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Higher Risk Hospital Based Maxillofacial Sedation for ASA 3 and ASA 4 Patients using Propofol Based Target Controlled Infusions

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Abstract

There is a paucity of information regarding the administration of Target Controlled Infusion (TCI) sedation to American Society of Anesthesiology (ASA) class 3 and 4 patients for maxillofacial procedures. We report a selection of ASA 3 and ASA 4 patients who recently underwent dental extractions by maxillofacial surgeons and were managed under TCI propofol sedation in mainstream hospital settings.

The utilisation of effective TCI propofol sedation, accompanied by an ultra short-acting narcotic (either alfentanil or remifentanil) given immediately prior to the administration of local anaesthetic (LA) by the surgeon, permits the avoidance of higher risk general anesthesia and its associated complications within this more infirm patient group.

Effective use of TCI sedation in ASA 3 and ASA 4 maxillofacial patients requires the provision of skills routinely employed for sedation in healthy patients, with the appropriate dosage modifications necessitated by age and associated patient co-morbidities. Properly administered in a hospital setting, TCI sedation with propofol accompanied with an ultra-short acting narcotic is safe and effective.

Keywords: Maxillofacial sedation; Dental sedation; Propofol; Target controlled infusion; Alfentanil; Remifentanil

Introduction

The ageing population represents a challenge to both maxillofacial/dental practitioners and anaesthesiologists. Many older patients present for dental extractions, in part related to their non-exposure to fluoridation of water as children. Cohorts born prior to the introduction of water fluoridation in Australia in the 1960's and 1970's have a significantly higher decayed, missing and filled teeth index (DMFT) compared with post fluoridation cohorts. The ageing process, accompanied by the development of medical co-morbidities, can present challengers to the delivery of both dental and anaesthesia care. The developing awareness of possible cognitive dysfunction in the elderly following general anaesthesia is also of increasing concern [1].

The practice of TCI sedation for dental surgery using TCI propofol accompanied by a narcotic is well established world-wide [2] with the exception of North America. In this domain, the United States of America (USA) Food and Drug Administration (FDA) never provided regulatory approval for the introduction of algorithmically based computerised delivery infusion systems. Hence the USA, in particular, lags far behind the rest of the world in the delivery of both TCI intravenous sedation (IVS) and TCI total intravenous anaesthesia (TIVA). American anaesthesia literature, arguably representative of the forefront of modern anaesthesia research, therefore provides a different emphasis in terms of sedation methods.

Over the past ten years we have safely sedated in hospital settings many maxillofacial patients with complex medical histories, using a slightly modified version of the TCI sedation method we routinely employ for healthy, ASA 1 and 2, patients in our office-based setting. The primary difference involved is the TCI dosage alterations required for these usually, but not always, older but always higher risk patients.

Our sedation technique is predicated upon the administration of an initial low dose propofol infusion to act both as an anxiolytic and anti-emetic. A bolus dose of narcotic is then introduced to counter the discomfort of local anaesthetic (LA) injection (2% lignocaine) by the surgeon, followed by progressively increasing levels of TCI propofol to achieve the desired degree of sedation.

The easiest narcotic to use is Alfentanil, although in some cases we do use remifentanil as both an introductory bolus and then, only very occasionally in this patient cohort, in an ongoing infusion manner. We consider the key to the delivery of safe TCI propofol sedation is the use of an effective nasal capnographic monitoring system. Capnography acts as an "early warning" system for the development of impending airway obstruction from over dosage, and a detectable change in the waveform occurs well before any reduction in pulse oximetry (SPO_2). Commercially available combined bi-basal capnograhy/oxygen (O_2) delivery systems are far more effective and reliable than systems "rigged up" by practitioners.

We use an interactive hand holding technique with the patients as we progressively move from an initial, primarily analgesic mode into a sedation mode. The patients are requested to squeeze the hand of the sedationist repeatedly if they wish to be more sedated, and by doing so the TCI propofol level is progressively increased to a level considered to be both safe and satisfactory for the patient. We have previously described our office-based sedation methods for healthy patients with alfentanil [3], remifentanil [4] and the evolution to our current technique [5].

In contrast with healthy, ASA 1 and ASA 2 patients, elderly and ASA3 and ASA 4 patients require far lower TCI propofol levels to achieve satisfactory sedation levels. Surprisingly, we find that the initial bolus doses of alfentanil, our preferred narcotic in the older or sicker patients, are not lowered to the same degree as that required of propofol. Both alfentanil and remifentanil provide effective analgesia, although the use of alfentanil, requiring no algorithmic input, is far easier.

Sedation process

All patients are contacted by the anaesthesiologist (DGW) 10-14 days prior to their scheduled surgery. Necessary laboratory tests are ordered and relevant medical records sourced. Instructions are given in relation to the pre-operative administration of current scheduled medications. Specific fasting guidelines are provided-in general, patients are instructed to sip on 200 millilitres (ml) of clear fluids per hour (hr) up until one hr before their scheduled arrival time at hospital.

Upon arrival at hospital, the patients receive a local anaesthetic patch to the dorsum of the left hand. Oral esomeprazole 40 milligrams (mg) is routinely administered, if not part of a pre-existing drug regime.

Immediately prior to the commencement of sedation, a 20gauge intravenous (IV) cannula is inserted at the topical local anaesthetic site.

Monitoring, appropriate for these patients' ASA status, is employed: SpO_2, electrocardiograph (ECG), nasal capnography via the combined bi-nasal /O_2 delivery system and blood pressure (BP) via a cuff applied to the non- infusion arm. The cycle time for the BP in ASA 3 and ASA 4 patients is initially set at 1 minute (min) but is then adjusted to a 2.5 min cycle time following the introduction of both narcotic analgesia and subsequent stable sedation. All patients receive supplemental nasal O_2 at 3 litre/min (l/min).

Utilising the interactive hand holding process, the targeted propofol infusion levels are adjusted using the Schnider model from an initial, baseline effect target (Cet) of around 0.4 microgram (mcg/ml) up to, if required, higher Cet levels. The Minto model in effect mode is used for remifentanil.

Patients are taken to a moderate level of sedation, corresponding to a modified Ramsay sedation level of no more than 4, (purposeful response to verbal commands at a loud conversational level), provided there is circulatory and respiratory stability.

We describe here seven ASA 3 or 4 patients recently sedated for dental extractions. All patients were considered, by virtue of their underlying co-morbidities, to be unsuitable for sedation in an office-based environment.

Case Reports

Case 1: Extraction of 46 and 47

Male: ASA 3

Age: 78 years, Height: 178centimeters (cm), Weight: 100 Kilograms (kg)

Basal Metabolic Index (BMI): 30 kilogram/meter^2 (kg/m^2)

Co-morbidities: Inoperable stage 3 carcinoma colon, Type 2 diabetes mellitus (DM), atrial fibrillation (AF), gastric reflux (GORD), hypertension (HT), vascular dementia.

Prior multiple deep venous thromboses (DVT) and one pulmonary embolus (PE).

Medications: Rivaroxaban 10 mg, Esomeprazole 40 mg, Amiodarone 200mg, Perindopril 10 mg.

Analgesia/Sedation: Initial propofol Cet 0.4mcg/ml (0.99mg/kg/hr)

Alfentanil 600 mcg bolus. No facial grimace to LA injection.

Propofol increased to Cet: 0.6mcg/ml (1.43mg/kg/hr)

0.9mcg/ml (2.14mg/kg/hr)

1.1mcg/ml (2.61mg/kg/hr)

Stable haemodynamics. Initial parameters: Mean BP: 84mmHg

Pulse rate: 67bpm

Spo_2%: 97-98%

Operative range: Mean BP: 84-92mmHg

Pulse rate: 67-81bpm

Spo_2: 99-100%

Total time in operating room (OR): 33 min

Patient was satisfied with sedation and surprised at the end of surgery that extractions had been performed.

Case 2: Extraction of 28 and 48

Male: ASA 4

Age: 27 years, Height: 149cm, Weight: 48.6kg BMI: 22 kg/m^2

Co-morbidities: Noonan syndrome (associated with short stature, cardiac problems, microagnathia and the potential for related airway issues)

Severe hypertrophic cardiomyopathy (HOCM), frequent ventricular ectopic beats (PVC) on holter monitor, microagnathia with obstructive sleep apnea (OSA). Stop-Bang score was 4/8.

Medications: Nil

Analgesia/Sedation: Cet propofol 0.8mcg/ml (3.5 mg/kg/hr)

Remifentanil bolus Cet 2.6nanograms/ml (ng/ml) (0.83mcg/kg).

Remifentanil discontinued after bolus dose

No facial grimace on injection of LA.

Cet propofol decreased to 0.7mcg/ml (2.74mg/kg/hr)

Cet propofol increased to 1.0mcg/ml (3.34 mg/kg/hr)

Stable haemodynamics. Initial parameters: Mean BP: 65mmHg

Pulse: 82bpm

Spo_2: 98%

Operative range: Mean BP: 65-75mmHg

Pulse rate: 82-97bpm

Spo_2: 98-99%

Total time in OR: 35 min

Amnestic at completion- surprised when the procedure was completed.

Case 3: Extraction of 12, 26 and 44

Male: ASA 3

Age: 88 years, Height: 168 cm, Weight: 78kg, BMI: 27.7kg/m^2

Co-morbidities: AMI 2005, AMI 2020. Cardiac stent to left anterior descending artery (LAD) 2020.

Echocardiogram 2023: Moderate-severely reduced global left ventricular (LV) systolic function and mild mitral regurgitation (MR).

Well controlled atrial fibrillation (AF).

Medications: apixaban 5 mg bd, bisoprolol 2.5mg bd, frusemide 40 mg daily.

Sedation: Cet propofol 0.4mcg/ml (0.88mg/kg/hr)

Alfentanil: 600 mcg

No reaction to LA injection

Cet propofol increased to 0.6mcg/ml

Cet propofol increased to 0.8mcg/ml

Stable haemodynamics. Initial parameters: Mean BP: 112 mmHg

Pulse rate: 73 bpm

Spo_2: 99%

Operative range: Mean BP: 101-108mmHg

Pulse rate: 75-85bpm

Spo_2: 99%

Total time in OR: 19 min.

Satisfied with procedure, but not amnestic.

Case 4: Extraction of 38

Female: ASA 3

Age: 49 years, Height: 158cm, Weight: 63kg, BMI: 25kg/m^2

Co-morbidities: autism/bipolar disorder with mild intellectual disability.

Echocardiogram: bicuspid aortic valve with moderate stenosis (mean gradient 36 mmHg).

Medications: aripiprazole 15 mg daily, rosuvastatin 10 mg daily.

Sedation/analgesia: Cet propofol 0.6mcg/ml (2.1mg/kg/hr)

Cet remifentanil 3.6ng/ml (0.70mcg/kg)

Remifentanil discontinued after bolus dose.

No reaction to injection LA.

Cet propofol progressively increased to 0.8mcg/ml (2.75mg/kg/hr)

Cet propofol increased to 1.0mcg/ml (3.40 mg/kg/hr)

Cet propofol increased to 1.2mcg/ml (4.26mg/kg/hr)

Stable haemodynamics. Initial parameters: Mean BP: 110 mmHg

Pulse rate: 82bpm

Spo_2: 98%

Operative range: Mean BP: 95-115mmHg

Pulse rate: 78-90bpm

Spo_2: 97-99%

Total time in OR: 30 min

Patient described amnesia towards end of the procedure and satisfied with sedation.

Case 5: Extraction of 36 and 37

Male: ASA 4

Age: 75 years, Height: 183cm, Weight: 100kg: BMI 30Kg/m^2

Co-Morbidities: Severe Multiple Sclerosis- diagnosed 1988.

Now has limited movement in left arm, no movement in right arm and wheelchair-bound re: paralysis of legs.

Right coronary artery (RCA) stent 2021.

Acute myocardial infarction (AMI) 4 months (mth) ago with coronary artery stent to LAD.

Ejection fraction (EF) 55%.

GORD.

Medications: oxycodone/naloxone 10 mg/5mg BD, atenolol 25 mg bd, gabapentin 100 mg qid, rabeprazole 20 mg daily.

Sedation/Analgesia: Cet propofol 0.4mcg/ml (0.85mg/kg/hr)

Alfentanil 800 mcg.

No reaction to LA injection.

Cet propofol increased to 0.7mcg/ml (1.48mg/kg/hr)

Cet propofol reduced (capnographic dampening) to 0.5mcg/ml (0.92 mg/kg/hr)

Cet propofol reduced to 0.3mcg/ml (0.53 mg/kg/hr)

Stable haemodynamics. Initial parameters: Mean BP: 82mmHg

Pulse rate: 61bpm

Spo_2: 96%

Operative range: Mean BP: 76-82mmHg

Pulse rate: 61-70bpm

Spo_2: 92-97%

Patient amnestic for word prompts mid and end procedure. Satisfied. BIS range: 97-80

Total time in OR: 33 mins

Case 6: Extraction of 37

Male: ASA 3

Age: 91years, Height: 177 cm, Weight: 88kg, BMI: 28 Kg/m^2

Co-morbidities: Bioprosthetic aortic valve replacement 2009- good cardiac function.

Hypertension.

Type 2 DM.

Mild anaemia- haemaglobin (Hb) 118g/L.

Moderate renal impairment (Cr 149umol/L, eGFR 35mls/min)

Dyslipidaemia

Medications: gliclazide 60 mg daily, metformin 1 gm bd, perindopril 10 mg daily, atorvastatin 40 mg daily.

Seadtion/Analgesia: Cet propofol 0.6mcg/ml (1.27 mg/kg/hr)

Cet remifentanil 2.8ng/ml (bolus 0.60 mcg/kg).

Remifentanil infusion discontinued after bolus dose.

No facial grimace with injection LA.

Cet propofol increased to 0.8mcg/ml (1.5mg/kg/hr)

Stable haemodynamics. Initial parameters: Mean BP: 97mmHg

Pulse rate: 72bpm

Spo_2: 100%

Operative Range: Mean BP: 93-103mmHg

Pulse rate: 72-83bpm

Spo_2: 100%

Total time in OR: 32 min

Patient aware throughout procedure but comfortable and satisfied with sedation.

Case 7: Extraction of 14,17,26,32 and 35

Male: ASA 4

Age 88 years, Height 162 cm, Weight 65 kg, BMI: 25kg/m^2

Co-morbidities: mitral valve replacement 2014.

ECG: atrial fibrillation with left axis deviation (LAD).

Patient dyspnoeic at rest with 2 pillow orthopnoea and swelling of ankles.

GORD.

Medications: digoxin 0.25 mg daily, frusemide 40 mg daily, spironolactone 20 mg daily, esomeprazole 40 mg daily, apixaban 10 mg daily.

Sedation/Analgesia: Cet propofol 0.3mcg/ml (0.74mg/kg/hr)

Alfentanil 350 mcg

No reaction to LA injection.

Cet propofol increased to 0.5mcg/ml (1.2mg/kg/hr)

Cet propofol increased to 0.7mcg/ml (1.65mg/kg/hr)

Cet propofol increased to 1.1 mcg/ml (2.54mg/kg/hr)

Patient demanding increased propofol.

Cet propofol increased to 1.5mcg/ml (3.4mg/ml)

Patient commenced snoring, procedure completed, and propofol discontinued.

Patient aware to word prompt mid procedure -Cet propofol 0.7mcg/ml

Patient unaware to word prompt end procedure -Cet propofol 1.5mcg/ml

Stable haemodynamics. Initial parameters: Mean BP: 116mmHg

Pulse: 74bpm

Spo_2: 100%

Operative range: Mean BP: 116-101mmHg.

Pulse rate: 74-90bpm

Spo_2: 100%

Total time in OR: 45 min

BIS: 98-87

Patient aware midway but not at completion of procedure- satisfied.

Discussion

Office-based maxillofacial sedation is usually thought to be suitable only for ASA 1 and ASA 2 patients [6]. Many office-based practices impose an age limit of around 70-75 years. This leaves an extremely large cohort of patients requiring dental work to be done under either LA alone, or in a mainstream hospital under the supervision of an anaesthesiologist.

We have sedated elderly or medically compromised, ASA 3 and ASA 4, maxillofacial patients in hospital settings for well over 10 yrs. On average we treat around 30-50 of these patients/yr. The anaesthetic technique we employ, being propofol based, evolved over a period of time but really took hold when we became aware of the work by Cashion et al. in 2016 [7] They reported on a propofol TCI office-based technique, albeit with the addition of midazolam and fentanyl and without always administering supplemental O_2.

We commenced an office-based sedation service shortly afterwards. Our primary intention was, and remains, to enable a fast-track patient discharge following surgery. With that in mind, we have always avoided the use of longer acting agents: midazolam, fentanyl and alpha 2 agonists. We practice with a philosophy of minimal drug exposure, in order to minimise the risk of severe side effects such as anaphylaxis.

Our standard, routine sedation technique administers four drugs only: propofol, a narcotic (either alfentanil or remifentanil), dexamethasone and a non-steroidal, anti-inflammatory agent (NSAID), parecoxib. Our aim is always to avoid benzodiazepine use and to facilitate an early awakening from the process. The sedation of patients requiring the supportive facilities of a mainstream hospital reflects these philosophies.

Elderly and ASA3 and ASA 4 patients require lower TCI propofol levels to achieve satisfactory sedation levels. Villeret et al. described the safe use of propofol sedation at a Cet of 0.9 mcg/ml in ASA 3 vascular patients under regional anaesthesia [8]. Surprisingly, we find that the initial bolus dose of alfentanil, our preferred narcotic in the older or sicker patients, is not lowered to the same proportionate degree as that of propofol. Both alfentanil and remifentanil provide effective analgesia for the injection of LA by the surgeon, although alfentanil, requiring no algorithmic input, is far easier to use, especially in the elderly.

We see no point in adding anti-emetics such as ondansetron- we are aware that other practitioners routinely do this when using propofol sedation. Our belief is that this utilisation, in conjunction with a propofol based TCI sedation process belies the understandings of propofol based anaesthesia and its profound anti-emetic properties. The available evidence in the presence of a propofol based anaesthetic supports the administration of only dexamethasone as an adjunctive anti-emetic. It must be given near the beginning of surgery. Serotonin antagonists should be left as a rescue treatment only. This philosophy was well documented in a comparative study of the rates of post-operative nausea and vomiting utilising six different anaesthesia techniques [9]. We have only ever had to resort to giving ondansetron on one occasion in over 3500 office-based and hospital administered sedations. Although the risk profile of anti-emetics such as ondansetron is low, the supporting data simply does not justify their routine use on either or both an efficacy/safety or cost basis.

Perhaps the most important aspects of successful TCI sedation, whether office or hospital based, are appropriate patient selection

coupled with the skill of the surgeon. TCI sedation is suitable for many maxillofacial procedures, such as some extractions, implants, sinus lifts and alveolar ridge augmentations. Some surgeons are adept at working under sedation. In particular, in older patients a high level of surgical expertise is required to achieve a successful sedation outcome. Extractions in this population are often more difficult. Patient selection is clearly important, although the elderly are usually compliant and more accepting of sedation conditions. Those younger patients, whom we would normally exclude from office-based procedures, such as heavy recreational drug abusers and very heavy smokers or vapers, are not representative of the older patient cohort.

If a patient requiring a hospital setting can be effectively managed under TCI sedation, we much prefer to utilise this management method rather than resorting to general anaesthesia. The patients described here are clearly at a higher risk of complications related to general anaesthesia than those of younger or healthier patients. There is certainly a higher risk of respiratory complications when the same procedures are performed under GA versus regional anaesthesia with accompanying sedation [10,11].

Our clear impression is that patients not only awaken more rapidly but are also cognitively more intact following TCI propofol sedation in comparison with those patients receiving GA. We have not encountered a case of post-operative delirium following propofol TCI sedation in maxillofacial patients, but have encountered this in elderly maxillofacial patients after general anaesthesia using volatile anaesthetic agents. It is possible that the avoidance of midazolam contributes in some manner to cognitive preservation following sedation of our patients. Midazolam has been considered to be one of the causes of delirium and post operative cognitive dysfunction (POCD) in the elderly [12,13]. As stated previously, we never use benzodiazepines as part of our sedation technique.

In comparison with younger or healthier patients, the TCI method with which we sedate our ASA 3 and ASA 4 patients differs primarily in the doses of especially propofol and whichever narcotic we use. A typical starting dose for TCI propofol in our office-based ASA 1 and ASA 2 patients is 0.6-1.0 mcg/ml. Our older or more infirm patients are usually commenced at a Cet of 0.4 mcg/ml, and if required, are stepped up to usually around 0.8-1.0 mcg/ml. We rarely go above 1.0 mcg/ml in this older cohort.

Amongst the patients described, patient 7 alone kept demanding more sedation and was taken to a propofol Cet of 1.5 mcg/ml at the very end of surgery. This proved to be excessive, and he commenced to snore. The snoring resolved quickly with the termination of the infusion, demonstrating the benefits of infusing propofol because of its short half-life.

In contrast, a typical propofol Cet end point for sedation in our office-based ASA 1 and ASA 2 patients is as high as 2.3-2.5 mcg/ml, sometimes going above 3.0 mcg/ml. Of note is that even at the lower Cet levels administered to older or infirm patients many, although seemingly co-operative during the procedure, seem to be amnestic as to events when the surgery has been completed.

We administer our narcotic of choice in a bolus dose tailored to the patient's age and weight. Essentially, our experience with healthy patients reflects that the narcotic dose required for effective analgesia at the time of LA injection by the surgeon is halved as the age increases from youthful adults to the very elderly. This concept is supported in the literature, with a 50% narcotic dosage reduction from age 20 years to 90 years [14]. Interestingly, the age of the patient rather than their ASA status seems to be a greater determinant in the required narcotic dosage reduction in order to achieve safe but effective analgesia. Again, the proportionate dose reduction with age seems to be much greater for propofol than for the narcotics.

As with our office-based patients, should apnoea occur following the administration of an ultra-short acting narcotic, the patients, provided responsive, will breathe on command. This is a primary reason as to why we always commence with very low, age dependant doses of propofol in both hospital and office-based settings.

Hypotension is a side effect of propofol that is frequently described in the anaesthesia literature, in relation to both general anaesthesia and sedation [15]. Notably, the definition of hypotension varies from author to author. Some describe falls below a defined point [16], whilst others include a certain percentage fall from baseline BP [17]. We have not found hypotension to be a major issue when propofol is given in age or condition appropriate TCI sedative doses. This finding is supported in ASA 1 and ASA 2 dental patients by Lobb et al. in their recent paper (2). Likewise, Villeret et al. [8] found no change in haemodynamic parameters in a group of elderly, ASA 3 vascular patients under regional anaesthesia sedated with TCI propofol from a Cet of 0.9 to 1.3mcg/ml (they cited mg/Litre).

In relation to maxillofacial/dental sedation only, excluding GA, with tempered doses of TCI propofol we remain guarded in relation to edicts such as "propofol remains the drug of choice for anaesthetist-delivered procedural sedation although concerns have been raised about its tendency to cause hypotension, especially in older people" [15].

Propofol certainly does cause venodilation and can cause bradycardia, but these side effects and consequent hypotension would seem to be more apparent when given in far larger TCI sedation doses than we use or for the induction of GA as part of a TIVA regime. Barents et al. [16] described a large series of patients, 65% of whom were ASA 2 or ASA 3, for gastroenterological, cardiac and radiologic procedures. They used fixed, lower doses of remifentanil (mean Cet 0.84ng/ml) and higher doses of propofol (mean Cet 2.6 mcg/ml) in an RN sedation programme. Their rates of severe complications (1.1%), significant complications (10.5%) and moderate complications (6%), with all groups exhibiting hypotension, might well be explained by the high propofol Cet's they used. Hypotension is well documented in propofol sedated colonoscopy patients, but it must be remembered that as a result of standard pre-operative bowel preparation these patients are significantly dehydrated and as such more susceptible to the effects of vasodilation.

We certainly do see reductions in BP following the introduction of propofol in very nervous patients or those with the so-called "white coat syndrome", but this is a reflection only of a return to more normal BP levels with the alleviation of anxiety upon commencement of sedation. Our preference, in starting at very low, primarily anxiolytic levels of propofol, is to ensue cardiovascular stability especially in the ASA 4 patients we are sedating.

Patient 5 demonstrates the synergistic respiratory depressant effects of pre-existing medications, in this case gabapentin and oxycodone/naloxone. This ASA 4 patient had severe MS and cardiac issues. At a propofol Cet of only 0.7 mcg/ml (propofol infusion of 1.48 mg/kg/hr) he began to show respiratory impairment via the capnograph trace. The propofol Cet was progressively lowered to 0.5 mcg/ml and then to 0.3 mcg/ml, with return to a normal capnographic trace. There were no changes in Spo_2 or cardiovascular haemodynamics.

This patient was additionally monitored with a Bispectral (BIS) electrode which indicated only light sedation (Bis level 80).

Nevertheless, even at these low propofol Cet's, he was clearly amnestic for events during the procedure, including several word prompts which he was asked to attempt to recall after the procedure was over. We do not usually utilise BIS monitoring, although we acknowledge that it is frequently used by some in sedation. Our experience with BIS monitoring is that it adds little benefit to the combination of direct patient and capnographic observation. Our aim is always to avoid sedation levels which result in either respiratory and/or haemodynamic compromise. We find that in the presence of good ventilation and a patient who remains responsive to verbal commands BIS does not add greatly to either safety or ease of sedation. We are also cognisant of cost. Recently we audited the cost of our sedation technique and found that a 30 min sedation procedure was adversely effected on a cost basis to the order of an additional 7% when a BIS electrode was applied.

All of the patients presented here were safely and effectively sedated on much lower propofol Cet's than are utilised for younger or healthier patients, yet all were satisfied with their sedation process and most seemed to be amnestic for at least portions of their time in surgery. The two patients who were definitely aware throughout the entire procedure (patient three and patient six) both expressed satisfaction with the process.

Conclusion

We consider that propofol based TCI sedation, with an accompanying initial ultra-short acting narcotic analgesic, provides both safety and comfort for high risk and older maxillofacial patients, and conditions facilitating the ease of surgery.

Author Contributions

Douglas G Wells: Conceptualisation, Formal Analysis, Validation - review and editing.

Andrew Bridgeman: Writing - review and editing.

Roland Barrowman: Writing - review and editing.

Declaration of Interests

The authors have no conflicts of interest to declare.

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