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Research Article

Adverse Events in Procedural Sedation for the Dental Chair: Analysis of 800 Patients Managed with the Association Midazolam/Fentanyl

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Abstract

Purpose: This study evaluated the adverse events [AEs] rates of sedation with midazolam and fentanyl in ambulatory patients undergoing oral surgery.

Methods: 844 consecutive patients aged 8-95 years, ASA class 1-4, undergoing oral surgery [multiple surgical extractions, wisdom teeths extractions, difficult conservative care, sinus lift, bone grafts] were sedated with small dosages of midazolam and fentanyl under full monitoring (ECG, NIBP [non invasive blood pressure], SaO2, et CO2] with the aim to maintain conscious sedation (CS) [Ramsay scale score 3-4]. AEs included episodes [number and/or duration] of hypertension, hypotension, tachycardia, bradycardia, hypercapnia, desaturation, sleepiness. AEs were correlated to demographic and study variables with a multivariate analysis .

Results: Patients were premedicated with various drugs before the induction of CS. Surgery lasted a mean of 118 +/- 55 min:mean midazolam and fentanyl dosage were 0.048 +/- 0.029 mg/kg and 0.79 +/- 0.60 microgr/kg respectively. Per cent of cases [%] occurrence of AEs was : desaturation [SaO2<90] 17.7% ,duration 2.5 +/- 8 min: hypertension [diastolic BP > 95 mmHg or BP >25% basal] 17.9, hypotension[systolic or diastolic <25% of basal] 3.7, bradycardia 4 [hr<50 or <25% of basal], tachycardia 0.6 [>25% of basal], hypercapnia 38.4 [etCO2 >40 mmHg], sleepiness 21 [Ramsay 5][duration 4.3 +/- 11 min]. All surgeries were completed and patients sent home with a responsible adult within 2 hours.

Hypertension hypotension, tachycardia occurrences were associated with ASA, age, dosages of midazolam .Bradycardia was associated with age. Desaturation cases,duration and episodes were linked to the drugs used [midazolam and fentanyl]. Sleepiness was accompanied by desaturation and hypotension and little influenced by drugs dosages.

Discussion: Aes were frequent but of relatively minor intensity and all responded to the approriate therapy. Titration of sedatives and analgesics was kept to a minimun compatible with the condition of the patient and the extent of surgery.

Safety in the dentist office can be maintained with the careful monitoring of the vital signs under this drug regimen [midazolam and fentanyl] assuring the maintenance of conscious sedation.Any derangement from the normal physiology must be timely and properly treated.

INTRODUCTION

Following the ban on the administration of general anesthesia in the dentist's office prompted by the UK government with the report "A conscious decision [1] prompted by a series of papers on morbidity and mortality on the dental chair [2,3] there has been a growing interest in the field of sedation and sedation plus analgesia for the dental chair, with the aim to render tolerable to an increasing number of patients the longer and more invasive procedures.

Quite few references in the dental literature specifically address the issue of the safety of sedation in the context of the dentist's office apart papers dealing with general recommendations [4] or special subgroups [5].

Data from the American Society of Anesthesiologists, Closed Claims database [6] suggest that anesthesia at remote locations poses a significant risk for the patient, particularly related to over sedation and inadequate oxygenation/ventilation during monitored anesthesia care [MAC].

While the published series of the closed claims analysis deals mainly with the most severe adverse events, like death or brain damage, it would be interesting to know which are the dangers, if any of the practice of analgesia-sedation for the dental chair.

Sedation is a continuum process [7] starting with full wakefullness with maintenance of the protective reflexes ,passing along minimal, then moderate, then deep sedation and arriving at general anesthesia, with a continuous increasing depression of all physiological systems when the patient vital signs need to be artificially supported;therefore a major concern for physicians

doing sedation is to maintain a balance between comfort for the patient [and surgeon alike] and safety,avoiding adverse events [AE's] which are still occurring during deep sedation and general anesthesia [8].

Adverse events [AEs] that may occur during procedural sedation include a host of problems and particularly respiratory depression and apnoea, hypoxemia, haemodynamic disturbances, nausea and vomiting that may lead to aspiration of gastric content into the lungs.

Quoting the Task Force Committee on AEs [9]. "Actual injury is usually averted by either spontaneous resolution of the event or by intervention of the sedation care provider. These events are often referred to as 'near misses' or 'close calls', but in fact, rarely pose any serious danger [permanent neurological injury or mortality] when managed by a skilled practitioner in an appropriate setting." Because AEs have been classified differently across the years and different institutions a recently appointed international task Force [ibidem [9] defined AE as a 'Unexpected and undesirable response[s] to medication [s] and medical intervention used to facilitate procedural sedation and analgesia that threaten or cause patient injury or discomfort'. Ae's were graded as major, intermediate and minor according to their grade of aggressiveness in endangering the patient and following the extent of the intervention required with the intention to treat the AE.

With the aim to contribute to the literature on safety for procedural sedation in the dentist office we present a case series of patients candidate to dental surgery in various dentist offices undergoing conscious sedation with midazolam and fentanyl.Ae's were classified according to the task force quoted above [ibidem] [9]. In particular attention was paid to haemodynamic derangements, [tachycardia, bradycardia, hypotension, hypertension], desaturation, as evidence of hypoxemia,hyercapnia as sign of respiratory depression.

Aim of the study has been to search the correlation, if any, between patient demographics, intraprocedural data and incidence of adverse effects.

MATERIALS AND METHODS

The study includes 844 patients operated upon by the same anesthesiologist [CM] between January 1 2010 and December 31 2016 in various dental offices ,mainly for multiple implants, extraction of wisdom teeth and/or conservative care.with or without bone graft.

Anesthetic aim was to maintain a state of conscious sedation ,evaluated every 5-10 min according to the Ramsay scale [10] and kept between the score 2-3[patiemt calm ,oriented,responding to commands, cooperative];any overshoot[Ramsay 4 or 5]was defined as "sleep" and considered an AE. Data of the patients were collected on a special sheet; vital signs were measured every 5/10 min and included HR derived from ECG or pulsoximeter], NIBP, SaO2, etCO2 [whenever possible]. EtCo2 was measured through silicone nasal prongs [Salter labs ^{R} divided cannulas] with a sidestream capnograph [NPB 70-75]. Patients were seated on a dental chair,maintained their own clothes and were covered with special surgical drapes on the chest and face .

The day before the procedure patients were reminded to refrain form solids for at least 6 hours; clear liquids were allowed until 2 hrs before the arrival in the office. On arrival in the dental suite patients filled in a detailed health questionnaire [often sent by email days in advance] and signed a consent form for conscious sedation; if time allowed, they were premedicated orally (Table 1). Patients were instructed to take at home all their usual medications including antihypertensive ,antiarrhythmic ,cardiotonic pills ;only metformin was avoided before surgery. Patients on vit K inhibitors were operated with INR <2.5 checked the day before or the morning of surgery. ASA or ticlodipine was not stopped when prescribed by the cardiologist for preexisting pathologies [TIA, AFs, DVT s etc.]. Patients were stratified according their physical status with the ASA classification [11]. Once seated on the dental chair, patients were attached to the monitors [Datex Cardiocap II,Datex Ohmeda S/5 light, Meditech] ecg continuous, [3 or 5 leads], NIBP every 5 min for at least the first 30 min, then every 5/10 min], SaO_2 continuously from finger tip or lobe of the right ear, $etCO_2$ continuously, if available; a suitable vein was cannulated [20-22g] [Jelco ^{R} or Terumo^{R}] in the hand or forearm and normal saline infused at a rate of approximately 1,5 ml/kg/h . Sedation [conscious] was cautiously induced with a small bolus of midazolam [1-3 mg] and fentanyl [25-50 microgr]; local analgesia was administered by the attending dentist as appropriate with articaine 4% or mepivacaine 2% with epinephrine when the patient appeared calm and cooperative [Ramsay score 2-3] .

Hypertension, hypotension,tachycardia ,bradycardia were defined as exceeding 25% of the basal values measured at the beginning of the procedure, following the premedication, if administered and before venous cannulation.

Hypercapnia was defined as $etCO_2$ >40 mmHg; its duration and/or number of episodes were noted.

Desaturation was defined as SaO2<90; its duration and /or number of episodes noted .

Diastolic pressure >95 mmHG >5 min was [also defined as hypertension] treated with clonidine 0.5-0.75 microgr/kg, i.v repeated after 30 min if ineffective.

HR <50 /min[also defined as bradycardia] was treated with atropine 0.5 mg.

All other complaints or symptoms [pain,nausea,etc] were treated at the discretion of the anesthesiologist:haloperidol 0.5-1 mg injected for nausea,ephedrine [5-10 mg] for hypotension,small boluses fentanyl [10-25 microgr] or meperidine [10-20 mg] or remifentanyl [10-20 nanograms] or ketamine [10-20 mg] given for breaktrough pain .In cases of severe gagging problems patients received additional small propofol boluses [10-20 mg], especially during moulding.

Patients were kept in the semireclined position [25-30°] or supine during the entire operation.

Supplemental oxygen at a rate of 1-2 lt min was given through the nasal prongs as long as needed to avoid hypoxemia .

Ketorolac 30 mg was given for articular/muscular /bone complaints either during or at the end of the operation ;at the

Table 1: Data of the patients and study.

	frequency or interval	%	mean	Std dev +/-
Sex: f m	 522 322	 61,8 38,1		
ASA class	1:422 2:308 3:106 4:8	50 36,5 12,6 0,9		
Age,years	8-96		59,2	14,6
Weight,kg	27-145		70,7	15,4
Height,cm	125-193		169	55
Time to procedure[min]	0-150		24	16,3
Procedure duration[min]	5-350		118	55
Midaz mg/kg total	0-0,18		0.048	0,029
Fentanyl microgr/kg total	0-3,64		0,79	0,60
SaO2<90%	149 pts	17,7		
Duration of desaturation min	0-85		2.50	8,8
Episodes of desaturation number	Min 0,max 15		0,34	1
Hypertension [yes/no]	151	17,9		
Hypotension[yes/no]	31	3,7		
Bradycardia[yes/no]	34	4,0		
Tachycardia[yes/no]	5	0,6		
Hypercapnia[yes/no]	324	38,4		
Hypercapnia duration min	0[357 pts]-335[1 pat]		29	33,2
Sleep[yes/no]	179pts,,0-95 min			
Sleep duration [min]	5-95 min		4.3	11
Premedication: drug name				
None	121	14,3		
Midazolam	152	18		
Midazolam+ haloperidol	45	5,3		
Midazolam +ibuprofen	86	10,2		
Diazepam	298	35,3		
Ibuprofen alone	3	0,4		
Diazepam +haloperidol	2	0,2		
Triazolam+ ibuprofen	18	0,1		
Triazolam alone	44	14,3		
Diazepam+ibuprofen	1	18		
Droperidol or haloperidol alone	1	0,1		
triazolam+codeine+paracetamol	27	3,2		
diazepam+codeine+paracetamol	19	2,3		
Midazolam+codeine+paracetamol	3	0,3		
Diazepam+haloperidol	16	1,9		
diazepam+ibuprofen	4	0,5		
etCO2 was not measured in 164 cases.				

completion of the procedure ibuprofen 400-600 mg p.os and/or dexamethasone [4 mg] i.v. were given at the discretion of the attending dentist.

In case of prolonged sleepiness [i.e. Ramsay 4] naloxone and/or flumazenil were given at the discretion of the anesthesiologist. All patients were discharged home within 2 hours from the end of the intervention accompanied by a responsible adult. Adverse events were defined as fluctuations of blood pressure , heart rate, etCO2 as defined ;other symptoms or discomforts were also noted. .

STATISTICS

Data are presented as mean +/- d.s.;univariate or multivariate analysis of variance was applied to search for correlation between adverse events and the data collected using version 24 of SPSS. Values of P>0.05 were accepted as significant. Anova was also applied in a comparison betweeen blood pressures [systolic and diastolic] of patients receiving a premedication or none (Table 2).

Table (3) presents the [minor] adverse effects that occurred; number and % quoted as well as duration of desaturation and

Table 2: Results of Anova for type of preanesthesia and basal blood pressures, before sedation Systolic Blood pressure.

Type of Premedication	Mean	S. D
1=midazolam	133	20
2=midazolam+neuroleptic	129	18
3=midazolam+FANS	131	17
4=diazepam	135	21
7=triazolam+FAns	127	20
8=triazolam	142	23
12=triazolam+paracetamol+codeine	142	23
14=diazepam+paracetamol+codeine	133	15
42=diazepam+neuroleptic	134	20
0=none	147	22

*groups<10 cases were omitted, F=6.408, p<0.001
Diastolic Blood pressure

	Mean	SD
1=midazolam	82	13
2=midazolam+neuroleptic	78	16
3=midazolam+FANS	78	14
4=diazepam	80	13
7=triazolam+FAns	75	15
8=triazolam	85	14
12=triazolam+paracetamol+codeine	80	12
14=diazepam+paracetamol+codeine	80	14
42=diazepam+neuroleptic	80	10
0=none	87	14

*Groups < 10 cases were omitted
F=4.64,p<0.001

hypercapnia.

RESULTS

Age and asa class were higly correlated.

The incidence of hypertension was associated with ASA class [signif 0.000], dosage of fentanyl [0.000], dosage of midazolam [0.001].

The incidence of hypotension was associated with ASA class [signif 0.012], age [0.000], with the dosage of fentanyl [but... P 0.052].

The incidence of bradycardia was associated with age [0.028]

The incidence of tachycardia was associated with age [0.000],ASA class [0.000], dosage of midazolam [0.013].

The incidence od desaturation was associated with the dosage of midazolam [but 0.049...].

The duration of desaturation was associated with the dosage of midazolam [0.001] and fentanyl [0.003].

The number of episodes of desaturation was associated with dosage of midazolam [0.005] and fentanyl [0.038].

The incidence f hypercapnia was associated with the dosage of fentanyl .

The duration of hypercapnia was associated with age [0.005] and dosage of fentanyl [=.000]. The % increase in the $etCO_2$ was associated with dosage of fentanyl [0.030]

Desaturation and hypotension were significantly linked [0.000]

Incidence of sleep was associated with hypotension [but

Table 3: Frequency and duration of the adverse effects.

Adverse effect	number	%	Mean	S.D.	95% +/-
hypertension	No:693	82.11			
	Yes:151	18.88			
hypotension	NO:813	96.33			
	Yes:31	3.67			
bradycardia	No:810	95.97			
	Yes:34	4.03			
tachycardia	No:839	99.41			
	Yes:5	0.59			
desaturation	No:695	82.35			
	Yes::149	17.65			
hypercapnia	No:362	42.89			
	Yes:324	38.33			
	n/a:158	18.72			
sleep	No:695	78.79			
	yes:179	21.21			
desaturation duration[min]			2.5	8.34	0.56
desaturation episodes [num]			0.33-1		0.07
sleep duration[min]			4.2	10.9	0.73
hypercapnia duration[min]			38.25	44.7	4.92
			median:20		

0.05…], desaturation[0.000].

Incidence of sleep duration was associated with dosage of fentanyl [0.024], desaturation [0.000], hypotension [0.001

Blood pressures of patients who received a premedication differed from those not receiving any form of premed.[P<0.001].

DISCUSSION

All Aes resolved rapidly with the appropriate measures,i.e. tapping or calling loudly the patients in cases of sleep, administering clonidine for hypertension or atropine for bradycardia etc.;in no case Aes proceeded from minor to intermediate;in no case there was the need for airway interventions except the administratioo of supplementary oxygen in cases of desaturation.Often borderline saturation [90%] was allowed to continue as long as $etCO_2$ was satisfactory or slightly elevated [41-42 mmHg] and the patient remained conscious and cooperative .

Elevated blood pressure and $etCO_2$ represented the most frequent AEs .

It is no wonder that hypoventilation was linked to the combined administration of the hypnotic[midazolam] and the analgesic opioid fentanyl; this is the price to pay in order to obtain a calm and cooperative patient allowing the surgeon to proceed with the intended operation without haste and stress .A moderate hypoventilation revealed by the increase in $etCO_2$ and its per cent increase above the basal values is the norm following these potent drugs: the potentiating effect of their combination [12] has been widely recognized.

Only the judicious titration of the respective dosages might forestall the complications arising in case of severe depression of ventilation with the need of rescue manoeuvres. As shown in the results dosage of both drugs was kept at a minimum compatible with the success of sedation and surgery, very rarely exceeding a total of 5 mg of midazolam and 100 micrograms of fentanyl for surgeries lasting more than 2 hours. Many studies show a clear link between use of sedation and risk of hypoxemia [13-14]. Few dental references were found in which the oxygen saturation levels recorded during sedations were used as a determinant of safety [15]. These authors noted that the variables that were significantly associated with low saturations were age, gender, and weight. either the dose of midazolam nor the additional use of propofol was a significant risk factor. These findings are quite different from ours where the addition of the opiate fentanyl is responsible for desaturation. Their impressive statistics of 3500 cases done by a single operator/sedationist supported by trained nurse demonstrated safe levels of oxygenation with sedative hypnotics like midazolam and/ or propofol;the problem changes with the addition of fentanyl,where our opinion is in favour of the presence of the anesthesiologist. Perrott and colleagues [16] reported on 34,191 patients who underwent oral surgical procedures using various anesthetic techniques. In this study, 5299 patients were under conscious sedation. The complication rate was 1.3%, although no details of the nature of the complications were given, other than that they were 'minor and self-limiting.' The authors concluded that conscious sedation was safe and associated with a high level of patient satisfaction. Milgrom and colleagues [17] reported on 207 sedations that tested the hypothesis that combined drug therapy [midazolam and fentanyl, or a double-blind placebo] results in significantly poorer safety but no difference

in efficacy, compared with the single drug approach.; in fact the addition of the narcotic resulted in apnea in 63% of cases versus 3% in the midazolam-only group. Interestingly, patients in the combination drug group were 4 times more likely to report an ''excellent sedation'' versus ''good, fair, or poor'' in the single drug group.

Jastak and Peskin [18] reported on 13 deaths under dental sedation between 1974 and1989 in the USA. They examined the physical status of the patient, anesthetic technique used probable cause of the morbid event; avoidability of occurrence, and contributing factors. They found that most patients were classified as ASA II or III with significant pre-existing conditions [obesity, cardiac disease, obstructive pulmonary disease]. Hypoxemia was the most common cause of untoward events. Most events were determined to be avoidable. The authors felt that sedation risks increased significantly in patients with a score of greater than ASA I and with extremes of age.

Midazolam, fentanyl, [and propofol] all depress respiratory drive and increase risk of apnea and hypoxemia; therefore a reasonable measure of safety is to examine the oxygen saturation levels recorded during sedations because it is hypoxemia that poses the greatest risk of morbidity or mortality. However our cases are the proof that with great care even some high risk patients could be sedated with no untoward sequelae.

Hypertension must be regarded probably more as a side effects of dental surgery than an AE;in fact ,beside the expected coincidence of high blood pressure and advanced age,the administration of the local anesthetic containing epinephrine could contribute to the rise of the blood pressure, albeit it has been shown its relative safety even in hypertensive patients [19-21].

Our analysis showed occasional peaks of blood pressure from the baseline following the top ups of the LA during the course of the procedure,but the nature of our data collection and flowchart precluded further analysis.In every case clonidine was administered when diastolic pressure exceeded 95 mmHG, 5 mmHg above the consensus limit of high blood pressure definition [22-23] : all cases were treated succesfully.Very often hypertension present at the induction of CS abated as soon as the patient reached a Ramsay score of 3, so that its aetiology could be attributed to the preexisting fear or anxiety rather than hypertension per se. Continuing in the same line of reasoning the association noted between hypertension and the additional doses of midazolam and fentanyl points out the need for more sedation and analgesia, so that it could be considered a case of a cause and effect consequence . Beside it is our opinion that pain occurring during the operation should be treated with top ups of local anesthetics ,being aware not to cross the boundaries of systemic local anesthetics toxicity , since more fentanyl means to increase the danger of hypoventilation and desaturation .The choice of clonidine offers a specific advantage:beside being a good hypotensive agent,it is acting synergistically with sedatives and fentanyl [24].

Bradycardia is a more serious concern in our practice,where the danger of vagal stimulation is always operating and constitutes a serious problem in young vagotonic athletes:however all patients responded promptly to the injection of atropine 0.5 mg : only 10 cases required supplementary doses. Its association with advancing age is surprising: the reason could be the fact that many hypertensive patients assumed their betablocker / calcium channel blocker shortly before surgery,hence arriving in the office at the peak of effect of the drugs. Again, these relationships were not investigated further because the nature of our data sheet.

Tachycardia increases the oxygen consumption and may provoke coronary constriction, angina and even heart failure.It was generally noted at the beginning of the procedure and hence considered more a sign of patient stress; it generally resolved after a few minutes in the vast majority of cases, without beta or calcium blockers.Its association with midazolam may represent the dominant sympathetic effect exhibited by the drug during CS as shown by Heuss et al.[25].

Desaturation, its duration and number of episodes were associated with the administration of midazolam and fentanyl; this is a well known consequence of their pharmacological actions and no other measures were taken except for the administration of supplementary oxigen: high risk patients [frail and elderly,overweight, ASA 3 and 4 patients] received oxygen at the induction prophylactically.

End tidal CO_2 monitoring has been included between the standard of care by the ASA since 2010 for general anesthesia, moderate sedation, and deep sedation [26].

Several studies demonstrate that respiratory depression is detected via end-tidal capnography30-60 seconds prior to detection via oxygen saturation [SaO_2][27].

Under clinical steady state conditions there is a good correlation between microstream capnometry and arterial carbon dioxide partial pressure; the relationship holds true even with patients intubated and mechanically ventilated [28].Values and tracings may differ between different types of cannula and various fresh oxygen flows [29] and cannot accurately follow arterial CO_2 in sleep hypoventilation [30] and even more so in COPD patients or whenever the tracing is erratic by mouth breathing,or other surgical disturbances .Notwithstanding these limitations ,it is however a vey useful adjunct to the monitoring of the vital signs of our patients.Moreover as arterial CO_2 tension rise ,as reflected in the etCO_2, a compensatory increase in minute ventilation occurs, accompanied by an increase in tidal volume [31] so that, in a certain sense,the increase in etCO_2 could be considered “useful” in our patients,provided that oxygenation and haemodynamics remain between physiological limits.

Desaturation,hypotension and sleep where significantly linked : the aetiology of these disturbances was attributed to the drugs used for sedation/analgesia.From this point of view it is of utmost importance to maintain the verbal contact with the patient,encouraging occasional deep breaths in order to correct hypoxemia and hypoventilation and at the same time improving the quality of the capnogram.

This study has several limitations: data are retrospective ,collected upon rigid templates ;therefore ,for instance , hypertension caused by the epinephrine contained in the LA cannot be differentiated by that arising from breaktrough pain or insufficient analgesia,either local or systemic. In some cases eleveations of blood pressure occurred during the last periods of surgery and we attributed its etiology to the fact that local anesthesia was wearing off.In these cases more systemic sedatives or analgesics could have postponed the discharge of the patient from the dental chair so that we chose not to administer more drug ;as a matter of fact we believe that the best option for breaktrough pain occurring near the end of surgery should again to inject more local..In these cases ketorolac was used, providing some comfort.

Premedication choice and dose was not standardized since very often it depended from the local drug availability and/ or dentist preference. We consider the administration of some tranquilizer before the induction of CS a very valuable way to attenuate fear and anxiety. This result has been shown in the difference between blood pressures measured at the induction of CS, significantly different betwen premedicated and non premedicated patients (Table 2). There is always a certain interval of time between the anesthesiologist arrival in the dentist office and the induction of CS [on the average around 20 min]; this time is spent by the anesthesiologist setting up all the necessary medications and monitors and checking all the equipment and could be used to sedate the patient and obtain valuable information about its drug tolerance.

We chose a very low level of SaO2 [90%] as a threshold limit for desaturation and classifying it as an AE; in many studies in the dental literature [and not limited to dentistry] the safety limit has been posted at 94% [32].

The steepness of the oxyhemoglobin curve indicates a dangerously low level of PaO2 at 90% saturation.However we were administering oxygen immediately as needed, monitoring etCO2, maintaining contact with the patient and were capable of any rescue manoeuver in case of need.We were dealing mainly with adult patients,where airway rescue manoeuvers are much more easily applied than in kids,where the desaturation threshold should be higher, at least 94%, since youngsters desaturate easily even following sedatives alone [33].

Finally, midazolam and fentanyl dosages were administered as needed at the discretion of the anesthesiologist following dosages based more on personal experience than as recommended according to age, weight, comorbidites [34].

In conclusion, careful titration of midazolam and fentanyl accompanied by non invasive full monitoring of patients vital signs by a dedicated anesthesiologist represent a safe practice for conscious sedation in the dentist office .

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