REVIEW ARTICLE

CURRENT CONCEPTS

Medical Evaluation of Patients Undergoing Electroconvulsive Therapy

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After a period of declining use, electroconvulsive therapy (ECT) is now used more widely as a treatment for major depression and other psychiatric disorders.[1] Many patients undergoing ECT are elderly and have multiple coexisting medical conditions. Consultants are often asked to provide a medical evaluation before ECT, although many may feel uncomfortable in this role. There is little summary guidance from the literature on the medical assessment of these patients. The technique and efficacy of ECT have been reviewed in the *Journal*.[1] In this article, we present an approach for medical consultants, with special attention to patients with coexisting medical conditions and to the management of complications that may occur after the procedure.

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BACKGROUND

Psychiatrists use ECT to treat a variety of psychiatric conditions (Table 1). Contrary to popular belief, ECT is safe. Procedure-related deaths are rare, and mortality rates have remained stable in recent decades. For example, Kramer[3] reported only two deaths per 100,000 treatments during the period from 1977 through 1983, and similar findings have been reported by Schiwach et al.[4]

ECT is performed in both inpatient and outpatient settings. Before an operator delivers an electric current through two electrodes placed in either a bilateral or unilateral temporal position, an anesthesiologist administers an intravenous anesthetic agent (e.g., propofol, etomidate, or methohexital) and a muscle relaxant (typically succinylcholine because of the rapid onset and short duration of its effects). Airway control is most commonly maintained with mask ventilation before the electrical stimulus is delivered. Anesthesiologists may also administer an anticholinergic agent such as glycopyrrolate or, less commonly, atropine to limit bradycardia and salivation. Patients undergo continuous electrocardiographic (ECG) and electroencephalographic monitoring, pulse oximetry, measurement of end-tidal carbon dioxide, and noninvasive blood-pressure monitoring during the procedure. The stimulus induces a seizure that typically lasts 30 seconds, followed by a postictal period that may include somnolence and confusion. A typical full course of ECT consists of 2 to 3 treatments per week, for a total of 6 to 12 treatments.[1]

PROCEDURE-RELATED CHANGES AND SUBSEQUENT MORBIDITY

ECT has a dramatic effect on blood pressure and heart rate. Between the stimulus and the onset of the seizure, bradycardia or frank asystole may last for more than 5 seconds.[5] After the seizure, tachycardia and hypertension occur. Most hemodynamic changes persist into the recovery period and resolve within 20 minutes. These changes result from increased vagal tone before the seizure and catecholamine surges during

Table 1. Principal Indications for ECT.*

Major depression (unipolar or bipolar) with a lack of response to medications, intolerance to medication due to side effects or coexisting conditions, a need for a rapid response because of other conditions, catatonia, psychosis, suicidality, or clinically significant dehydration or malnutrition
Mania
Schizophreniform disorder or schizoaffective disorder

* Indications are from the American Psychiatric Association.[2]

and after the seizure.[6] There are substantial variations in hemodynamic sequelae; Takada and colleagues reported a 25% increase in mean arterial pressure and a 52% increase in heart rate.[7] In addition, in one study involving 53 patients undergoing ECT, transient decreases in the ejection fraction were detected in approximately one third of patients after the first treatment, although these changes were not clinically apparent. The effect of ECT in patients with underlying heart disease is unknown.[8]

Early studies showed high rates of cardiovascular complications, though most of the complications were minor and transient.[9-11] According to recent reports, preexisting cardiac disease has been associated with increased complication rates, although most complications remain minor and the vast majority of patients can safely complete treatment (Table 2).[12-15] Age is also a risk factor; rates of cardiovascular complications among patients who are older than 80 years of age are higher than those among patients who are 65 to 80 years of age (36% vs. 12%).[16]

The most common neurologic sequelae of ECT are memory loss and delirium. A detailed discussion of these effects is beyond the scope of this article. The medical consultant should be aware, however, that memory loss can be retrograde (i.e., loss of recall of events before treatment), anterograde (i.e., inability to retain new memories), or both. The degree and type of memory loss are related to the electrode placement, type of stimulus, and age of the patient. In a meta-analysis, bilateral lead placement and more frequent treatments were risk factors for memory loss and disorientation.[17] In a more recent prospective study involving 347 patients in seven hospitals, advanced age was associated with an increased severity of deficits.[18] Most cognitive deficits except for loss of psychomotor function and autobiographical memory resolved within 6 months after the initiation of treatment. In contrast, in a systematic review of patients' perceptions of ECT, 29 to 55% of patients with depression reported persistent memory loss more than 6 months after ECT.[19]

Headache may occur after ECT. In a study involving 54 patients, 5 reported new persistent headache after ECT, 9 had exacerbation of or no change in headache, and 2 reported improvement of headache.[20] Although patients may report nausea, fatigue, dry mouth, or "feeling slowed," these symptoms are no more common after ECT than before treatment, and they may be related to the underlying disease itself or to antidepressant medications.[21] The use of succinylcholine as a muscle relaxant may result in myalgias, sore throat, and in rare cases, the malignant hyperthermia syndrome. Succinylcholine is contraindicated in patients with pseudocholinesterase deficiency.

Elderly patients may fall after ECT. A larger total number of ECT treatments and the presence of Parkinson's disease are associated with higher rates of falling.[22] Patients who are older than 80 years of age have higher rates of falling than those who are 65 to 80 years of age (36% vs. 14%).[16]

EVALUATION BEFORE ECT

Most ECT centers have local protocols and guidelines for pre-ECT evaluation. In a 2001 consensus statement, the American Psychiatric Association (APA) listed no absolute contraindications to ECT.[2] A few conditions, however, confer an increased risk of complications from ECT and warrant evaluation and treatment before proceeding to ECT.

ROUTINE EVALUATION

The history taking and physical examination serve to screen patients for conditions that may increase the risk associated with ECT, including cardiovascular disease (ischemic heart disease, heart failure, and arrhythmia), intracranial mass lesions, recent stroke, and pulmonary conditions (chronic obstructive pulmonary disease, asthma, and pneumonia). Before administering anesthesia, the anesthesiologist should perform an evaluation that includes an interview of the patient, a review of his or her medical history, a physical examination, and a review of laboratory data. The physical examination should include an assessment of the airway to determine the degree of difficulty one might encounter if intubation became necessary.

Table 2. Cardiac Complications and Other Outcomes in Patients Undergoing ECT.*

Study†	Publication Date	Age Range of Patients	Cardiac Complications				Other Outcomes	
			Among All Patients	Among Patients with Cardiac Disease	Minor‡	Major§	Death	Discontinuation of ECT
		yr	*no. of patients/total no. (%)*			*no. of events*		*no. of patients*
Gerring and Shields[9]	1982	20–89	12/42 (29)	12/17 (71)	20	5	1	1
Alexopoulos et al.[10]	1984	27–79	19/293 (6)	NA	9	10	1¶	2
Dec et al.[11]	1985	34–86	4/26 (15)	2/7 (29)	4	0	2¶	1
Zielinski et al.[12]	1993	53–84	25/80 (31)	22/40 (55)	31	11	0	2
Rice et al.[13]	1994	50–89	22/51 (43)	16/26 (62)	19	5	0	3
Tecoult and Nathan[14]	2001	25–88	18/75 (24)	NA	21	1	19	4
Rumi et al.[15]	2002	18–40	12/47 (26)	0	12	0	0	0

* NA denotes not available.
† Some of the studies analyzed data from consecutive patients, and others analyzed data from patients who met the investigators' inclusion criteria.
‡ Minor complications are defined as persistent hypertension, transient arrhythmia, premature atrial or ventricular contractions, transient ST-segment or T-wave changes in the absence of enzyme changes, asystole lasting more than 5 seconds, and isolated chest pain.
§ Major complications are defined as ventricular tachycardia, arrhythmia complicated by heart failure or ischemia, asystole lasting more than 10 seconds, and chest pain with electrocardiographic or enzyme changes.
¶ These deaths were not related to ECT.

Occasionally, it may be necessary to perform endotracheal intubation to maintain and protect the airway because of difficult mask ventilation, a high risk of aspiration, or the need for prolonged ventilation. Laboratory testing can be tailored to the patient's medical history and medications. ECGs are not mandatory but are advisable in patients who are older than 50 years of age, since the majority of major cardiac complications occur in this age group (Table 3).

RISK STRATIFICATION AND MEDICAL OPTIMIZATION BEFORE ECT

Unstable Cardiac Disease

There are no specific guidelines for the stratification of cardiac risk before ECT. However, we believe that ECT is analogous to a low-risk procedure as defined in 2007 in the clinical guidelines issued by the American College of Cardiology and the American Heart Association (ACC–AHA) for the perioperative care of patients undergoing noncardiac surgery.[23] ECT belongs in this category because of the short duration of anesthesia, the absence of significant fluid shifts, and the relatively low rate of major cardiac complications (Table 2). In patients with no active cardiac conditions (e.g., decompensated congestive heart failure, unstable angina, significant arrhythmias, and valvular disease), noninvasive cardiac testing is unnecessary, and practitioners can proceed with risk-factor modification as appropriate. In patients with active cardiac conditions, the particular condition informs the pre-ECT evaluation and management. The details of this evaluation are beyond the scope of this review. Data from published trials indicate that once cardiovascular conditions are stable, patients can safely complete full courses of ECT.[12,13]

Space-Occupying Lesions or Intracranial Vascular Lesions

Intracranial masses or space-occupying lesions were long considered to be contraindications to ECT because of concern that increased intracranial pressure would lead to herniation and death. Although in early case reports of such patients, the reported neurologic outcomes were poor, these studies were probably subject to selection bias, since neurologic deterioration after ECT prompted diagnosis of an intracranial lesion in all but 1 of the 35 patients.[26] In more recent case series, patients with known intracranial lesions who have normal neurologic examinations and minimal or no edema or mass effect on neuroimaging have safely undergone ECT.[27] In patients with abnormal neurologic examinations or known masses, neuroimaging should be performed to look for changes that are

Table 3. Evaluation of the Healthy Patient Undergoing ECT.*

Test	Recommendation	Rationale
History and physical examination	Screen for symptoms and signs of unstable angina, congestive heart failure, and lower respiratory tract infection; inquire about exercise capacity; measure vital signs, including oxygen saturation; ask about symptoms that may indicate acute stroke or increased intracranial pressure; perform a detailed neurologic examination, including funduscopy if necessary	Unstable angina, congestive heart failure, and lower respiratory tract infection all warrant further investigation before proceeding[23]; low self-reported exercise capacity suggests possible undiagnosed cardiopulmonary disease; new symptoms or an abnormal neurologic examination warrant neuroimaging to rule out intracranial mass or other structural brain disease; symptoms or signs of acute stroke warrant delay of ECT and immediate consultation with a neurologist
Medication and family history	Obtain a complete medication history, including use of herbal medications; inquire about drug allergies, including previous reactions to anesthetics (e.g., history of malignant hyperthermia); inquire about a personal or family history of prolonged neuromuscular blockade due to pseudocholinesterase deficiency	Ginkgo biloba, ginseng, St. John's wort, valerian, and kava are potential central nervous system depressants that may alter the seizure threshold or increase the risk of cognitive side effects of ECT[24]; a previous adverse reaction to anesthesia may require modification of the anesthetic technique and needs to be communicated to the anesthesiologist
Laboratory studies	Measure serum electrolyte, blood urea nitrogen, and creatinine levels in patients receiving diuretics or antihypertensive medications and in patients with malnutrition, congestive heart failure, diabetes, or known renal disease; administer a pregnancy test in women of childbearing age	Electrolyte abnormalities may increase the risk of post-ECT arrhythmia; hyperkalemia increases the risk associated with succinylcholine; ECT is probably safe in pregnancy, and in most cases the benefits will outweigh the risks, although pregnancy will require modification of the anesthetic technique, positioning of the patient, and monitoring; the obstetrician should participate in the informed-consent process[25]
ECG	Obtain an ECG in patients with symptoms suggesting cardiac disease, in patients older than 50 years of age, and in patients with known cardiac disease or a previously abnormal ECG	ECG may detect silent cardiac disease but rarely leads to intervention before ECT in the absence of findings from the history or examination; major cardiac complications due to ECT are rare, and most occur in patients with known cardiac disease

* ECG denotes electrocardiogram.

consistent with increased intracranial pressure. We are aware of one published report of successful ECT in a patient with an intracranial lesion and surrounding edema. Prospective studies are needed to assess the safety of ECT in this high-risk group.[28]

The evidence regarding the safety of ECT in patients with intracranial vascular lesions is limited. The APA lists this as a high-risk condition because of concern that the increased rate–pressure product during and after the seizure could lead to aneurysmal rupture.[2] We are unaware of any reports of ruptured cerebral aneurysms due to ECT. In the largest case series to date, Najjar and Guttmacher reported that there were no complications in six patients with intracranial vascular lesions who underwent ECT.[29] In most cases, short-acting intravenous medications (e.g., beta-blockers, sodium nitroprusside, and hydralazine) were used to manage blood pressure, and in all cases the lesions were small (<10 mm in diameter). Before ECT is performed in patients with intracranial masses or vascular lesions, consultants in neurology, neurosurgery, or both, as well as the anesthesiologist, should participate in the evaluation of the patient and in the process of informed consent.

Recent Stroke

Data regarding preexisting cerebrovascular disease in patients undergoing ECT are limited, but in one study involving patients with a history of strokes there were no lasting neurologic complications after ECT.[30] Transient delirium developed in approximately one quarter of the patients. Among patients with a recent or acute stroke, changes in intracranial pressure and cerebral blood flow induced by ECT pose a risk of ischemia or hemorrhage. In the above study, 5 of the 14 patients received ECT within 1 month after a stroke, and none had major complications.[30] In keeping with suggested approaches to the treatment of patients undergoing noncardiac surgery, we suggest a delay of ECT until at least 1 month after acute stroke.[31] In addition, tight control of blood pressure that minimizes both hypertension and

hypotension may reduce the risks of bleeding and further ischemia, respectively.

Uncontrolled Hypertension

Given the expected increase in arterial pressure due to ECT, clinicians should delay elective ECT in patients with uncontrolled hypertension and begin antihypertensive therapy. The available literature does not provide data with which to estimate a threshold blood pressure for the safe administration of ECT. However, there is an expected increase of more than 25 mm Hg in both diastolic and systolic blood pressures.[7] In the absence of clear guidelines, we recommend the use of the guideline in the seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure for patients with hypertension who are preparing for ECT.[32] Clinicians should institute antihypertensive therapy if the patient's blood pressure is 140/90 mm Hg or higher unless he or she has had a recent stroke. We suggest avoiding the use of beta-blockers given the potential for reduced seizure duration and a possible resultant decrease in the efficacy of ECT.[33,34]

MANAGEMENT OF PREEXISTING MEDICAL CONDITIONS

Table 4 details recommended strategies for the management of chronic medical conditions in patients for whom ECT is planned. In most cases, patients should take their usual medications, including cardiac and antireflux medications, until the morning of the procedure. Exceptions include theophylline, herbal medications, and oral diabetes medications.

Although the absolute risk of cardiac complications is low, patients with underlying cardiac disease are at higher-than-average risk. Changes in blood pressure and heart rate increase myocardial oxygen demand and may increase the risk among patients with coronary artery disease, congestive heart failure, or aortic stenosis. In these patients, clinicians should establish that the cardiac condition — congestive heart failure, coronary disease, or valvular disease — is stable and that no exacerbation is present that might increase the risk. The consultant should make the anesthesiologist aware of the coexisting cardiac condition and collaborate on the proposed pre-ECT care.

MANAGEMENT OF COMPLICATIONS AFTER THE PROCEDURE

PROLONGED BLOOD-PRESSURE ELEVATION

Asymptomatic elevation of blood pressure may extend beyond the expected recovery period (typically 20 to 30 minutes). Intravenously administered antihypertensive medications that may prevent postprocedural tachycardia and hypertension include labetalol, esmolol, nicardipine, and diltiazem. Labetalol and esmolol blunt the blood-pressure and heart-rate response to ECT in a dose-dependent fashion.[35,42]

The routine use of prophylactic beta-blockers is controversial. Several studies have shown a shortened duration of seizures in patients treated with beta-blockers; it remains uncertain whether the potential reduction in seizure duration leads to reduced treatment efficacy.[33,34] This is not a consistent observation; in other studies, beta-blockers had no effect on the duration of seizures.[35,43] For patients who are not already receiving a beta-blocker and who do not meet independent criteria for beta-blocker therapy, we believe that the risk–benefit calculation favors selective rather than universal use. In low-risk patients, the potential for reduced efficacy of ECT outweighs any potential benefit of beta-blockers. We recommend reserving the use of prophylactic, short-acting intravenous beta-blockers for patients at high risk for complications, such as those who have had previous prolonged hypertension or have a coexisting condition that requires tight blood-pressure control (e.g., moderate or severe aortic stenosis, intracranial or other aneurysms, or recent myocardial ischemia or infarction).

ASYSTOLE OR BRADYCARDIA

Prolonged asystole or symptomatic bradycardia that does not resolve spontaneously should be managed according to advanced cardiac life-support guidelines. Early case reports suggested that the use of beta-blockers was a risk factor for prolonged asystole.[44,45] Larger studies designed to assess the effect of intravenous beta-blockers on hemodynamics have not shown higher rates of prolonged asystole.[42,43]

Subconvulsive stimuli, bilateral electrode placement, and advanced age are risk factors for asystole.[44,45] Burd and Kettl prospectively studied patients undergoing ECT and documented asystole lasting 5 seconds or more in 25 of 38 elderly pa-

Table 4. Management of Preexisting Conditions.*

Condition	Recommendations	Rationale
Stable chronic hypertension with blood pressure ≤140/90 mm Hg	Continue usual antihypertensive medication through the morning of procedure	Blood pressure increases during the postictal phase of ECT; systolic pressure increases from 29–48% during ECT, and diastolic pressure from 24–60%[7,15]
Chronic or new-onset hypertension with blood pressure >140/90 mm Hg	Start antihypertensive medications according to JNC-7 guidelines[32]; delay ECT until blood pressure is <140/90 mm Hg; avoid beta-blockers	Beta-blockers may shorten the seizure duration and reduce the efficacy of ECT[32-35]
Asymptomatic or stable coronary artery disease	Continue medications such as aspirin, statins, antihypertensive agents, and antianginal medications, including nitrates for chronic cardiac conditions; continue aspirin and clopidogrel in patients with coronary stents	Discontinuation of long-term cardiac medications on the morning of the procedure increases the risk of cardiac ischemia
Aortic stenosis	Perform echocardiography to assess severity, if it has not been performed within the past year or if there is a change in symptoms; consult cardiologist and reassess indication for ECT if stenosis is moderate or severe	Limited data suggest that ECT is safe with the use of short-acting intravenous beta-blockers to minimize procedure-related hypertension and tachycardia[36]
Implanted pacemaker	Test the pacemaker before and after ECT; place magnet at the patient's bedside in the event that electrical interference leads to pacemaker inhibition and bradycardia	In a study involving 26 patients with pacemakers who were undergoing ECT, 1 patient had postprocedural supraventricular tachycardia, but no clinically significant arrhythmias occurred; all pacemakers functioned normally after ECT[37]
ICD	Turn off detection mode of ICD during ECT; perform continuous ECG monitoring throughout treatment with careful attention to grounding; place resuscitative equipment by the patient's bedside in the event that external defibrillation is necessary	ECT appears to be safe in patients with an ICD[37]
Atrial fibrillation	Continue outpatient medications for control of heart rate; control heart rate with calcium-channel blockers if needed; manage anticoagulation as described below	Few data exist, but ECT appears to be safe in patients with atrial fibrillation[38]; patients may have conversion to and from sinus rhythm during ECT; the effect of spontaneous rate conversion on embolization rates is unknown
Need for long-term anticoagulation	Continue anticoagulation to maintain an international normalized ratio of up to 3.5, unless there is an increased risk of intracranial hemorrhage (e.g., intracranial mass or aneurysm)	In a study involving 33 patients with an international normalized ratio of ≤3.5, there were no complications from ECT[39]
Asthma or chronic obstructive pulmonary disease	Discontinue theophylline by tapering the dose, if possible; continue outpatient regimen of bronchodilators and inhaled corticosteroids; if an exacerbation is present on evaluation, provide standard treatment — inhaled beta-agonists and, if necessary, corticosteroids — before proceeding with ECT	Theophylline increases the risk of status epilepticus after ECT[40]; in a study involving 34 patients with asthma, 12% of the patients had an exacerbation, all of whom had a response to standard therapy and were able to complete ECT[41]
Diabetes	Measure blood glucose levels before and after ECT treatment; give half the usual amount of long-acting insulin the morning of the procedure; withhold oral agents until patient can eat; provide short-acting insulin to treat elevations in blood glucose level; perform ECT early in the morning if possible	The effect of ECT on blood glucose is unpredictable because of changes in diet, appetite, and energy level that may result from ECT; individual ECT treatments raise blood glucose levels in patients with diabetes to the same degree as in patients without diabetes
Pregnancy	The informed-consent and risk-stratification process should include an obstetrician and an anesthesiologist; in addition to standard monitoring of the patient, noninvasive fetal monitoring should be used after 14–16 weeks; after 24 weeks, a nonstress test with a tocometer should be performed before and after treatments[24]	Pregnancy would require modification of the anesthetic technique, positioning of the patient, and monitoring requirements[24]

* ECG denotes electrocardiographic, ICD implantable cardioverter–defibrillator, and JNC-7 seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure.

tients.[5] Surprisingly, rates of asystole were lower among patients with preexisting heart block or rhythm abnormalities than among patients without conduction-system disease (16.0% vs. 53.8%; P<0.05). The basis for this observation is unknown. Pretreatment with atropine in patients who had a history of asystole after ECT reduced the rate of recurrent asystole during subsequent treatments from 7.3% to 0.7%. Although pretreatment with atropine may protect patients from asystole, it also increases the periprocedural rate–pressure product through anticholinergic-mediated tachycardia.[46] Because of this theoretical increase in cardiac stress, the routine use of prophylactic atropine is controversial. We recommend restricting its use to patients with a history of ECT-induced asystole. Since glycopyrrolate also prevents bradycardia during ECT and has a smaller effect on the rate–pressure product than atropine, it may be preferable in patients in whom the added chronotropic stress of atropine is undesirable.[47,48]

MYOCARDIAL ISCHEMIA

Symptomatic myocardial ischemia is rare after ECT, and there is insufficient evidence to support pharmacologic therapy for the prevention of post-ECT myocardial ischemia. The potential for an effect on seizure duration argues against the routine use of prophylactic beta-blockers, and the 2001 APA guidelines make no specific recommendation on this point.[2] The 2007 ACC-AHA guidelines for noncardiac surgery do not recommend beta-blockers for low-risk surgery, even in patients with multiple risk factors.[23] Although this issue is controversial, we recommend that prophylactic beta-blockers not be used routinely. If a patient is already receiving beta-blocker therapy because of a recent myocardial infarction (within the previous 6 weeks) or another indication for beta-blocker therapy that is independent of ECT, then it is reasonable to continue treatment with the beta-blocker.

Clonidine, an α_2-agonist, reduces the rates of death and myocardial ischemia after major noncardiac surgery by reducing the sympathetic outflow.[49] However, the value of clonidine in reducing the risk of cardiac complications after ECT is untested. Pending further study, we do not recommend this strategy. More recently, the prophylactic use of the opioid agent remifentanil has been shown to decrease the postprocedural heart rate and blood-pressure elevation in patients undergoing ECT.[50]

HEADACHE

Post-ECT headache generally responds to ketorolac, ibuprofen, or acetaminophen. Serotonin receptors may be mediators of ECT-induced headache. In a study involving eight patients in whom 13 post-ECT headaches developed, intranasal sumatriptan provided a response rate of 85% (11 of 13 headaches) at 1 hour.[51] The prophylactic use of a single dose of 600 mg of ibuprofen also reduces the likelihood of an ECT-related headache.[52]

CONCLUSIONS

ECT is generally a safe procedure with predictable hemodynamic responses. There are no absolute contraindications. Pertinent preexisting medical conditions that put patients at higher risk include hypertension, coronary artery disease, congestive heart failure, aortic stenosis, implanted cardiac devices, atrial fibrillation, obstructive lung disease, and asthma. A standardized pre-ECT evaluation will optimize the safety of this procedure. In an initial evaluation of a patient who is at high risk for complications from ECT, including prolonged blood-pressure elevation, asystole, myocardial ischemia, and headache, the medical consultant should address the possible need for risk stratification, management of coexisting medical conditions, and strategies to reduce the risk of these complications.

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