To the Editor—The interesting article by Flaishon et al.1 included prospective data on the difficulties of performing anesthetic and critical care duties in mass casualty situations. An additional factor to consider is the challenge of continuing to perform one’s duties while wearing full antichemical protective gear. As a member of the Air National Guard, I am obligated to perform chemical warfare training. Wearing the full MOPP 4 antichemical warfare ensemble is uncomfortable, especially in warm environments such as the Middle East. The clinician must contend with the intrinsic difficulties of wearing the suit and be mindful of preventing his own severe dehydration and the need to be resuscitated himself.

Most United States civilians do not appreciate the need to maintain adequate hydration. In the Middle East, constant hydration is a way of life. Military personnel have become familiar with disaster management concepts. In the future, such training will be mandatory for civilians as well. The Society of Critical Care Medicine (Des Plaines, IL) sponsored its first course on disaster medicine at its annual meeting this year to provide civilian clinicians with the basic concepts of disaster management. The course will heighten awareness of the challenges of airway management as well as the challenges and hazards of extended wear of chemical warfare gear.

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Reference


To the Editor—We thank Dr. Bloomfield for his comments. We share his concern regarding the troublesome antichemical gear and discussed it in our article.1 The psychological and performance effects of the protective gear have also been previously addressed.2 Stressful exercise causes fluid shifts, dehydration, and heat stroke, which must be prevented or treated if encountered.3,4 The antichemical suit further hampers temperature regulation and is recognized as a cause of dehydration.

In the Middle East, especially during the summer, dehydration and heat stroke are common, especially in the elderly and in civilians or army corps involved in outdoor strenuous exercise.1

We agree with Dr. Bloomfield that the first step in preventing dehydration and heat stroke is training and education.

It is important to note that according to chemical attack protocols, civilians are expected to wear only an antichemical facemask rather than the full protective gear. The problems mentioned are of major concern for those wearing the full protective suit, namely trained personnel, military and medical, who are required to treat and evacuate attack victims as well as perform other military operations while under such an attack.

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Cardiac Arrest, a Preventable yet a Possible Risk of Dexmedetomidine: Fact or Fiction?

To the Editor—We read with interest the well-written case report ‘Dexmedetomidine and Cardiac Arrest’.4 The inference of the authors seems to be simple, but a closer look reveals the omission of many complicating factors that would have contributed to the morbidity attributed to dexmedetomidine. Therefore, we dispute the conclusions. We believe that the patient received an excessive dose of dexmedetomidine after significant doses of other anesthetics and there was delay in treating the bradycardia. We believe that the progression to cardiac arrest could have been potentially prevented.

The interesting article by Flaishon et al.1 included prospective data on the difficulties of performing anesthetic and critical care duties in mass casualty situations. An additional factor to consider is the challenge of continuing to perform one’s duties while wearing full antichemical protective gear. As a member of the Air National Guard, I am obligated to perform chemical warfare training. Wearing the full MOPP 4 antichemical warfare ensemble is uncomfortable, especially in warm environments such as the Middle East. The clinician must contend with the intrinsic difficulties of wearing the suit and be mindful of preventing his own severe dehydration and the need to be resuscitated himself.

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Dexmedetomidine and Asystole

To the Editor—We read with great interest the report of a cardiac arrest during dexmedetomidine use in a patient with myasthenia gravis. In this case the interaction of pyridostigmine and epidural anesthesia was cited as possible contributors to this complication. We would like to add our own experience with dexmedetomidine. We began to use it in 20–40-yr-old healthy patients scheduled for laparoscopic gynecological procedures under sevoflurane and fentanyl anesthesia plus cisatracurium for neuromuscular blockade. Dexmedetomidine was infused by the ‘sufentanil’ program of an Amne® intravenous infusion pump (Abbott Laboratories, North Chicago, IL) with an initial infusion of 4 μg·kg⁻¹·h⁻¹ for 15 min followed by 0.3 μg·kg⁻¹·h⁻¹. After 40 patients were anesthetized using this technique, there was one case of severe bradycardia (32 beats/min) and one case of asystole. No patient received pyridostigmine or had epidural anesthesia instituted.

This event of asystole occurred while the patient was in Trendelenburg position with the peritoneal cavity insufflated with carbon dioxide with spinal anesthesia instituted.

One case of asystole. No patient received pyridostigmine or had epidural anesthesia plus cisatracurium for neuromuscular blockade. Dexmedetomidine was infused by the ‘sufentanil’ program of an Amne® intravenous infusion pump (Abbott Laboratories, North Chicago, IL) with an initial infusion of 4 μg·kg⁻¹·h⁻¹ for 15 min followed by 0.3 μg·kg⁻¹·h⁻¹. After 40 patients were anesthetized using this technique, there was one case of severe bradycardia (32 beats/min) and one case of asystole. No patient received pyridostigmine or had epidural anesthesia instituted.

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be the case, but the loading dose (1 μg/kg over 10 min) was exactly as recommended by the manufacturer in the package insert and the infusion rate (0.2 μg·kg⁻¹·h⁻¹) was at the low end of the recommended range. Whether or not our dosing was excessive, it was in line with the recommendations of the manufacturer.

We agree there were multiple factors involved; this was a main point of the article. Also, the assertion that the asystole could have been avoided if the bradycardia had been treated earlier is well taken. The bradycardia in question, however, was an easily explained (by the dexametomidine loading dose) decrease in heart rate to 46–50 beats/min with stable blood pressure. Treating this in a patient with good cardiovascular health would ordinarily seem unnecessary. We now believe that even mild, hemodynamically stable bradycardia in the presence of dexametomidine and other negative chronotropic influences should be treated. The view through our “retrospectoscope” confirms this.

We appreciate Drs. Videira and Ferreira sharing their experiences and agree that a large-scale safety study of the drug should be considered. We believe that when used appropriately, dexametomidine is very safe and useful. Perhaps by performing such a study and sharing our experiences with the drug, we, as a community, can avoid future closed claims analyses and unnecessary “black box” Food and Drug Administration warnings.

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TREATMENT OF REBOUND PULMONARY HYPERTENSION: WHY NOT SILDENAFIL?

To the Editor— I read with great interest the case report by Augoustides et al.1 published in the April issue of Anesthesiology. They highlight the important issue of rebound pulmonary hypertension after withdrawal of inhaled prostacyclin and make a case for the use of inhaled iloprost. They propose that inhaled iloprost may allow gradual controlled withdrawal of perioperative inhaled selective pulmonary vasodilation, probably as a result of its favorable pharmacokinetics. Hence, in their opinion it has great promise in the management of perioperative pulmonary hypertension after cardiac surgery. However, I think that if the authors had highlighted the advantages of using sildenafil, instead of iloprost, in this scenario their case report would have made a more lasting and useful contribution to the existing literature on the topic of management of rebound pulmonary hypertension.

Pulmonary hypertension remains a major complication after surgical correction of congenital and long-standing valvular heart disease. Inhaled nitric oxide has been shown to reduce, but not eliminate, potentially life-threatening episodic pulmonary hypertensive crises.2 Nitric oxide increases intracellular cyclic guanosine monophosphate, resulting in smooth muscle vasodilation. Phosphodiesterase type 5 is responsible for cyclic guanosine monophosphate breakdown in lung tissue. Abrupt discontinuation of nitric oxide may be complicated by life-threatening events, and phosphodiesterase activity may play a role in this phenomenon.3 Sildenafil (Viagra; Pfizer Laboratories, New York, NY), a selective and potent inhibitor of phosphodiesterase type 5, is equally effective and selective in reducing pulmonary vascular resistance, probably as a result of its favorable pharmacokinetics.4 Compared with the standard treatment, inhaled nitric oxide, sildenafil is superior in decreasing the mean pulmonary artery pressure and cardiac output 2 hours after withdrawal.5 It also causes a significant increase in the cardiac index.6 Its availability in oral, inhaled and intravenous forms, longer half-life of 4 h,7 and proven efficacy in randomized controlled trials7–9 are some of the distinguishing features which make sildenafil first-choice agent for managing rebound pulmonary hypertension.

Thus, my question for Augoustides et al. is “Why inhaled iloprost and not sildenafil?”

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In Reply.—I thank Dr. Raja for an excellent appraisal of the role of sildenafil (Viagra; Pfizer Laboratories, New York, NY) in the management of rebound pulmonary hypertension after withdrawal of inhaled prostacyclin, as highlighted in our recent case report. Dr. Raja has correctly highlighted that sildenafil is an alternative to iloprost in this setting.8,10 Our discussion of iloprost in the case report focused on its advantages over inhaled prostacyclin in the withdrawal of inhaled pulmonary vasodilator therapy. The pharmacokinetics of iloprost highlight a limitation of inhaled prostacyclin, namely its short half-life, that may facilitate serious rebound pulmonary hypertension.

However, this discussion was by no means intended to minimize the role of alternative approaches to the management of rebound pulmonary hypertension. As emphasized, a tiered multimodal therapeutic approach to pulmonary hypertension is essential for successful management.1,8,5 Indeed, this multimodal therapeutic approach to this clinical scenario not only includes sildenafil but also extends beyond this agent. The withdrawal of inhaled pulmonary vasodilators with a short half-life (nitric oxide, prostacyclin) should be approached in a tiered multimodal fashion. Although sildenafil is eminently suitable, it is but one of a possible menu of pharmacologic choices.

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In Reply.—We thank Dr. Huang for his interesting comments. We agree that frequent control of the correct needle position by the use of fluoroscopy is important during interventional pain procedures.1

The depth of the needle position on a lateral view is important because antioxidant agents must be injected as close as possible to the site of pathology; i.e., in the anterior plane of the epidural space.2 With respect to the presented anteroposterior view (Fig. 2a in our article)3 and in accordance with another review,4 the insertion of the needle did not further medial than the six o’clock position in the anteroposterior view reduces the risk of dural puncture.5 Moreover, when our needle had been positioned intradurally at this level, a discogram could not have been explained.

Finally, low back pain and sciatica resulting from migrated disc herniations are an indication for transforaminal epidural infiltrations.6 By placing the needle in a correct fluoroscopic position, the needle can accidentally encounter a rostrally displaced disc herniation, which possibly explains this unexpected event. Our finding emphasizes the use of fluoroscopy in interventional pain procedures.

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(Accepted for publication July 28, 2004.)
To the Editor:—I read the fascinating review by Dr. Ghoneim1 with great interest, especially the section on memory-enhancing or memory-imparing drugs. Anesthetics impair memory function in perisurgical periods, whereas cholinesterase inhibitors enhance memory3 and act at central muscarinic cholinergic receptors involved in the process of memory consolidation.4 Cholinesterase inhibitors donepezil, galanthamine, and rivastigmine, currently in clinical use,1,5 represent the first line of treatment in Alzheimer disease and the only drugs of proven benefit.5,6 Other cholinesterase inhibitors (i.e., physostigmine) are under clinical evaluation.5

Drugs affecting central cholinergic activity also influence the anesthetic effect. Increasing central cholinergic tone with physostigmine antagonizes the hypnotic effect of propofol, shown by the return of consciousness (defined as responsiveness to commands) or wakefulness (appearance of being awake with open eyes but without cognitive content).6 Plourde et al.7 measuring the action of physostigmine on the hypnotic effect of inhaled volatile anesthetics, conclude that physostigmine can, at least partially, antagonize the hypnotic effect of sevoflurane (subanesthetic concentrations) and that the resulting arousal is reflected by an increase in the amplitude of auditory steady-state response and, to a lesser extent, of the bispectral index. An interesting possibility is the antagonism of the anesthetic effect with physostigmine that results from potentiation of 40 Hz oscillations via increased muscarinic tone,8 whereas anesthetic-induced unconsciousness is associated with a reduction of gamma or 40 Hz oscillations in thalamocortical systems.7 These rhythms constitute background activity reflecting depolarization of thalamic and cortical neurons, a physiologic condition required for consciousness.9 In addition, Hill et al.10 demonstrated that physostigmine decreased the time for return of consciousness after halothane anesthesia.

These data, taken together, suggest not only that if reversal of the neuromuscular blockade occurs during anesthesia using cholinesterase inhibitors patients could be at risk of intraoperative awareness, as we recently underlined,11 but also that these drugs may promote an enhancement of implicit memory for any awareness event that occurs. It may occur above all during light levels of anesthesia, common during the final period of anesthesia. During this period, cholinesterase inhibitors are given by anesthesiologists to reverse neuromuscular block. In other words, patients may better recall memories of the awareness experienced intraoperatively.

It was also reported that inhibition of central nicotinic acetylcholine receptors contributes to secondary effects attributed to anesthesia such as impairment in memory and cognitive performance,12 whereas nicotinic acetylcholine receptors agonists improve memory.13 Other drugs used in anesthesia, as well as the neuromuscular blocking drugs atracurium and the atracurium and cisatracurium metabolite laudanosine, activate nicotinic acetylcholine receptors at concentrations comparable to those measured in the central nervous system during, and for several hours after, general anesthesia.14,15 Administration of these neuromuscular blocking drugs, resulting in laudanosine production, has been suggested to improve postoperative cognitive functions,16,17 with the clinical relevance that they could have a potentially therapeutic effect in patients with Parkinson’s disease.18 We ask if atracurium, cisatracurium, and their metabolite laudanosine should be included in the list of drugs acting at the cholinergic receptors and therefore potentially enhancing memory, with advantages and disadvantages mentioned above, and if these data merit, as do the anticholinergic agents, more detailed exploration by laboratory and clinical studies.

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To the Editor:—The authors must be congratulated for undertaking the study attempting to answer the question of protocol-based airway management in the event of unanticipated difficult intubation.1 However, this raises some serious questions about the content and conclusions of the study.

First, we are unclear as to how the investigators have concluded that their local protocol-based approach to airway management in the event of unanticipated difficult intubation after induction is efficacious. In an 18-month interval, 100 patients who were anticipated to be easy to intubate on preoperative work-up were subsequently found to be difficult to intubate. Sixteen percent of these patients suffered severe hypoxemia. Although the authors have not provided any data regarding the incidence of hypoxemia at induction among the true positive participants, it is unlikely that the incidence in that population could be as high as 16%. One patient suffered significant dental trauma and one ended up aspirating gastric contents. In addition, 89 patients were subjected to multiple attempts at direct laryngoscopy. The authors fail to acknowledge that these adverse events could very well have been the result of the sticking with the proposed airway algorithm. It appears that most of the patients suffered hypoxemia as a result of multiple attempts at laryngoscopy. Hypoxemia, as we understand, is a clear sign of ventilatory failure under these situations unless it is attributable to other causes. Failure to keep a substantial number of patients oxygenated highlights the inefficiency of the proposed algorithm. Unless the study was designed to evaluate the efficacy of the Intubating Laryngeal Mask Airway™ (LMA North America, Inc., San Diego, CA) as a tool for rescue ventilation, the conclusion that 100 percent of the patients were successfully ventilated underestimates the significant problems at ventilation encountered by the anesthesiologists while following the algorithm.

We are also unclear on what basis the authors claim that the study has validated the local protocol-based approach to airway management. The study has neither the design nor the power to answer this question, as we do not know what would happen if the anesthesiologist were not restricted by the protocol to the use of direct laryngoscopy, gum elastic bougie, Intubating Laryngeal Mask Airway™, or the transtracheal jet ventilation. Whether anticipated or unanticipated, the approach to airway management in the event of failed intubation at induction depends on multiple factors. The result of preliminary laryngoscopy, the view of the glottis, the primary reason for intubation failure (is it the poor laryngoscopic view or the failure to pass the tube?), ease of ventilation with the mask, the muscle relaxant used, emergency or elective surgery, state of oxygenation of the patient, presence or absence of risk factors for aspiration, the condition of upper dentition, and, above all, the skill and expertise of the anesthesiologist all must be taken into account before defining the next step. A protocol-based approach like the one proposed by the investigators may limit anesthesia providers from applying individual problem-based solutions in the event of inadvertent difficult intubation. The end result: the patient with the poor dentition suffers dental trauma, the patient with full stomach may wind up aspirating gastric contents; failure of the Intubating Laryngeal Mask Airway™ regardless of the cause (morbid obesity/limited mouth opening) commits the anesthesia provider to expose the patient to the risk of transtracheal jet ventilation although switching to simple a laryngeal mask airway or laryngeal tube might have solved that problem. A broad-based protocol that incorporates all the fundamental goals and objectives of airway management, e.g., the American Society of Anesthesiologists airway protocol, allowing for stepwise evaluation based interventions while taking into account factors specific to operator skill and experience, available resources, and patient continues to be the most prudent approach to management of inadvertent difficult intubation.²

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References


To the Editor:—We read with great interest the recent report on un- anticipated difficult airway in anesthetized patients by Combes et al.¹ The article confirms that by strictly adhering to a simple pre-defined algorithm most problems occurring during management of an unexpected airway can be solved. This has already been proven in two other large prospective studies.²³ Using the gum elastic bougie as the first choice in a “can ventilate” but “cannot intubate” situation is a well-established technique, especially in Great Britain, and, of course, is much cheaper than, for example, a fiberoptic bronchoscope.⁴

Unanticipated Difficult Airway: What about Emergency Cases?

Anesthesiology 2004; 101:1484–5

To the Editor:—We read with great interest the recent report on un- anticipated difficult airway in anesthetized patients by Combes et al.¹ The article confirms that by strictly adhering to a simple pre-defined algorithm most problems occurring during management of an unexpected airway can be solved. This has already been proven in two other large prospective studies.²³ Using the gum elastic bougie as the first choice in a “can ventilate” but “cannot intubate” situation is a well-established technique, especially in Great Britain, and, of course, is much cheaper than, for example, a fiberoptic bronchoscope.⁴

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To the Editor—We recently encountered a case that required extubation strategy for difficult airway as recommended by American Society of Anesthesiologists task force.1 A 50-yr-old lady underwent segmental mandibulectomy and radical neck dissection with deltopectoral flap for carcinoma parotid gland. At the end of surgery the oral endotracheal tube was left in place and she was shifted to the intensive care unit.

In the absence of either a jet stylet2 or commercially available tube changer3 that is "rigid to facilitate intubation and/or hollow to facilitate ventilation," we thought of using a readily available tube changer. We were wary of using the previously described tube changers because of their lack of lumen to provide oxygen,4 lack of stiffness,4–6 or small size.7

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Therefore, we decided to test these on mannequins (Ambu International, Copenhagen, Denmark). In the course of our experiments we realized that the sheath provided with Fogarty catheters (Willy Rusch AG, Kernen, Germany) is hollow and stiff and has a length of 80 cm and a varying external diameter (3–5 mm for sheaths of 6-French, 7-French, and 8-French Fogarty), making it a potentially good tube changer. We tested it on mannequins and found that it was successful in guiding the endotracheal tube every time and was adequate for both oxygen insufflation and jet ventilation.

We extubated the trachea of our patient over this tube changer (sheath of Fogarty 8-French), confirmed its correct position by end-tidal carbon dioxide monitoring before and after extubation, and kept it in place for over 2 h, giving oxygen at 4 l/min for the initial 30 min. For monitoring the end-tidal carbon dioxide we connected the sample line of the side-stream capnometer (Capnomac Ultima; Datex Ohmeda Division Instrument Corporation, Helsinki, Finland) to the machine end of the Fogarty sheath (it could be easily screwed into the machine end). On the other hand, for delivering oxygen, we could slide the oxygen delivery tubing from the wall-mounted flowmeter over the machine end of the Fogarty sheath. We also tried keeping both carbon dioxide sampling tube and oxygen delivery tube together after mounting a three-way connector to the machine end of our tube changer (carbon dioxide sampling tube on the side port and oxygen delivery tube on the end port). The problem with this arrangement was that the end-tidal carbon dioxide readings became too low at oxygen flow rates greater than 1.5 l/min. The patient tolerated our tube changer well until it was removed; our patient did not require reintubation.

We feel that in the absence of a commercially available tube changer, this tube changer fulfills all the criteria of rigidity, hollowness, and length required of such a device. Its two minor limitations are 1) its nonstreamlined tip, which is of little practical consequence if it is used correctly when it passes through an already placed endotracheal tube and remains in mid-trachea and 2) its material is probably not tested for tissue compatibility.

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Foreign Body in the Airway

To the Editor.—The NIM® electromyographic endotracheal tube (Medtronic Xomed, Jacksonville, FL) is a wire-reinforced endotracheal tube equipped with surface electrodes often used to monitor recurrent laryngeal nerve function during thyroid surgery. Recently, while preparing a new NIM® tube we noticed a kink midway along its shaft. The mechanism of this tube kink was not known but at least one other anesthesiologist in our department has experienced a similar problem with this tube. Closer examination of our tube revealed that the reinforcing metal coil had separated from the internal surface of the tube (fig. 1). To see how the coil had come to be dislodged we hooked part of the separated metal coil with a flexible stylette and intentionally pulled it out with minimum effort. It is possible to pull out the entire length of the reinforcing wire once a portion of it has been dislodged (fig. 2). The coil in these tubes is normally held in place by a thin plastic layer that keeps it adhered to the internal surface of the tube. This is in contrast with other reinforced tubes in which the reinforcing wire is embedded within the wall of the tube.

Such a foreign body within the lumen of an endotracheal tube can potentially cause airway management problems, including difficulty suctioning the trachea and advancing a fiberoptic instrument or airway obstruction. From our experience, it is possible for the reinforcing wire to become dislodged during normal use of this tube. For instance, a patient could bite on the tube separating the wire. Anesthesiologists should be aware of this possibility. Clinicians are cautioned not to excessively bend the NIM® tube and to carefully inspect its lumen before each use.

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(accepted for publication June 28, 2004.)
To the Editor:—Linezolid is a valuable drug that is finding increased use in the hospitalized surgical patient for the treatment of infections resulting from resistant, gram-positive organisms. Along with its known efficacy as an antibacterial agent, linezolid is a mild, reversible monoamine oxidase inhibitor. Reviews on the subject of its monoamine oxidase inhibitor-like profile have expressed caution about the use of linezolid in the clinical setting, specifically when combined with sympathomimetic agents, but there have been few, if any, reported clinical examples of a significant interaction. Recently, however, we observed unexpected intraoperative hemodynamic lability, as well as severe intermittent hypertension, in a psychiatric patient maintained on bupropion who was subsequently placed on linezolid for treatment of an infected vascular graft. We raise the concern that we have seen one of the first examples in the perioperative setting of a potentially dangerous interaction between linezolid and bupropion.

The patient was a 57-yr-old male status post axillary-femoral bypass graft who presented to the emergency room with evidence of a graft infection and was admitted for antibiotics. After a trial of several antibiotics, he was placed on linezolid for treatment of resistant, gram-positive organisms. As an outpatient, he had been stably maintained on bupropion for long-standing depression; this drug was continued throughout his hospital course. After about 24 h of linezolid therapy, the patient was taken to the operating room for graft removal, where he underwent a propofol/succinylcholine induction with standard doses and a maintenance anesthetic of 1.5% isoflurane and fentanyl 250 μg. His intraoperative course was notable for several episodes of severe hypertension (as high as 260/145 mmHg), despite an otherwise stable anesthetic. The unexpected hemodynamic lability was severe enough to result in an unplanned admission to the intensive care unit, where the patient had an unremarkable postoperative course.

The possibility of a significant drug interaction between linezolid and bupropion was suspected immediately and is supported by a careful analysis of the underlying pharmacologic mechanisms. Bupropion is an antidepressant that, in concert with its primary metabolite hydroxybupropion, acts as a norepinephrine reuptake inhibitor as well as a mild dopamine reuptake inhibitor. Both norepinephrine and dopamine are monoamine compounds metabolized by monoamine oxidase. The use of bupropion with older, more traditional monoamine oxidase inhibitor drugs (such as phenelzine and tranylcyproline) has long been contraindicated in standard psychiatric practice because of the risk of a hypertensive crisis. The older monoamine oxidase inhibitors do differ from linezolid in that they are strong, irreversible inhibitors of monoamine oxidase. As linezolid is a weak, reversible monoamine oxidase inhibitor, it had not been appreciated that coadministration with bupropion might cause a similar hypertensive state.

However, linezolid clearly resembles the stronger monoamine oxidase inhibitors in its capacity to interact adversely with certain drugs. Combining the older monoamine oxidase inhibitors with serotonergically active drugs, such as selective serotonin inhibitors, meperidine, and dextromethorphan, may lead to a severe central serotonin syndrome. Similarly, linezolid has been implicated in producing a central serotonin syndrome when combined with either paroxetine or citalopram (both selective serotonin reuptake inhibitors). Furthermore, it is known that sympathomimetic agents, when administered in combination with the traditional monoamine oxidase inhibitors, may produce severe hypertensive events. Again, linezolid mimics the interaction profile of the stronger monoamine oxidase inhibitors by producing statistically significant increases in blood pressure when
combined with pseudoephedrine and phenylpropanamine. Based on this information, it is not surprising that linezolid acts like a more traditional monoamine oxidase inhibitor when combined with buproprion, especially in the context of the well-known physiologic stimulation and adrenergic stress of surgery.

It is hoped that this letter will alert clinicians to the monoamine oxidase inhibitor-like profile of linezolid and prevent the combination of linezolid with agents that enhance the function of any of the monamines (serotonin, norepinephrine, epinephrine, and dopamine).

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