Anesthesiology 2001; 94:376

Ophthalmic Anesthesia

To the Editor:—I was happy to see the well-illustrated technique of ophthalmic anesthesia described by Ripart *et al.*[1] I also like the concept of using akinesia as an endpoint because it is easy to define and independent of the skills of the surgeon. Working with surgeons with surgical times ranging from 5 (clear cornea technique) to 100 min, I find that the faster surgeons do well with a patchy block.

I question the use of the word "efficient" in describing the medial canthus injection technique. Although their medial canthus technique is superior to their peribulbar technique, it is not superior to the peribulbar technique used by others.[2,3] Some of the discrepancy may be in the definition of a successful block because it is possible to have akinesia but not analgesia.

For the past 10 yr, we have used the following technique for more than 30,000 patients. Ten milliliters of a mixture containing half 0.75% bupivacaine and 4% lidocaine with 25 IU hyaluronidase is injected with a 16-mm, 25-gauge needle through the lid as deep as possible into the peribulbar space, half inferior lateral and half medial superior. There have been no perforations and approximately 15 cases of bradycardia, easily treated, 25–45 min after block, which may represent central effects. We have seen several cases of postoperative ptosis of the upper lid, which resolved within 1 month, without treatment. There were no other complications.

For another study, we prospectively evaluated akinesia 10 min after block, as previously described, for cataract surgery on 458 patients without glaucoma and achieved 94% akinesia. Akinesia was not graded. Any motion was considered lack of akinesia.

I suspect that the higher success rate is caused by the injection of more drug and volume. Using 2% lidocaine rather than 4% or giving only the inferior injection reduced the rate of akinesia. Based on personal communications with other ophthalmic anesthetists, I believe that my results are typical and that a 39% failure rate for the peribulbar technique is atypical. A technique necessitating that almost 40% of the patients be reblocked would be of limited use clinically. Although a meta-analysis has not been performed, from these communications, I would expect a peribulbar perforation rate of less than 1:20,000. More cases will be necessary to show that the medial canthus technique described also has a low perforation rate.

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On the Use of Topical Anesthesia for Cataract Extraction Surgery

To the Editor:—I read with interest the recent article by Ripart *et al.*[1] about ophthalmic regional anesthesia for cataract surgery. The authors stated, 'Cataract surgery requires a potent motor blockade (akinesia) of the eyeball and eyelids.' However, this statement is contradicted by the fact that excellent operating conditions can be obtained by using topical anesthesia with or without sedation.[2,3] Moreover, the topical technique avoids the rare but severe complications that may occur with injection anesthesia for ophthalmic surgery, such as perforation of the globe, retrobulbar hemorrhage, and dural or intravascular injection of local anesthetics.[4–5] The use of topical anesthesia for cataract surgery increased to more than 37% of cases in 1998.[6] At my facility, an increasing portion of the cases is treated with use of topical anesthesia, and the advantages in comparison with regional or injection anesthesia are dramatic.

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Sub-Tenon Eye Block: Approaching the Ideal?

To the Editor:—We read with interest the article by Ripart *et al.*[1] that provides clinical evidence to conclude that single-injection, high-volume medial canthus episcleral anesthesia is an efficient and safer alternative to peribulbar anesthesia. The salient features of their method are the injection of a relatively high volume (8–10 ml) of anesthetic solution and the use of adjuncts, such as clonidine premed-

ication and propofol sedation. The high volume of the injected fluid is in contrast with the low volumes (less than 4 ml) normally injected into the episcleral space. The high-volume injection provides good motor blockade, including akinesia of the lids, in addition to sensory block.

We would like to share our experiences at Moorfield Eye Hospital, London, during the period of training in the module of ophthalmic anesthesia. The method used for induction of block is basically the same as described by Stevens[2] and Guise.[3] A volume of 2.5–4 ml local anesthetic was injected. In patients in whom akinesia was deemed to be inadequate, a 1–1.5-ml top-up was administered at the original sub-Tenon site. Digital palpation was used to assess intraocular pressure, and digital compression was applied for a few minutes with a view to aid the diffusion of the injectate. We performed nearly 200 blocks, with only 10% of the patients being topped-up. The akinesia produced was regarded as adequate by the surgeons. No complications attributable to the block were noted.

The reports regarding the variation of intraocular pressure with the volume of the fluid injected into the episcleral space are scanty. Bowman *et al.*[4] reported an immediate mean pressure increase of 11.4 mmHg after injection of 10 ml fluid in the peribulbar space. Preliminary results of the study conducted by Ripart *et al.*[5] did not show a large increase above baseline after injection of a large volume of local anesthetic, probably because of the decrease produced by the previously mentioned adjuncts. Incidentally, fears that a large volume injection may cause a sustained increase in intraocular pressure, jeopardizing the retinal blood supply and impairing the surgical field, have not been confirmed.[6] Use of clonidine and propofol can be hazardous, especially for elderly patients. As per our experience, explanation of the procedure and assurance renders sedation unnecessary. The presence of a sympathetic nurse, a relative, or an interpreter helps the patient to relax and cooperate. We believe that more reliable data on the variation of intraocular pressure with the volume of injectate needs to be generated. This will help in fine-tuning the technique based on high-volume injection by way of determining the optimal volume and the ways of minimizing its adverse effects.

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In Reply:—We thank our colleagues for their interest in our work and pertinent comments. The exigency of akinesia and surgical times vary dramatically among teams, but analgesia is much easier to obtain than akinesia. A nonakinetic technique, such as topical anesthesia, may be sufficient in some cases of cataract surgery. Total akinesia is required in other cases. Our study was not aimed to determine whether akinesia is required for cataract surgery. We tried to determine which technique provides the better akinesia.

The rate for reinjections varies from less than 1% to 50%.[1,2] Unless the criteria for those reinjections are controlled using a standardized measure of akinesia, the rate of reinjection is not a good index of block quality to compare different studies. We aimed to obtain total akinesia. This might be considered an increase in the sensitivity of our study: An unperfect block was reinjected, although, in some cases, the surgeon might have dealt with it.

The technique used by Dr. Pinsker is a standard.[1,2] However, it is clear that a single injection may be sufficient in many cases.[1–3] The use of a systematic second injection can be considered a 100% reinjection rate, whether it is useful or not. It theoretically increases the hazards of the puncture twofold. A second injection should be performed only when required.

Lidocaine, 4%, and 0.75% bupivacaine are not available in France because of potential toxicity. The concentrations we used are classic.[1–3] They may contribute to a relatively low success rate. However, with those concentrations, the fact remains that single-injection medial canthus episcleral anesthesia is more efficient than peribulbar anesthesia (100% *vs.* 61% total akinesia).

Safety of medial canthus episcleral (sub-Tenon) block has to be confirmed. The rate of complications after eye block is more dependent on experience than technique. Regarding the low incidence of complication, a comparison among different techniques is difficult; a randomized study would require thousands of patients. However, since our technique has been described, other teams have used it, and it seems to be relatively safe.[5,6] The perforation rate is estimated between 3/4,000 and 1/16,000.[1,4] At our institution, we did not encounter globe perforation in any of more than 4,000 cases, nor did we observe any symptoms attributable to an impairment in retinal blood supply because of intraocular pressure increase caused by the high volume injected. If such complications occur, theirs frequencies are probably very low.

The use of sedation, although questionable, has been documented in many articles. It should not modify the akinesia scores. We use a very light sedation to help the patient to stay calm, awake, and cooperative when block is performed.

The technique used at Moorfield Eye Hospital necessitates a surgical approach to the episcleral space. Such a surgical approach avoids the risk of blindly inserting a needle. It would be interesting to compare both techniques. We are convinced that the future of eye block is episcleral (sub-Tenon) anesthesia, whichever approach is used.

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Anesthesiology 2001; 94:378

American Society of Anesthesiologists Physical Status Classification System Is Not a Risk Classification System

To the Editor:—In the article about variability in surgical procedure times published in the May issue of ANESTHESIOLOGY,[1] I was dismayed to find numerous (15) references to the American Society of Anesthesiologists (ASA) Physical Status Classification system as "ASA risk class," "ASA values," and "ASA risk scores." None of these characterizations of the Physical Status Classification system is appropriate. Drs. Meyer Saklad,[2] Ivan Taylor, and E. A. Rovenstein originally designed the system as a categorization system for statistical studies. They recognized from the beginning that "operative risk" was not an item to be included in the classification system because it is altered by the nature of the surgical procedure. The American Society of Anesthesiologists House of Delegates has modified the system several times in the past 59 yr, but risk was not included in any modification.

Many studies have shown a correlation between mortality and physical status class, but these are only in a surgery-specific analysis. That does not make the system a risk stratification system. The ASA Physical Status system was, and is, a means to stratify a patient's systemic illness. Certainly, a patient's inherent protoplasm is a part of systemic illness and indirectly may lead to adverse occurrences. The kind of operative procedure is not a part of the classification system because a physical status III patient is still in that status if scheduled for an excision of a skin lesion with monitored anesthesia care or if scheduled for a pancreatectomy with general anesthesia. The operative risk is different because of the surgery, but the physical condition of the patient is the same preoperatively.

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In Reply:—We regret our use of terminology with respect to the term "ASA risk class" in our recent manuscript. We will be careful to refer to the classification by its correct name, "ASA Physical Status Classification," in the future.

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Negative-pressure Pulmonary Edema in a Child with Hiccups during Induction

To the Editor:—I was interested to read the case report by Stuth *et al.*[1] about negative-pressure pulmonary edema. The pathogenesis of negative-pressure pulmonary edema is multifactorial and associated commonly with sustained negative intrapleural pressure.[2] It develops rapidly after the event. Although hiccups produce sharp and intermittent negative intrapleural pressure that is nonsustained, they produced pulmonary edema in this case. The authors have discussed the possibility of other causes of noncardiogenic pulmonary edema (NCPE). However, the possibility of cardiogenic pulmonary edema (CPE) was not discussed. Could this be a case of pulmonary edema resulting from an acute cardiac event?

This patient developed severe hypertension followed by a nonpalpable pulse and possibly hypotension. This hypertension could have been the result of a response to laryngoscopy and endotracheal intubation. High blood pressure could have contributed to the development of acute left ventricular failure and pulmonary edema. After left ventricular failure, the patient's blood pressure dropped, and, thus, the pulse could not be palpated.

There are few other findings in this case report that indicate that this could be a case of CPE. Using chest radiography, the authors found a central pattern distribution of pulmonary edema. Centrally distributed pulmonary edema could be suggestive of cardiogenic origin. A radiograph may show a normal-sized heart in the presence of acute left ventricular failure. It has been stated that radiograph film proves to be of little help, partly because of the portable nature of the film and suboptimal interpretative quality.[3] Thus, the absence of cardiomegaly by no means excludes cardiogenic pulmonary edema.[3]

An echocardiogram shows normal left ventricular systolic and diastolic function in NCPE.[3] The intraoperative echocardiogram of this patient showed good biventricular function but probably abnormal ventricular function. The resolution of all of the intraoperative echocardiogram abnormalities during follow-up also may imply an intraoperative cardiac event.

The most specific method for differentiating NCPE from CPE is the demonstration of increased alveolar–capillary permeability, which is characteristic of NCPE.[3] Alternatively, pulmonary capillary wedge pressure measurement could be useful to differentiate CPE from NCPE. These investigations may not always be feasible, and they may not be necessary when the diagnosis is obvious.

I believe that this patient developed acute pulmonary edema secondary to acute left ventricular failure. The physiologic mechanism ofhiccups is the stimulation of the epipharynx.[4] This patient was drooling. It could be that the saliva stimulating the epipharynx of the partially anesthetized patient produced hiccups. Could this be prevented by an anticholinergic premedication?

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In Reply:—We agree with Dr. Mandal that the pathogenesis of negative-pressure pulmonary edema is likely multifactorial and that the most obvious cases are associated with sustained negative pulmonary pressure, *e.g.*, during laryngospasm. However, there is increasing evidence that the initiating cause of negative-pressure pulmonary edema can be a relatively subtle airway obstruction that might be unnoticed until unexplained pulmonary edema develops.[1]

We also agree that an acute impairment of left ventricular function was likely present, and this probably contributed to the development of edema. This impairment is considered part of the pathogenesis of negative-pressure pulmonary edema and is secondary to a significantly increased left ventricular afterload caused by increased negative intrathoracic pressure with a closed airway, *i.e.*, in our case, during hiccups. The intraoperative echocardiogram that was obtained to rule out ongoing cardiac dysfunction only showed residual mild abnormalities consistent with right ventricular afterload increase. The full report that was provided in the original manuscript but shortened for editorial reasons was: "Mild mitral valve prolapse with mild mitral regurgitation. Normal aorta. Good left ventricular function with shortening fraction of 33%. Trivial pulmonary insufficiency with gradient of 13 mmHg predicting mild pulmonary hypertension. Good right ventricular function with no tricuspid regurgitation." Even after review, the cardiologist believed that this was inconsistent with a primary cardiogenic cause for the edema.

Dr. Mandal suggests that laryngoscopy and endotracheal intubation might have caused sufficient arterial hypertension to cause cardiogenic edema. This would imply either a very light level of anesthesia during intubation or the initiation of a neurogenic edema. We believe neither was the case. The halothane induction lasted more than 10 min, and the end-tidal halothane concentration (1.2–1.3%) was sufficient to cause no response to several cannulation attempts just 60 s before paralysis and intubation, which makes inadequate anesthesia unlikely. Secondly, neurogenic edema, as discussed in the case report, is not a transient, short-lasting phenomenon.

Lastly, Dr. Mandal mentions that hiccups could have been elicited *via* stimulation of the epipharynx by the patient's copious salivation. This reference and the related discussion were also removed in the review process of the original manuscript. We, too, believe that saliva might have had a hiccup-triggering effect in the partially anesthetized patient. Indeed, when this patient presented months later for her canceled dental procedure, she again was administered oral midazolam for premedication, and it was planned to insert an intravenous cannula during nitrous oxide sedation. Within 30 s of nitrous oxide administration, vigorous hiccups developed. The patient rapidly was induced intravenously, and the rest of the anesthetic procedure was uneventful.

Finally, we agree that an anticholinergic premedication administered early enough and in sufficient amount might help to prevent this triggering by decreasing oral secretions.

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Dantrolene: Opening the Anesthesiologist-proof Packaging

To the Editor:—Dantrolene, an emergency resuscitation drug, should be sold in packaging that facilitates drug access. It is not. Each dantrolene vial is topped with a butyl rubber stopper encased in a crimped aluminum cap (fig. 1). Before reconstituting the drug, one must remove the sharp-edged cap center. This can be hazardous; the first author experienced a finger laceration while attempting bare-handed cap center removal during a malignant hyperthermia resuscitation.

To identify efficient, safe ways to remove the cap center, we obtained expired dantrolene vials (Dantrium® Intravenous; Procter & Gamble Pharmaceuticals, Cincinnati, OH) from City Avenue Hospital, Philadelphia, Pennsylvania. Cap centers were removed relatively easily using the point of a ballpoint pen, a hemostat, or a screwdriver. Tools too thick to insert under the edge of the cap center (*e.g.*, car keys) were effective but mangled the outer cap ring in the process. Of course, reconstitution would be faster and easier if Procter & Gamble packaged dantrolene (average wholesale price, $65.87) with a convenient flip-top lid, such as that atop 5-ml vials of 0.9% sodium chloride (Abbott Laboratories, North Chicago, IL; average wholesale price, $1.20).

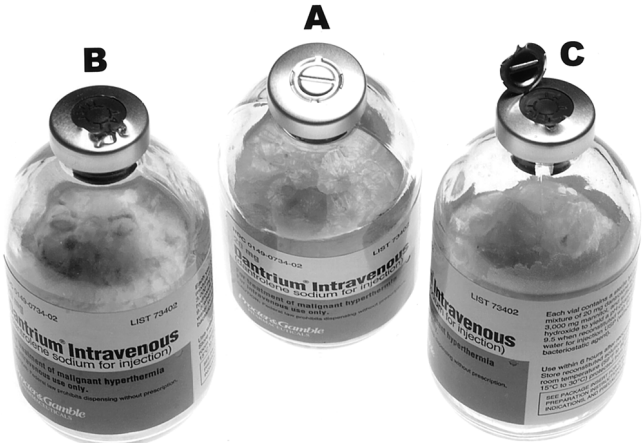

Fig. 1. Dantrolene vial cap: (*A*) intact, or after cap center removal with (*B*) a car key or (*C*) the point of a ballpoint pen.

Until Procter & Gamble improves dantrolene packaging, an anesthesia department might keep a safe, efficient cap center removal tool with the departmental dantrolene. Alternatively, the pharmacy department could remove the cap centers prophylactically and cover the stopper with sterile tape. Dantrolene dissolution is notoriously difficult; the packaging should not make the job more dangerous.

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In Reply:—In response to Drs. Leighton and Mitchell's letter to the editor about their difficulty in opening our Dantrium® Intravenous (dantrolene sodium for injection) cap center, we recognize the importance of appropriate packaging for all of our drugs, and, coincidentally, we are updating the Dantrium® Intravenous closure system, incorporating more user-friendly materials that are now available. The current Dantrium® Intravenous package has been used for 20 yr, and, during this time, we have had only a few complaints about the removal of the center of the crimped aluminum cap. Because of the global nature of this product and the requirement of compliance with many regulatory authorities, implementing this change is a lengthy process.

For the record, we do not agree with prophylactically removing the cap centers and covering the stopper with sterile tape, as suggested in the letter to the editor. This may compromise the sterility of the product. An ampule, vial, or prefilled syringe should be opened at the time of use. This recommendation is supported by the American Society of Anesthesiologists guideline entitled Recommendations for Infection Control for the Practice of Anesthesiology (2nd edition). Healthcare professionals may consult the American Society of Anesthesiologists Web site for additional information.

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Only Explicit Memory Seems Affected by Depth of Hypnotic State

To the Editor:—Recently, two studies[1,2] reported probably the first evidence of a relation between memory for words presented during general anesthesia and depth of hypnotic state, as measured by the electroencephalographic Bispectral Index (BIS).[3] The studies seem to diverge with respect to whether explicit (also direct, voluntary, or conscious) or implicit (also indirect, automatic, or nonconscious) memory for intraoperative events decreases with increasing depth. It is argued herein that, when the results of the first study are reanalyzed along similar lines as in the second study, both studies support the conclusion that explicit memory is primarily affected by depth of hypnotic state.

In both studies, the measurement of BIS was combined with a new procedure for separating explicit and implicit contributions to memory performance. The Process Dissociation Procedure[4] uses two opposing memory test conditions. When the test regards word completion, a participant is instructed (*i.e.*, the inclusion instruction) either to complete a word stem (*e.g.*, BA . . .) as much as possible to a previously presented word (*e.g.*, BASIS), or to avoid such old words (*i.e.*, the exclusion instruction) and replace them as much as possible with new words (*e.g.*, BAKER). When no old word can be given as completion, the participant is instructed simply to complete the stem with the first word that comes to mind (*e.g.*, FO . . . : FOCUS). Even in the exclusion condition, however, some old words (*i.e.*, BASIS) may be completed despite the instruction not to do so, which represents a form of memory that is apparently not under conscious control (*i.e.*, implicit memory). Inclusion performance roughly equals the sum of explicit and implicit memory performance. The performance difference between the two opposing test conditions thus reflects control over memory performance (*i.e.*, explicit memory).

The first study[1] examined a large sample of trauma patients with highly variable depths of hypnotic state. An individual BIS value could be calculated for each word per participant because every word was repeated 40 times in a 3-min interval. The subsequent study[2] was performed on patients undergoing emergency cesarean section who received only relatively light levels of anesthesia. This study[2] found some explicit, but no implicit, memory performance postoperatively at these light levels of anesthesia (average BIS, 76.3). Lubke *et al.*,[1] in contrast, concluded in the trauma study that there was no evidence for

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Table 1. Number of Target Hits (%) Observed in the Stem Completion Task with Exclusion Instructions, Number (%) of Target Words Presented during Surgery, and Probability of Explicit and Implicit Memory Performance from the Trauma Study,[1] Calculated According to Jacoby,[4] All at Categorized BIS Levels

BIS Level	20–30	30.1–40	40.1–50	50.1–60	60.1–70	70.1–80	80.1–97
Targets	17 (2.2)	105 (13.8)	221 (29.1)	172 (22.7)	124 (16.3)	92 (12.1)	31 (4.1)
Hits	8 (47.1)	41 (39.0)	76 (34.4)	68 (39.5)	52 (41.9)	27 (29.3)	14 (45.2)
Explicit	−0.082	−0.027	0.062	0.050	0.060	0.207	0.227
Implicit	0.471	0.390	0.367	0.416	0.446	0.364	0.585

Base rate completion performance was 0.32 in the inclusion condition and 0.33 in the exclusion condition. Without previous presentation, base level explicit and implicit performances should be 0.0 and 0.33, respectively.

BIS = Bispectral Index.

explicit memory over the whole BIS range (from 20 to 97) and that, consequently, the small decrease in "general memory performance" (*i.e.*, inclusion performance) at low BIS ranges should be attributed to a dependence of implicit memory on depth of hypnotic state. It should be noted, however, that the latter conclusion was not based on a direct test of implicit memory performance at different levels of anesthesia but was inferred indirectly from the aggregate measure of implicit and explicit memory (*i.e.*, inclusion performance). If this combined memory measure decreased with depth of hypnotic state and there was no overall (*i.e.*, over all BIS values) explicit memory performance, then only implicit memory can be responsible for this decrease. The absence of significance, however, cannot be seen as evidence in favor of the null hypothesis. Relatively few words in the trauma study were associated with high BIS values. The overall difference between inclusion and exclusion performance only narrowly missed conventional significance levels. Only a small increase in average BIS value in the trauma study (average BIS, 54 ± 14) in the direction of the BIS levels of the cesarean section study (average BIS, 76.3 ± 3) probably would have sufficed also to yield significant overall explicit memory performance in the first study.

In the cesarean section study, direct tests on both explicit and implicit memory performance were performed, and a fully significant explicit memory effect was obtained because, in this study, most words were processed at a light level of anesthesia. Applying the direct comparison of inclusion and exclusion performance (in a binomial test) of the cesarean section study to the performances at categorized BIS levels in the trauma study, however, may solve the apparent discrepancy between the conclusions of the two studies. Inclusion performance at categorized BIS levels was reported by Lubke *et al.*[1] in their table 2. Exclusion performance at categorized BIS levels in the trauma study[1] was calculated for the purpose of this letter (table 1). Binomial tests revealed that, only at the highest BIS levels, inclusion and exclusion differed significantly (BIS 80.1–97: $z = 1.75$, $P < 0.05$; BIS 70.1–80: $z = 3.01$, $P < 0.002$), indicating some level of control over memory performance (*i.e.*, a form of explicit memory[4]). At the same BIS level as in the cesarean section study, explicit memory performance thus also has been obtained in the trauma study. The indirect reasoning in the trauma article that there was no explicit performance and that, consequently, any dependence of memory on BIS should be accounted for in terms of implicit memory therefore seems to be contradicted by the finding of significant explicit memory only at higher BIS levels.

The conclusion drawn from these studies has practical relevance if anesthesiologists want to set BIS during surgery at the highest level for which no memory of intraoperative events is probable. According to the reasoning of Lubke *et al.*,[1] even the highest BIS level in the study would suffice to prevent explicit memory. The present reinterpretation of the data suggests that the chance of finding explicit memory may be reduced sufficiently only below a BIS level of 70. The form of explicit memory obtained in the two studies[1,2] probably should not be equated with free recall of intraoperative events,[2] but the successful exclusion at testing implies that patients can make some type of conscious reference to the presentation of the words during anesthesia, when explicit memory is cued by word stems. Theoretically, implicit memory also may be harmful by affecting postoperative well-being and recovery without the patient being aware of its source. It is plausible that implicit memory may be disrupted at high levels of distraction[5] or at deep levels of hypnosis, but the present data do not provide a clear indication of the BIS level at which implicit memory disappears. Seeking the lowest BIS level also to avoid implicit memories may bring hazards to the patient. Although it is still unclear whether the Process Dissociation Procedure[4] is able to provide pure and independent estimates of explicit and implicit contributions to memory, the two studies[1,2] at least seem to support a dissociation between the Process Dissociation Procedure estimates of explicit and implicit memory.

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