness in any experience of pain or suffering. Indeed, Block argues that we may sometimes experience pain without access consciousness.

Depending on their concentration and half-life, inhaled or infused anaesthetics cause amnesia by interfering with neural mechanisms regulating the formation and storage of memory. They allow the encoding of information but cause it to be forgotten. This explains why many patients emerging from anaesthesia during a procedure later report having no recall of being aware. If an anaesthetic has an amnestic effect when a patient becomes aware, then any encoding and consolidation would only be for a brief period, and the experience would not go into long-term memory storage. Lower concentrations of anaesthesia are effective in preventing memory consolidation. Administering a higher concentration of an anaesthetic during surgery would cause a return to unconsciousness but would not restore the amnestic effect.

If a surgical patient became aware and was in distress, then the anaesthetist could infuse a drug such as midazolam into her. This or other benzodiazepines could calm the patient by preventing an anxious response to becoming aware. These drugs do not cause retrograde amnesia and thus do not erase memories that already have been encoded and consolidated. Midazolam could cause anterograde amnesia in a dose-responsive manner and prevent the formation of a memory of awareness if it was administered preoperatively before general anaesthesia. Presumably, though, there would have to be some risk of intraoperative awareness for the drug to be used for this preventive purpose. In such a case, an anaesthetist could administer midazolam to both calm the patient and prevent a memory of awareness because of the drug’s anxiolytic and amnestic effects. The sedative propofol could block the consolidation of a memory if it was administered intraoperatively as soon as awareness was detected, assuming that it could be detected (p. 290).

If anaesthesia awareness is not known until the patient reports it postoperatively, then one way to mitigate harm would be to take propranolol. This drug does not induce amnesia but can attenuate the emotional content of a traumatic memory. One could recall an experience without being overwhelmed by any negative valence attached to it. Yet propranolol is most effective in weakening the emotional content of a memory when it is administered within 6 h after the event. Also, the therapeutic effects of propranolol for PTSD are variable. Not everyone taking the drug has reported a reduction in symptoms. Even when it is effective, propranolol would mitigate but not prevent harm because it would leave the neural representation of the memory intact.

A potentially more effective postoperative intervention would be to infuse a protein synthesis inhibitor such as anisomycin into the lateral and basal amygdala when the memory was being retrieved by the patient. Research on memory erasure has been limited to animal models. It is not yet known what the clinically relevant effects of this intervention might be in humans. In the hypothetical scenario described here, an amnesia-inducing drug would be offered as an innovative intervention. Because encoding, consolidation and reconsolidation of fear memories in the amygdala require protein synthesis, inhibiting protein synthesis theoretically could disrupt long-term retention of the memory and extinguish it. It would not merely dampen its emotional content but erase the actual representation of trauma in the brain. There would be no risk of recalling a traumatic memory after this intervention because there would be no memory to recall.

Fear memories have neural representations in both the amygdala and neocortex corresponding to their affective and cognitive components. The representation in the amygdala may be an implicit rather than explicit memory and fall outside of phenomenal consciousness. For this reason, implicit memories of fearful or traumatic events may not be amenable to cognitive therapy. Implicit fear memories could cause greater harm than explicit fear memories if they disposed one to automatic excessive fear reactivity to stimuli misperceived as threatening. A protein synthesis inhibitor might erase both explicit and implicit memories. Removal of the neural representation in the amygdala while leaving the one in the neocortex intact may be sufficient to prevent harm. The representation in the amygdala has emotionally charged content, and it is this content that can cause psychopathologies such as generalised anxiety, panic disorder and PTSD. This representation may be more amenable to manipulation because memory storage in the amygdala is more localised than the more distributed pattern of memory storage in the neocortex. Like the effects of propranolol on the emotional content of memory, however, time is a crucial factor in pharmacologically induced amnesia. Protein synthesis inhibitors are most effective in disrupting reconsolidation when they are taken within a few hours of experiencing a particular stimulus. If a patient did not recall and report intraoperative awareness until days or weeks after it occurred, then it would be difficult to extinguish what could be a firmly embedded traumatic memory of that experience. This is significant because only 50% of affected patients report awareness immediately after surgery and may not report it up to a month after the event, if not later.

A major challenge in using amnesia-inducing drugs would be to make them selective enough to extinguish maladaptive or pathological memories while leaving adaptive memories intact. It is unclear whether they would have an expansive effect on other memories or how this could be controlled. The selectivity, safety and efficacy of these interventions need to be considered by anaesthetists administering an amnesia-inducing agent during or after an episode of anaesthesia awareness. These factors would also have to be considered by patients presented with a choice of retaining or erasing a memory of awareness when they recall it. Still, the problem of anaesthesia awareness cannot be resolved simply by administering a higher dose of an anaesthetic or a memory-blocking or memory-erasing drug. More fundamentally, the problem arises and may not always be prevented because phenomenal consciousness cannot be detected in every case.

**Awareness and Harm**

Suppose that a patient unexpectedly becomes aware during surgery despite being anaesthetised and experiences pain. The
Awareness is detected, and analgesia and a higher dose of the anaesthetic are administered. He quickly returns to unconsciousness until the surgery ends, and he gradually regains consciousness. Postoperatively, he does not recall being aware or feeling any pain. Was the patient harmed by the experience?

Distinct brain networks mediate pain perception: a sensory network consisting of the lateral thalamic nuclei and somatosensory and parietal cortices; and an affective network consisting of the medial thalamus, anterior cingulate and prefrontal cortices. These two networks mediate physical and emotional aspects of pain.20 Some responses to noxious stimuli might indicate activation of the sensory network. But as Athena Demertzi and Steven Laureys point out, most of the behaviours associated with noxious stimulation—eyes opening, quickened breathing, increased heart rate and blood pressure—are of subcortical origin and “do not necessarily reflect conscious perception of pain” (p. 92).20 Demertzi and Laureys further divide pain into nociception and suffering (p. 95).20 The first refers to responses to noxious peripheral stimulation, while the second refers to the emotional experience of this stimulation. Although these definitions have been provided in the context of minimally conscious patients following traumatic brain injury, they also apply to cases of anaesthetised patients emerging from unconsciousness to consciousness.

Many neuroscientists assume that nociception is evidence of pain and that this recommends analgesia. Yet just because the nociceptive network is active and the patient consciously perceives pain does not imply that the affective network mediating emotional reactions to pain is also active. Any pain can harm one who experiences it, especially the pain from being cut and cauterised during surgery. Yet what is more harmful than feeling pain is one’s emotional response to it, which can cause suffering. If noxious stimulation activated the sensory network but not also the affective network during anaesthesia awareness, then the patient could feel pain but not have a negative affective response to it. He would not suffer and be harmed in this respect. Any harm to the patient would be limited to the period when he felt pain.

In addition to being caused by emotional responses to pain, suffering may also be caused by anxiety in anticipating pain.21 Anticipation of pain can cause mood changes and behavioural adaptations associated with suffering. To suffer in this way, a patient experiencing intraoperative awareness would have to form and retain a memory of perceiving pain while he was aware. The memory of a painful experience can cause the fearful anticipation of a continuation or new experience of pain. Suffering could be prevented by preventing the capacity to recall the pain. This could occur through the amnestic effects of the anaesthetic or a memory-blocking drug when the patient was aware. A drug’s effect of disrupting memory consolidation would cause a patient to forget a painful episode and prevent her from suffering from it in the sense that it would prevent her from anticipating additional episodes of the same type. In some cases of anaesthesia awareness, a patient who has been given analgesia may not feel pain but experience and suffer from panic. Here too the amnestic effect of the anaesthetic could make the patient forget the panic and prevent the development of adverse psychological effects from the experience.

The cases with the greatest potential for harm are those with unintended and unexpected full intraoperative awareness and complete recall. These often involve patients with compromised physiological functions who can only tolerate a lower dose of an anaesthetic. The risk of harm is greatest when the surgery involves neuromuscular blockade. Patients are given a paralysing drug to prevent them from moving and not interfere with intubation and the ventilator that breathes for them under general anaesthesia. Awareness in these cases can be especially traumatic because the patient is unable to indicate that she is aware. When the patient cannot open her eyes, the experience is similar to that of patients with severe locked-in syndrome. Devices such as the bispectral index (BIS) and the end-tidal anaesthetic-agent concentration (ETAC) monitor may fail to detect activation of subcortical and cortical circuits mediating consciousness. Because these devices do not measure consciousness, there may be no objective indices of anaesthesia awareness. When such a patient becomes aware, he may panic from the feeling of suffocation in not being able to breathe on his own. This feeling and the experienced loss of bodily control can be intensified by the lack of proprioceptive and somatosensory feedback from the body to the brain, given the paralysing effect of the drug. Local anaesthetics blocking pain pathways in limbs have much more limited effects on these types of feedback because they involve only parts of rather than the entire body. The risk of panic could be minimised if the anaesthetist preoperatively informed the patient that he would receive a paralysing drug, provided that he formed and retained a memory of this information. This could be a necessary component of the patient’s informed consent to surgery. But because awareness is not intended or expected in these cases, becoming aware could cause the patient to perceive it as a life-threatening experience and develop a traumatic memory of it.

Allowing different periods and levels of awareness during some procedures involving general anaesthesia or sedation may avoid unwanted intraoperative and postoperative outcomes because it allows interaction between the patient and the anaesthetist. Informing the patient preoperatively that she will be aware during a procedure for this purpose might generate an expectation of awareness that would allay any anxiety or distress she might experience when she becomes aware. In expected awareness, fear and panic systems in the amygdala and brainstem might be primed for the experience and not be activated to the same degree as they would be in a case of unintended and unexpected awareness. But allowing patients to be awakened would not be feasible when they are being cut and cauterised. So, the instances in which awareness was allowed during surgery would be limited.

In outpatient procedures such as biopsies and endoscopies involving amnestic sedatives, a physician may tell a patient that she may feel minor pain or discomfort but not have a memory of it. This could influence the patient’s expectation, allay any anticipatory anxiety and mitigate her perception of and emotional response to any pain she might feel during the procedure. Surgery with general anaesthesia is significantly different. It would not be appropriate to tell a patient that any pain felt during major surgery would not be bad for her because she would not remember it. This is because the experience of pain or panic would not be expected and should not occur, the degree of pain or panic would be much greater than it would be in an outpatient procedure, and it could not be predicted whether the patient would or would not recall it.

Given the potential of unintended intraoperative awareness, should the anaesthetist inform the patient preoperatively that she might become aware? Some would argue that the higher probability of harm from preoperative anxiety and the lower probability of intraoperative awareness would provide a reason against the anaesthetist informing the patient of something that might not occur. Others would argue that patients should be informed of the probability of any untoward events that might
occur during surgery. Limiting information out of concern about preoperative anxiety could be unduly paternalistic. More importantly, it could preclude interventions that could prevent long-term harm to the patient from these events. Before surgery, a patient could conditionally consent to these interventions to prevent memory consolidation on the basis of this information. Whether the patient should or should not be informed of the probability of intraoperative awareness depends on how one interprets informed consent and respect for patient autonomy. A stronger interpretation would warrant telling the patient of all possible scenarios while emphasizing the low probability of awareness. A weaker interpretation would warrant telling the patient of only scenarios likely to occur, and this could rule out any mention of intraoperative awareness. Not all patients want to be fully informed of all events that might occur during a procedure. Some want more information than others. How much information should be given to the patient could be judged on the basis of discussion between the anaesthetist and patient before surgery. This would depend on whether the patient was deemed to be at risk of becoming aware. It would also depend on whether drugs could be administered preoperatively or intraoperatively to prevent or erase a memory of awareness if it did occur.

There is variability among patients in their mechanisms of memory consolidation and reconsolidation. Memories of traumatic experiences may be consciously accessible to some patients but not others, and this may be a function of gene expression in the brain or comorbid neuropsychiatric conditions. Factors other than awareness itself could influence whether a patient developed a pathological fear memory of it. The core problem is the difficulty in ascertaining whether a patient is or was aware during a procedure. A related problem is ascertaining whether an anaesthetic causes amnesia despite periods of awareness. Some patients postoperatively deny being aware during a procedure despite following commands and carrying on a conversation with the surgeon and anaesthetist. These cases should be distinguished from absence seizures, where a patient may follow commands despite severely diminished or absent awareness. Cases of behaviour indicating awareness illustrate that retrospective oblivion is not proof of lack of awareness. Without an accurate report of awareness and without an accurate report of awareness, it may be impossible to know whether or at what level the patient was phenomenally conscious.

This problem is ethically significant because a patient may experience anaesthesia awareness that is not objectively detectable by observation or monitoring devices. She may experience pain, distress or panic and form a memory of these phenomenal states yet not retrieve the memory until considerably later. By then it may not be possible to erase the memory because it would be embedded beyond the point where a drug could disrupt consolidation and reconsolidation. Thus, detection of awareness during surgery or reporting it immediately thereafter would be critical for an intervention to prevent what could be permanently harmful effects of the memory. The concern is not just with the intraoperative period when a patient might experience pain and distress but also the postoperative period and the potential to develop a psychiatric disorder from a memory of an emotionally charged intraoperative experience. Let us consider some pharmacological interventions that might prevent the retention of such a memory and the harm associated with it.

THE ETHICS OF INDUCED AMNESIA

If a patient undergoing surgery showed signs of being aware, then a higher dose of the anaesthetic could be given to the patient to cause a return to unconsciousness. Yet too much anaesthesia or sedation can be as harmful as too little. In addition to potential adverse effects on cardiorespiratory function, higher concentrations could suppress some cognitive functions and result in cognitive impairment that could last for days, months or even years after the surgery. Deep sedation, for example, has been associated with a high incidence of postoperative delirium. Nor would a higher concentration of the anaesthetic erase a memory of being aware. While anaesthetics can interfere with memory consolidation, they do not affect memories that have been reconsolidated. But preoperative infusion of midazolam could prevent the formation of a memory of awareness. Without a memory of the experience, any pain or distress would be limited to the time when the patient felt them and would not be recalled after the procedure.

Would the anaesthetist be ethically justified in administering a memory-blocking drug if the patient did not consent to it? If becoming conscious was unforeseeable, then administering such a drug would be justified to prevent psychological sequelae from remembering it. Provided that this intervention did not have any known or foreseeable negative consequences and did not interfere with other memory systems, most patients would probably want it. Not administering the drug might be judged as a negligent omission, though this would require a clearly quantified risk of awareness for a particular patient.

In cases where a memory of anaesthia awareness could not be prevented by a preoperative infusion of midazolam or an intraoperative infusion of propofol, a protein synthesis inhibitor could be given to the patient after the procedure to disrupt reconsolidation and possibly erase the memory. Like other memories, it would be more labile and amenable to manipulation when the patient was retrieving it during recall. As noted,
however, the potential of achieving this effect would diminish the longer the period was between consolidation and retrieval. If this period was brief and the neural representation of the fear memory was localised in the amygdala, then it might be possible for such a drug to target and extinguish it. Directly targeting the memory and inducing amnesia would be preferable to retaining it and only dampening its emotional content with a noradrenergic-blocking drug such as propranolol. This could prevent psychological sequelae by eliminating not just some of its content but the entire memory. There would be no residual emotional associations with the memory if the drug erased its emotional representation in the brain. This intervention would also be superior to cognitive behavioural therapy, where repeatedly describing the experience to cope with it could reactivate rather than attenuate the negative valence of the memory. PTSD, panic and generalised anxiety are disorders of memory traceable to a particular stimulus. Erasing the memory of the stimulus would be a way of preventing the disorder and thereby preventing harm.

Nevertheless, pharmacologically induced amnesia would have to be selective enough not to disrupt memory mechanisms mediating normal fear and panic responses in the amygdala and brainstem. Not all fear memories are pathological. Many are adaptive and critical for survival. The inability to retain memories of some fearful events can be as harmful as the ability to retain memories of others. Taking a drug to prevent or control one disorder could come at the cost of creating another. The encoding and consolidation of fear memories involves interaction between the amygdala and hippocampal complex.25 Targeting these memories would have to be selective enough not to inhibit protein synthesis necessary for encoding, consolidation and reconsolidation of emotionally positive or neutral episodic memories in the hippocampal complex and its projections to storage sites in the neocortex. It would also have to avoid implicit procedural memory mechanisms necessary for learning and applying motor skills. Any attempt to erase a memory of a fearful experience would require careful consideration of the neurochemical effects of the drug and how it might affect other memory systems.

If the drug could precisely target a small region of the brain such as the amygdala, and if the memory was localised in this region, then it might be possible to erase the representation of the memory and minimise the risk of the drug affecting other memories. But the specificity of the drugs cannot be assumed. The patient would have to weigh the risk of possibly developing a psychopathology by not taking the drug against the risk of taking it and possibly causing unwanted side effects on other memories. Weighing these options would be complicated by uncertainty about what the effects of taking or declining a memory-erasing drug would be. Respect for autonomy would suggest that the patient should have the right to make this decision since he would have to live with its consequences. Given the narrow temporal interval for memory erasure, the patient reporting awareness would have to quickly process information presented to him by the anaesthetist. This temporal factor and the uncertainty surrounding the information could make this a difficult decision. Yet difficult decisions about how interventions might affect the brain and mind do not warrant limiting patients’ autonomy by withholding relevant information from them. If a drug that could erase a memory of a traumatic intraoperative experience was available, then a patient who had this experience and a memory of it should have the right to know of its availability and take the drug if she judged that it was in her best interest to do so. Provided that the patient was competent enough to weigh the potential benefit against the potential cost, respect for her autonomy would obligate the anaesthetist to present this information about the drug and its known and unknown effects to her. Stating that the drug had unknown effects would not mean that the anaesthetist would fail to discharge a duty of disclosure or nonmaleficence. Because there are no data from clinical trials on the safety or efficacy of protein synthesis inhibitors for memory erasure in humans, there would be genuine uncertainty about how the drug would affect a person’s brain and mind.

Alternatively, the patient could be asked preoperatively whether he would want pharmacologically induced amnesia in case of intraoperative awareness.26 This assumes that awareness could be detected and that a drug would have a particular amnestic effect on a particular memory. These are critical assumptions. Even if monitoring devices improved to the point where they could detect awareness and a drug could selectively erase a memory of it, mentioning the possibility of becoming conscious during surgery could harm the patient by causing him to anxiously anticipate it. But the potential harm from preoperative anxiety would have to be weighed against the potential harm of developing a memory from intraoperative awareness and losing the opportunity to prevent it. A decision about what should be done in this case would have to be informed by the fact that not all patients experiencing awareness develop a memory or a psychiatric disorder from it. Still, providing information about the risk of awareness and recall would give the patient some control of what she might experience during a procedure. This reason for informing the patient of the possibility of intervening intraoperatively to prevent memory consolidation would outweigh any competing reason for withholding the information. The patient’s control would transfer from the time when she consented to or refused such an intervention to the time when she was anaesthetised and had no decisional capacity.

Ethical issues may arise not from the experience of anaesthesia awareness itself but from events occurring while a patient is aware. Some patients may hear disturbing comments from a surgeon or anaesthetist that were not intended to be heard. Suppose that a patient with colorectal cancer undergoes a resection of the large intestine. The surgeon comments on the poor prognosis after closing the incision, and the patient hears this as the anaesthetist wears off. A memory of these words could add to the suffering the patient will endure with the disease after the surgery. If the anaesthetist detected that the patient was aware and heard the surgeon, then infusing an amnestic drug to disrupt consolidation of a memory of the comment could mitigate postoperative harm. A similar action was performed in an actual case. A woman undergoing a biopsy while conscious heard a comment about a tissue sample being cancerous. The comment provoked an emotionally charged outburst from the patient. The anaesthetist then immediately infused the patient with propofol, not to sedate her, but to prevent consolidation of a memory of the comment. She could not recall the experience after the procedure.27 Whether the anaesthetist was obligated to inform the patient of this intervention and, if so, how the information should have been presented are separate questions. As an action intended to prevent a memory of an unforeseeable traumatic event with potentially harmful consequences, the anaesthetist’s memory-erasing act in these hypothetical and actual cases seems justified. The patient was not given a choice about the propofol. Although she was not consciously sedated when the incident occurred, her heightened emotional state probably would have interfered with her cognitive capacity to make an informed decision. Also, given the narrow time interval...
for disrupting memory consolidation with propofol, asking the patient during the procedure if she wanted this intervention would extend beyond this interval and preclude its intended effect. For these reasons, asking a patient in this situation whether she wanted it would not be feasible.

Cases in which an amnesia-inducing drug could be given post-operatively may seem less ethically problematic because the patient recalling the memory would be able to choose to take or decline the drug. But the limited time for the drug to erase the memory could constrain the patient’s deliberation and decision. If awareness is not detected during surgery and not known until the patient reports it later, then this would allow the memory of the experience to consolidate and reconsolidate in the patient’s brain. While a patient should have the choice to erase a memory of being aware, it is clearly preferable to prevent the memory from forming in the first place, especially given the difficulty in manipulating memories in long-term storage. This provides a stronger reason for preoperatively informing the patient of the probability of awareness and intraoperative interventions to prevent the formation of a memory of the experience.

Some would argue that the value of erasing a memory of an offensive comment or criminal act on a patient’s body while she was aware during surgery would be outweighed by the value of retaining the memory to testify against the offender in a court of law. Yet psychologists and legal theorists have questioned the probative value of memory in eyewitness testimony. Memory is a reconstructive process that provides a fallible account of past events. The emotional content of a memory of a traumatic experience can make it vivid for the subject but may distort the actual details of the event when it is recounted. Distortion can increase with the passage of time. As Joyce Lacy and Craig Stark point out, “in the courtroom ‘memory’ is often misunderstood and undue assumptions are made about its veridicality”.

Consciousness comes in degrees. Full awareness and oblivion fall at opposite ends of a continuum. It is around the middle of this continuum where the most vexing empirical and normative questions about intraoperative awareness arise. It is also here that these same types of questions arise regarding manipulation of traumatic memories. Whether one forms a memory of being aware, and whether the memory captures the experience, depends on how the brain encodes, consolidates and reconsolidates information. These mechanisms can be disrupted by anaesthesia at an earlier time or by amnesia-inducing drugs at a later time. Because pharmacologically induced amnesia for traumatic memories and disorders of fear regulation is still speculative to some extent, questions about how individuals can benefit from or be harmed by it may not yet yield definitive answers. But given neuroscience’s increasing ability to manipulate human consciousness and memory, there is much to be learned from discussing these questions.

CONCLUSION
Since William Morton’s demonstration in 1846 that inhaled ether caused anaesthesia and amnesia, research in anaesthesia has done much to elucidate the neurophysiological underpinning of consciousness and memory. For patients under general anaesthesia or deep sedation, the boundary between consciousness and unconsciousness can be nebulous. Because consciousness cannot be directly observed but only indirectly inferred from observation, imaging or electrophysiology, and because these methods are fallible, there are cases where it cannot be known from outside observers whether a patient experiences intraoperative awareness. It may only be known from a post-operative report from the patient. In the absence of recall, if ontological and epistemological questions about awareness cannot be answered, then the normative question of whether the patient is harmed by it cannot be answered either.

When intraoperative awareness is detected during surgery, an anaesthetist would be justified in infusing a drug such as propofol or anisomycin to prevent the consolidation of a memory of this experience without the patient’s consent. Assuming that the drug was safe and effective, the anaesthetist would be acting to prevent potential harm to the patient from an unforeseeable state of affairs. The patient could be asked preoperatively if she wanted such an intervention in case of detected awareness. She could exercise her autonomy by indicating what she would want the anaesthetist to do or refrain from doing when the anaesthetic prevented her from acting. Some patients could be identified as being at greater risk of anaesthesia awareness than others. This could obligate anaesthetists to inform these patients of this risk and of possible interventions to prevent or minimise its effects. In cases where awareness was not detected intraoperatively but reported by the patient postoperatively, the patient should have the right to decide whether or not to take a drug to erase a memory of it. This decision would have to be informed by the fact that not all patients who have this experience develop psychopathologies, that the drug might not erase the memory and that it might adversely affect other memories. It would also have to be informed by the fact that not all memories are accurate representations of experiences and can be distorted by many factors.

Acknowledgements I am grateful to three anonymous reviewers and the editors of the Journal of Medical Ethics, Dominic Wilkinson and Julian Savulescu, for helpful comments on earlier versions of this article.

Competing interests None.

Provenance and peer review Commissioned; externally peer reviewed.

Correction notice This article has been corrected since it was published Online First. The page range in reference 28 has been amended to from ‘639–58’ to ‘649–58’.

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*J Med Ethics* 2014 40: 651-657 originally published online January 21, 2014
doi: 10.1136/medethics-2013-101543