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22.–23.5.2003, Tampere

Hypertrophic cardiomyopathy overview and management strategies

Martin D. Abel

Introduction

Our understanding of hypertrophic cardiomyopathy (HCM) has undergone a significant change over recent years. In the old definition, patients with HCM were considered to have severe myocardial hypertrophy with no known etiology. In the new conception of the disease, HCM is thought always to be a genetic disorder, arising from either an inherited genetic abnormality with an autosomal dominant inheritance pattern or, perhaps, more commonly due to spontaneously occurring gene mutations.[1] Pathologically, there is hypertrophy of myocytes and disarray of sarcomeres. Left ventricular function is normal or supranormal but there is usually significant diastolic dysfunction. The clinical course may be benign or result in heart failure and sudden death. Historically, many terms have been used to describe variants of HCM including, idiopathic hypertrophic subaortic stenosis (IHSS), subaortic stenosis, dynamic subaortic stenosis (DSS), idiopathic hypertrophy, asymmetric septal hypertrophy (ASH), and familial hypertrophy. Today the preferred terminology is hypertrophic cardiomyopathy without or with obstruction (hypertrophic obstructive cardiomyopathy or HOCM).

Genetic etiology

Numerous gene mutations have now been discovered that lead to HCM. Greater than 36 genetic mutations in the β-myosin heavy chain are responsible for 35–50 % of all cases of HCM. Genetic modifications in myosin binding protein C account for 15–20 % of HCM cases. More than 7 genetic mutations coding for troponin T cause 15–20 % of HCM cases and mutations in tropomyosin, troponin I and myosin light chains are responsible for less than 5 % of HCM cases. Amino acid substitutions can result in very different phenotypic characteristics. For example, in variants of HCM caused by β-myosin heavy chain abnormalities, valine for leucine substitution at position 908 results in a population with a low penetrance of the gene, a benign course and a low incidence of sudden death. On the other hand, glutamine substitutions for arginine at positions 403 or 719 result in populations with a high penetrance of the abnormal gene, severe ventricular hypertrophy and sudden death.[1]

Pathogenesis

It is thought that mutations in the β-myosin heavy chain results in destabilization of the sarcomere due to increased breakdown of myosin or through impaired actin-myosin binding. This, in turn leads to compensatory hypertrophy which is modulated by local factors. The exact reason for asymmetric hypertrophy is unknown. Other genetic factors may also play a role. For example, patients with HCM who have the angiotensin converting enzyme (ACE) genotype DD are much more likely to have severe left ventricular (LV) hypertrophy and sudden death.[1] HCM caused by amino acid substitutions

in the β-myosin heavy chain or troponin T manifests by the 2nd or 3rd decade of life whereas mutations in cardiac myosin-binding protein C are associated with reduced penetrance until mid-life and better survival than that observed with the former two causes of HCM.[2]

Diagnosis

The clinical diagnosis of HCM is established most easily and reliably with 2-dimensional (2D) echocardiography.[3] Typically, the LV is hypertrophied but not dilated. Obviously, other causes of LV hypertrophy must be excluded, e.g., hypertension or valvular heart disease. The ECG is abnormal in 75 % to 95 % of HCM patients ranging from LV hypertrophy to dysrhythmias. However, when trying to establish a family pedigree it is likely that not all genetically affected individuals will be either symptomatic or have echocardiographic or electrocardiographic features of HCM.[3] Genetic studies combined with phenotyping will likely become the diagnostic approach in future years.

Clinical manifestations

Of those patients presenting clinically with HCM about two-thirds will have features of obstruction (figure 1) resulting in dyspnea, hypotension and syncope. Diastolic dysfunction also can cause dyspnea. LV hypertrophy can result in angina, and dysrhythmias can produce syncope and sudden death.

In patients with LV outflow tract (LVOT) obstruction there is typically asymmetric septal hypertrophy producing a narrowing of the LVOT which is accentuated during systole. The increased velocity of blood traversing the LVOT produces a drop in pressure (Venturi effect) thereby pulling the anterior mitral valve leaflet more anteriorly and causing secondary coaptation problems of the mitral valve apparatus. This in turn leads to mitral regurgitation (figure 2).

Medical management

Medical therapy consists primarily in the use of negative inotropic medications like beta-blockers, calcium-channel blockers, principally verapamil, and occasionally disopyramide, a type IA antiarrhythmic agent that may reduce LVOT gradients at rest. This effect is thought to be mediated by slowing of the LV ejection acceleration. In the hemodynamic management of patients with HOCM attention must be paid to loading conditions and contractility. Increases in contractility of the LV will result in an increased pressure gradient from the LV to the aorta across the LVOT. Beta-blockers and volatile anesthetics have a salutary effect in mitigating the catecholamine stress response to noxious surgical stimuli or even laryngoscopy and intubation of the trachea. Other factors that result in an increased LVOT gradient are decreases in afterload as may occur with vasodilatation from any cause and loss of preload as occurs with hypovolemia. Thus, the optimal management of these patients includes maintenance of afterload by avoiding vasodilatation and adequate fluid therapy to keep preload relatively high. However, in patients failing medical therapy some kind of intervention is usually necessary to ameliorate obstructive symptoms.

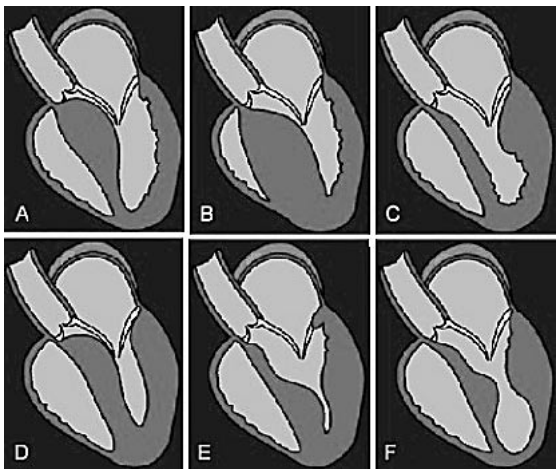
Interventional therapy

Interventional therapies to relieve obstruction include dual-chamber pacing, surgical myectomy and more recently alcohol septal ablation therapy. In patients at high risk for sudden death an internal cardiac defibrillator (ICD) may be indicated.

1. Chronic dual-chamber pacing

Chronic dual-chamber pacing has been associated

Fig. 1
HCM – patterns of hypertrophy:
A – basal septum
B – entire septum
C – isolated lateral free wall
D – concentric
E – apical
F – mid-ventricular



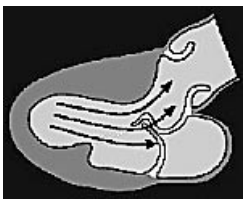


Fig 2. (See text for explanation)

with improvement in symptoms with a reduction in LVOT gradient. However, many of the earlier studies were uncontrolled and observational in nature. More recently, several randomized, crossover clinical trials have shown little objective evidence of improvement with a significant placebo effect.[3,4] Nevertheless, a dual-chamber pacing trial prior to surgical myectomy may be of value in selected patients but its effects must be carefully evaluated in a hemodynamic laboratory.

2. Surgical septal myectomy

LV septal myectomy is the "gold-standard" of therapy for severely symptomatic patients with large LVOT gradients unresponsive to or intolerant of medical therapy. Typically, such patients have provocable LVOT gradients > 50 mm Hg. The operation involves resection of a section of muscle from the LV septum just beneath the aortic valve opposite the anterior mitral valve leaflet and extending just beyond the distal margins of mitral leaflets. Adequacy of the resection is judged echocardiographically and hemodynamically by measurement of the LVOT gradient before and after myectomy with provocative maneuvers.

3. Alcohol septal ablation

The anatomy of the blood supply to the LV septum is delineated using conventional angiographic techniques common to coronary angiography and angioplasty. Favorable anatomical features include a well-defined first septal perforating artery that supplies only the basal septum. However, because of considerable variability in the distribution of the first septal perforating artery it is essential to define its take-off from the left anterior descending coronary artery and its distribution in patients with HCM who are being considered for alcohol septal ablation.[5]

Variability in the size and distribution of the FSPA (first septal perforating artery) in patients without HCM was substantial. Areas of the heart other than the basal septum were supplied in some patients by the FSPA. In other patients the FSPA did not supply the entire basal septum. Similar findings were noted in patients with HCM.

A small volume of absolute alcohol is injected into the target septal perforator coronary artery branch to produce a focal myocardial infarction. This infarct results in reduced basal septal thickening thereby enlarging the LVOT and decreasing the Venturi pull on the anterior mitral valve leaflet. Reductions in LVOT gradient are claimed to be similar to those resulting from surgical myectomy.[6] However, a recent comparative analysis showed surgery to be superior to ablation in reducing resting and provocable gradients.[7] However, there are no published randomized or controlled studies comparing alcohol septal ablation to surgical myectomy in HCM.[8] Septal ablation with surgery or alcohol is associated with a finite morbidity and mortality. Complications include complete heart block requiring a permanent pacemaker (much higher incidence with alcohol)[9], coronary dissection (alcohol), ventricular septal defect (both), and large anterior infarction (alcohol). Long-term follow-up is still lacking for alcohol septal ablation. Maron has raised the concern that "ablation alone potentially creates a permanent, electrically unstable substrate for lethal reentrant ventricular tachyarrhythmias by virtue of the healed intramyocardial septal scar in some HCM patients who are already undoubtedly predisposed to arrhythmogenesis; this consideration raises some uncertainty regarding the long-term risks of alcohol septal ablation."[3,10]

Sudden death

In the majority of HCM patients (55 %) sudden death is very uncommon. However, in certain subsets of HCM, sudden death occurs more commonly often with vigorous physical exertion. HCM is the most common cause of cardiovascular sudden death in young people, including trained competitive athletes (basketball, football especially in black athletes).[3] The following markers are associated with sudden death: prior cardiac arrest or spontaneous sustained ventricular tachycardia; family history of premature HCM-related death; exertional syncope or near-syncope, when arrhythmia-based or unrelated to neurocardiogenic mechanisms; prolonged bursts of nonsustained ventricular tachycardia on ambulatory (Holter) ECG recordings; hypotensive blood pressure response to exercise; and extreme LV hypertrophy in adolescents and young adults.[3] Presentation of HCM in young children is very uncommon because the development of the disease is usually progressive over time. Certain genetic defects responsible for HCM are associated with a higher propensity for sudden death as noted above. Premature death is seen in some β-myosin heavy chain mutations (e.g., Arg403Gln and Arg719Gln) and some troponin T mutations. On the other hand, mutations of myosin-binding protein C and α-tropomyosin result in a more benign natural history. Access to the molecular biology of HCM does not yet represent a clinically relevant strategy that routinely affects disease management.[3] The role of invasive

strategies like electrophysiologic testing with programmed stimulation to induce ventricular dysrhythmias to detect patients at high risk for ventricular fibrillation is still experimental.[3] According to Maron most HCM patients should undergo a risk stratification assessment.[3] This assessment would involve a cardiologist taking a careful history and performing a physical examination followed by noninvasive testing utilizing 2-D echocardiography, ambulatory (Holter) electrocardiography, and treadmill or bicycle exercise testing.[3]

Conclusion

A substantial evolution in our understanding of the etiology, pathogenesis, genetic basis, clinical presentation and treatment for HCM has occurred over the last few years. Further medical advances will likely lead to management strategies that actually alter the natural history of the disease itself. It is also important to remember that while we, as physicians, see a disproportionate number of symptomatic HCM patients at all ages, many people with the HCM genotype live a normal life-span with a good quality of life. ❐

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TEE – a mirror of cardiac performance

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Introduction

Echocardiography was first used in the operating room in the 1970s. Initially, echocardiographic images were obtained epicardially by cardiologist. The widespread use of echocardiography intraoperatively followed the introduction of transesophageal echocardiography (TEE) in the 1980s and did not become commonplace until two-dimensional (2-D) and color Doppler imaging with high-frequency transducers became available in the mid-1980s. With the improved quality of imaging, anesthesiologists and surgeons were able to use TEE intraoperatively to confirm the adequacy of valve reconstruction and other surgical repairs, diagnose myocardial ischemia, determine the cause of acute hemodynamic collapse and other hemodynamic perturbations, and provide diagnostic information that was not obtained preoperatively. Intraoperative TEE (IOTEE) information has enabled surgeons to correct inadequate surgical repairs before patients leave the operating room thereby reducing the need for early reoperation. IOTEE also has facilitated the prevention and early treatment of perioperative complications.[1]

Although other intraoperative monitoring devices can provide much of the information necessary for taking care of patients during surgery, TEE offers important advantages over other diagnostic monitoring techniques. By mirroring both anatomic detail and blood flow IOTEE can facilitate diagnosis, allow the institution of specific treatments, and monitor interventions throughout the operative course without disrupting surgical technique.[1]

There are some important limitations to the use of IOTEE. Some regions of the heart and great vessels cannot be well visualized, e.g., ascending aorta and aortic arch. IOTEE is generally considered to be safe, but insertion and manipulation of the TEE probe can produce pharyngeal and/or laryngeal trauma, dental injuries, esophageal trauma or bleeding, arrhythmias, respiratory distress, hemodynamic effects and even deaths attributable to TEE. The inaccurate interpretation of TEE images by inexperienced examiners might result in incorrect clinical decisions producing unnecessary perioper-

ative complications. IOTEE assessment may divert the anesthesiologist from the care and attention that he/she needs to pay to other intraoperative responsibilities.[1]

Role of echocardiography in surgery

In 1996 the American Society of Anesthesiologists and the Society of Cardiovascular Anesthesiologists created a task force to develop evidenced-based practice guidelines related to the use of IOTEE perioperatively. Members of this task force included anesthesiologist from academic institutions across the USA and cardiology representation from the American College of Cardiology and the American Society of Echocardiography. In the report the indications for the use of IOTEE were graded into 3 categories based on the level of scientific evidence available at the time.

cardiac output, filling pressure) and measurements obtained by more conventional tests (e.g., thermodilution). Studies comparing TEE and thermodilution measurements of cardiac output report wide ranges in correlation coefficients (R = 0.72–0.97), bias estimates (0.03–1.01 l/min), and limits of agreement. The frequency with which TEE detects hemodynamic disturbances has not been studied. Moreover, there is little evidence beyond case reports to confirm that hemodynamic monitoring by TEE results in improved clinical outcomes.

EXPERT OPINION: Quantitative analysis of TEE information may increase its sensitivity in detecting small changes in ventricular dimensions or ejection, a capacity that would dramatically enhance conventional hemodynamic monitoring, but it is time consuming and requires considerable skills. Until automated analysis systems overcome these limitations, intraoperative TEE will remain a largely qualitative tool for assessing hemodynamic function. Even with these limitations, however, the task force believes that TEE provides more accurate estimates of preload (end-diastolic volume) than the pulmonary artery catheter. Preload is physiologically determined by sarcomere length, a variable more accurately estimated by volume than by pressure measurements. When compared with the pulmonary artery catheter, TEE may be more expedient, because it can be inserted more quickly and without sterile technique; safer, because it does not enter the great vessels and heart; and more comprehensive, because it provides more global hemodynamic information about the performance and structure of the heart.

Although direct evidence is lacking, the task force believes that detecting acute hemodynamic distur-

Criteria	Indications for the Use of Intraoperative TEE.[1]		
	Category I	**Category II**	**Category III**
Scientific evidence or Expert Opinion	Strongest	Supported by weaker evidence and expert consensus	Little current scientific or expert support
Clinical Outcomes	Frequently improved	May be useful in improving clinical outcomes in these settings	Infrequently useful in improving clinical outcomes in these settings
Clinical Indications	Often indicated, depending on individual circumstances	Appropriate indications are less certain	Appropriate indications are uncertain

The table above and some of the following sections are selected and abridged from this report: "Practice guidelines for perioperative TEE. A report by the ASA and the SCA Task Force on TEE." Thys DN, Abel MD et al. Anesthesiology 1996; 84: 986–1006[1]

Hemodynamic function

TEE has been used extensively for the evaluation (monitoring of presence, followed by determination of etiology) of hemodynamic and global ventricular function. Some have used TEE to estimate standard hemodynamic variables (e.g., filling pressures, cardiac output) that are normally obtained by other invasive techniques, such as pulmonary artery catheterization, whereas others have used it to quantify cardiac dimensions, intracardiac flow rates, and overall cardiac performance. Such measurements were previously not readily obtainable in the operating room or ICU.

SUMMARY OF THE EVIDENCE: Current studies provide conflicting information about the correlation between TEE estimates of hemodynamic indexes (e.g.,

bances during surgery improves clinical outcomes. These benefits are realized by using TEE to diagnose the hemodynamic problem (e.g., hypovolemia, myocardial depression) and to suggest appropriate therapy (e.g., volume expansion, inotropic therapy). The task force believes that failing to take action to correct or prevent hemodynamic disturbances increases the risk of end-organ damage and perioperative mortality.

RECOMMENDATIONS (INDICATIONS): An increased risk of hemodynamic disturbances during the perioperative period is a category II indication for perioperative TEE. Increased risk may occur when conditions associated with the patient (e.g., congestive heart failure, valvular heart disease, abdominal aortic aneurysm, preeclampsia, trauma, burn injuries), procedure (e.g., vascular surgery, CPB, extensive tumor resection, liver transplantation, total hip replacement), or clinical setting (e.g., difficulties in inserting central venous pressure catheters, inability to estimate blood loss, poor hospital-specific conditions, such as complication rates for a specific procedure) predispose to hemodynamic disturbances. The emergent use of perioperative TEE to determine the cause of acute, persistent, and life-threatening hemodynamic disturbances in which ventricular function and its determinants are uncertain and have not responded to treatment is a category I indication.

RECOMMENDATIONS (PROFICIENCY): Anesthesiologists with basic TEE training should be able to make qualitative assessments of hemodynamic status and should have a cognitive understanding of more sophisticated TEE techniques for quantifying hemodynamic function.

Wall motion, myocardial ischemia, and coronary artery disease

Hemodynamic and other physiologic stresses during the perioperative period increase the risk of perioperative myocardial ischemia, especially among patients with coronary artery disease and peripheral vascular disease. Traditional methods for monitoring myocardial ischemia during surgery, such as continuous ECG, have limited sensitivity in the early detection of tissue injury. A growing body of evidence therefore has examined the role of TEE in detecting ischemia during both cardiac and noncardiac surgery.

SUMMARY OF THE EVIDENCE: There is good evidence that the development of regional ventricular dysfunction during surgery increases a patient's risk of developing perioperative myocardial infarction (MI) and sudden death. TEE can detect regional ventricular dysfunction, but there is little evidence about its accuracy, because neither a reliable reference standard is available, nor is it certain that the wall motion abnormalities reflect true myocardial ischemia. Most studies have examined the accuracy of TEE relative to intraoperative ECG tracings, an imperfect reference standard, and find weak concordance. Intraoperative TEE evidence of regional ventricular dysfunction is reported to occur in 27–100 % of cases in which there is ECG evidence of ischemia and in 56–85 % of cases in which ECG evidence of ischemia is lacking. The reportedly high incidence of intraoperative regional ventricular dysfunction (21–30 % of cardiac surgery cases, 10–52 % of vascular cases, and 20–60 % of non-cardiovascular cases) and postoperative dysfunction (47–60 % of cardiac surgery patients) raises important questions about the frequency of false-positive findings.

Moreover, there is little direct evidence that the detection of regional ventricular dysfunction or other TEE evidence of ischemia results in improved perioperative clinical outcomes or long-term survival. This lack of evidence is mainly due to the absence of studies examining these outcomes. Studies of cardiac surgery patients report that TEE findings were "valuable or essential" or resulted in a change in therapy (graft revision, hemodynamic support) in 2–12 % of patients, but there is no direct evidence that patients experience better outcomes as a result of these changes. Intraoperative TEE also is capable of evaluating myocardial perfusion patterns, coronary artery anatomy, and graft patency, but few studies have examined whether this information improves clinical outcomes.

EXPERT OPINION: Evidence from animal experiments and angioplasty studies suggests that wall motion abnormalities generally precede ECG changes during myocardial ischemia. The task force believes that TEE provides a more meaningful reference standard for ischemia than ECG. The limitations of TEE also are recognized, however. Interpretations of wall motion and thickening often are more subjective than quantifiable ECG changes (e.g., ST-segment depression). TEE interpretations can be influenced by translational motion of the heart, bundle branch block, and ventricular pacing. A marked worsening of segmental wall motion and thickening is required (in the absence of similar global changes) to strongly suggest the diagnosis of ischemia; less pronounced changes are interpreted inconsistently, even by experts. Moreover, all segmental wall mo-

tion abnormalities are not indicative of myocardial ischemia. Segmental wall motion abnormalities not caused by ischemia can occur because of preexisting disease (e.g., prior MI, myocarditis) or confounding intraoperative events (e.g., myocardial stunning after cardiopulmonary bypass [CPB]). Protocols have recently been suggested for distinguishing intraoperatively between stunned and infarcted myocardium, but they require further evaluation to be validated. Because an automated system for analyzing wall motion and thickness is currently unavailable for TEE, only qualitative wall motion assessment is available in real time. Moreover, real-time assessment of segmental wall motion may be less accurate than off-line (laboratory) assessment. When TEE is used to evaluate wall motion, the yield will increase with the use of multiple planes and methods that facilitate temporal comparisons (e.g., side-by-side cine loops).

Although there is little direct evidence that TEE detection of myocardial ischemia improves clinical outcome, the task force believes that indirect evidence can be extrapolated from non-TEE studies showing that early treatment of myocardial ischemia and MI improves survival. Intraoperative TEE detection of ischemia permits corrective interventions, including alterations in surgery, anesthetic management, and postoperative triage, which can prevent perioperative complications. For example, TEE detection of post-CPB myocardial ischemia during coronary artery bypass graft surgery permits, if indicated, immediate revascularization before the patient leaves the operating room, thereby reducing the risk of perioperative MI. Both indirect evidence and expert opinion suggest that these measures improve clinical outcomes.

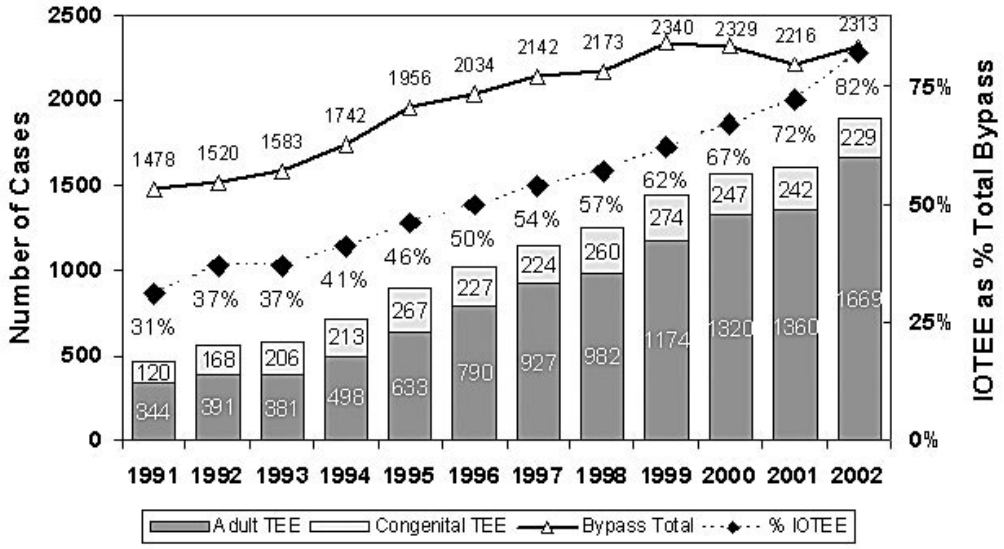
RECOMMENDATIONS (INDICATIONS): An increased risk of myocardial ischemia or infarction during the perioperative period is a category II indication for perioperative TEE. An increased risk may occur when conditions associated with the patient (e.g., history of prior MI or coronary artery disease, left ventricular dysfunction, dysrhythmias), procedure (e.g., coronary artery bypass graft, operations on the great vessels or that involve aortic cross-clamping, noncardiac intrathoracic surgery, upper abdominal procedures), or clinical setting (e.g., anticipated duration of surgery, hospital-specific factors) predispose to myocardial ischemia or MI. Use of perioperative TEE to evaluate myocardial perfusion, coronary artery anatomy, or graft patency is category III indications. The argument for using TEE is strengthened when ECG monitoring cannot provide accurate information, such as in patients with conduction disorders or in procedures that interfere with ECG lead placement. The argument for using TEE is weakened when clinical factors (e.g., preexisting regional ventricular dysfunction) limit the accuracy of wall motion interpretations.

RECOMMENDATIONS (PROFICIENCY): Anesthesiologists with basic TEE training should be able to use TEE to detect unequivocal changes in segmental wall thickening and motion (e.g., from normal wall motion to akinesis) and should be able to distinguish these changes from artifacts (e.g., translational motion of the heart, changing cross section, image dropout, abnormal ventricular activation). Subtle changes in segmental wall motion and thickening, however, are difficult to detect, even for experts. The ability to evaluate or quantify such changes in association with myocardial ischemia and MI requires advanced training.

Air emboli

SUMMARY OF THE EVIDENCE: Intraoperative TEE can detect air bubbles in 8–60 % of patients undergoing neurosurgery and 11–79 % of patients undergoing cardiac surgery. Current evidence is inadequate, however, to determine whether these emboli increase the risk of neurologic complications or whether intraoperative TEE monitoring for air emboli improves clinical outcomes.

EXPERT OPINION: TEE is an extremely sensitive test for air (emboli as small as 2 micro meter usually can be detected) but the clinical significance of these bubbles is unclear. Animal studies suggest that air entrainment greater than 1 cc/kg increase the risk of neurologic complications, but the threshold value for safe air volumes in humans is uncertain. The task force believes that patients benefit when TEE detects air during cardiotomy and neurosurgical procedures. During cardiotomy, venting procedures before cessation of CPB can eliminate retained air, and the task force believes that such measures decrease the patient's risk of embolic neurologic events, right ventricular failure due to right coronary artery air embolism, and MI. TEE offers similar benefits during sitting craniotomies, especially if the patient has not been screened preoperatively for patent foramen ovale. Patent foramen ovale appears to be a risk factor for stroke, and intraoperative maneuvers can induce intracardiac pressure changes that open probe-patent defects to permit paradoxical emboli. TEE is the only intraoperative tool for detecting these abnormalities; few other tests can detect air and sourc-



es of right-to-left shunts as accurately. The task force recognizes that the use of TEE during upright neurosurgical procedures may increase the risk of vocal cord injury, although using proper technique and equipment may reduce these risks.

RECOMMENDATIONS (INDICATIONS): The detection of air emboli during cardiotomy and heart transplant operations is a category II indication for using intraoperative TEE. It also should be considered on an individual basis for patients undergoing upright neurosurgical procedures (category II).

RECOMMENDATIONS (PROFICIENCY): Anesthesiologists with basic TEE training should have an understanding of the physiologic effects of air emboli and should be technically capable of detecting air emboli intraoperatively, especially during upright neurosurgical procedures. The use of intraoperative TEE to accurately detect patent foramen ovale should be performed by, or in timely consultation with, a physician with advanced TEE training.

Mayo clinic experience

The figures below describe the intraoperative use of TEE (IOTEE) in our practice. In 15 % of cases we found new information prebypass that directly impacted the surgical management. The most common new prebypass information found was a patent foramen ovale resulting in closure in most cases. New information was found postbypass in 6 % of the cases, which resulted in further surgery and/or a change in hemodynamic management 4 % of the time. The most common postbypass finding was valvular dysfunction requiring repeat cardiopulmonary bypass in most cases to re-repair the valve or, in some cases, valve replacement.[12]

In a study that prospectively compared the IOTEE findings with the operative findings in 1918 consecutive cardiac cases, we found only 48 discordant findings. Discordant findings occurred in 2.5 % of cases and were mostly related to valve pathology. This resulted in an alteration of the planned surgical procedure in a small number of cases (0.3 %).[13]

Performing TEE in a patient with unexplained, unstable hemodynamics, cardiac arrhythmia, hypoxemia, or in the evaluation of cardiac trauma has an even greater impact. We found a cause for these unexplained physiological perturbations in 80 % of patients that resulted in an alteration of the planned surgical procedure in one quarter.[14] We concluded that emergency TEE is a feasible and safe method for assessment of unstable patients in the operating room and has a high diagnostic yield. ❐

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Cardiovascular risk factors for anesthesia

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INCREASED SURVIVAL OF CHILDREN with congenital heart disease (CHD), particularly complex CHD, has resulted in an increase in both children and adults with CHD who require anesthesia for non-cardiac surgical procedures. Pts with CHD who undergo non-cardiac surgery may be at increased anesthetic risk, particularly if their underlying condition is critical, or their physiological balance precarious. Assessment of anesthesia risk includes a full history, physical exam, and cardiovascular assessment, with attention to underlying anatomy, surgical history, and current cardiovascular function. Issues, which may need to be addressed during the conduct of anesthesia, include oxygen saturation at baseline, ventricular function, underlying cardiac rhythm, pulmonary artery pressures, and important hemodynamic abnormalities. Assessment of cardiac status includes a full understanding of the underlying disease. Patients can be categorized based on underlying physiology, ventricular function, and systemic side effects of their CV disease. CHD "anatomic" physiology can be divided into 4 basic categories: left-to-right shunt, right-to-left shunt, mixing lesions and single ventricles, and obstructive lesions. For pts in the first 3 categories the anesthetist needs to pay careful attention to the balance of systemic and pulmonary blood flow, so as not to overcirculate one at the expense of the other. For example, in pts with left-to-right shunt and a reactive or responsive pulmonary bed, the amount of pulmonary blood flow may increase dramatically with the administration of oxygen or with hyperventilation; normocapnea and room air may keep the patient in balance. The pt with unrepaired tetralogy of Fallot or with "tet-physiology" may be prone to hypercyanotic episodes, precipitated by the administration of a general anesthetic. In this situation, the administration of a systemic vasoconstrictor can promote more pulmonary blood flow. In addition, induction of cyanotic pts may take longer, and because of the intracardiac shunting, the correlation of end-tidal CO_2 to arterial CO_2 is needed. Each single ventricle pt is unique; those with a systemic right ventricle may be less able to tolerate changes associated with anesthetic, making avoidance of myocardial depressant agents critical. Systemic to pulmonary artery shunt pts depend on maintenance of systemic BP for perfusion of the pulmonary bed. Pts with left ventricular outflow obstruction may respond poorly to changes in systemic perfusion pressure and careful avoidance of such changes is necessary. ❐

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Enteraalinen ravitsemus

Maaret Castrén

SELAILTUANI VANHOJA LUENTOJANI näin aloittaneeni vuonna 1998 luentoni kertomalla, että puolet niistä potilaista, joille lääkäri on määrännyt parenteraalista ravitsemusta, on toimiva suoli. Tämän jälkeen olen jatkanut kertomalla, että jopa 40 % potilaista on aliravittuja ja että ravitsemuksen toteutus on huonoa, ongelmia ei tiedosteta eikä tunnisteta (McWhirter 1994). Luennostani on viisi vuotta. Onko mikään muuttunut?

Kudsk (2003) kirjoittaa tuoreessa artikkelissaan, että ravitsemuksen toteutus jätetään huomiotta tai unohdetaan kokonaan. Kliinikot eivät usko ravitsemuksen etuihin ja ravitsemushoito aloitetaan tehokkaasti vasta komplikaatioiden kehityttyä. Tehopotilailla tehdyssä tutkimuksessa (Heyland 2003) potilas sai vain noin 60 % tarvittavasta kalorimäärästä ja samankaltaisia tuloksia on vuosien varrella julkaistu useita. Vailla ravitsemushoitoa jäi kokonaan 39,7 % potilaista ja vain 10 % syöttö aloitetaan ensimmäisenä hoitopäivänä.

Enteraalisen ravitsemuksen toteutus voi joskus olla hankalaa. Turhia keskeytyksiä on kuitenkin paljon, jopa 67 % johtuu vanhakantaisista ajatuksista, että ravitsemus tulee keskeyttää, jos potilaalle tehdään toimenpide tai potilas on makuuasennossa. Ja kukapa päivystäjistä jaksaisi alkuyöstä laittaa paikoilleen poisluiskahtanutta nenämahaletkua (McClave 1999). Useamman vuoden ajan on tiedossa ollut, että leikkauksenkin aikana voi enteraalisesti antaa ravintoa. Käytännössä tiedän ainakin Töölön sairaalan palovammojen kohdalla näin toimitun. Jo vuonna 1918 Andersen kertoi enteraalisen ravitsemuksen eduista aloitettuna leikkauspöydällä. Vielä ei kovin monia julkaisuja asiasta ole, mutta Bengmarkin itseuivan syöttöletkun kehittäjä toteaa jatkuvan enteraalisen ravitsemuksen antavan immuunijärjestelmälle hyvän tuen komplikaatioita vastaan varsinkin korkeariskisissä leikkauksissa kuten maksan ja hai-

man alueen toimenpiteissä. Onhan immuunijärjestelmästä 80 % juuri suoliston alueella (Bengmark 2002). Syöttöletkun sijainti jejunaalisesti on osoitettu hyväksi vähentämään syöttöletkuihin liittyviä infektioriskejä ja ravitsemushoito toteutuu myös tehokkaammin eli potilas saa suuremman määrän kalori- ja proteiinitarpeestaan (Heyland 2002). Bengmarkin tyyppinen letku ui itsestään paikoilleen jopa 93 % yrityksistä eikä skopistien kallista aikaa tarvita (Mangianto 2000).

Hyvin tärkeää on muistaa, että enteraalinenkin ravitsemus tulee räätälöidä potilaan tarpeiden mukaan. Preoperatiivisesta ravitsemuksesta ei näyttäisi olevan hyötyä kuin tietyissä riskiryhmissä, kuten suusyöpä tai haiman isot leikkaukset. Elektiiviseen leikkaukseen tuleva potilas, jonka leikkaa taitava kirurgi ei siitä hyödy, ei myöskään lievästi vammautunut potilas. Ravitsemushoitoa suunniteltaessa on hyvä muistaa, että kaloritarve on noin 25 kcal/kg. Tehokkainta olisi saada REE ja antaa 110–12 % arvosta kaloreita. Proteiineja tulisi antaa 2,1 g/kg laskettuna ennen sairastumista mitatusta painosta tai sitten 1,0 g/kg päivän nolla painon mukaan. Yli 1,5 g/kg proteiineja ei vähentänyt tutkimuksessa proteiinihukkaa (Ishibashi 1998). Liuoksina kannattaa suosia standardiliuoksia. Immunonutrientteja tulee antaa jos potilas tarvitsee ravitsemusta yli viikon muistaen kuitenkin, että ne voivat olla jopa haitallisia septisille potilaille. Glutamiinista on hyötyä vain parenteraalisesti annettuna ja hyöty on lähinnä infektioiden väheneminen. Tosin tehopotilailla on viitteitä jopa kuolleisuuden laskusta. Lihavien potilaiden kohdalla on tärkeää muistaa, että heillä on aivan erilainen reaktio sairauteen kuin ei-lihavilla (BMI < 30). Rasvavarastojen käyttö estyy vammautumisen seurauksena eli emme pysty laihduttamaan heitä olemalla antamatta kaloreita. Lisäksi proteiinivarastojen mobilisaatio kiihtyy. Suositus onkin antaa heille 50 % energiasta sokerina ja proteiineja 1,5 g/kg.

Varhainen enteraalinen ravitsemus on turvallista ja hyvin siedettyä. Yhdeksän vuoden ja 650 potilaan aineistossa syöttö jouduttiin ongelmien takia lopettamaan 8,9 %, 1,7 % sai komplikaation jejunostomiastaan ja yksi potilas kuoli (Braga 2002). Se on myös kustannuksiltaan monin verroin halvempaa kuin parenteraalinen ravitsemus. ❐

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Verensokeri tehohoitopotilaalla

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HYPERGLYKEMIA ON TAVALLINEN ilmiö tehohoitopotilaalla. Siihen vaikuttavat mm. stressihormonit (glukagoni, kasvuhormoni, katekolamiinit, glukokortikoidit), jotka ovat insuliinin vastavaikuttajia sekä sytokiinit. Myös hyperalimentaatio altistaa hyperglykemialle. Akuuttitilanteessa voi kehittyä insuliiniresistenssi ja suhteellinen hypoinsulinemia[1]. Hyperglykemiaa on pidetty adaptiivisena ilmiönä mm. aivojen ja punasolujen glukoosin saannin takaamiseksi. Toisaalta hyperglykemiaa lisääviin tekijöihin kuten kasvuhormonin annosteluun tehopotilaalle liittyy lisääntynyt kuolleisuus. Usein lisäinsuliinin annostelurajana on pidetty yli 10–12 mmol/l verenglukoositasoa ja insuliinia on annosteltu boluksina ihon alle tai laskimonsisäisesti. Laskimonsisäisenä boluksena annosteltuna insuliinin puoliintumisaika on lyhyt ja huippuvaikutus saavutetaan 20–60 minuutin kuluttua, joten tasaisen verensokeritason saavuttaminen on hankalaa. Oraalisia diabeteslääkkeitä ei yleensä pidä käyttää kriittisesti sairailla potilailla, sillä mm. metformiini altistaa etenkin munuaisinsuffisienssin yhteydessä laktaattiasidoosille ja uudet glitatsoniryhmän lääkkeet vesiretentiolle ja sydämen vajaatoiminnalle.

Hyperglykemian tiukemman hoidon on osoitettu parantavan akuutin sydäninfarktin ennustetta diabeetikoilla. Malmberg ym.[2,3] osoittivat DIGAMI-tutkimuksessa, että insuliini-kalium-infuusion ja osastolla jatketun monipistosinsuliinihoidon ansiosta 1 ja 5 vuoden mortaliteetti alenivat selvästi. Ensimmäisen vuorokauden jälkeen fP-glukoosi oli intensiivihoitoryhmässä merkittävästi parempi, 9,6 ± 3,3 mmol/l, kuin tavanomaisesti (vain selvimmät hyperglykemiat hoitaen) hoidetuilla, 11,7 ± 4,1 mmol/l, mutta intensiiviryhmässäkään ei siis saavutettu normoglykemiaa valtaosalla potilaista.

Septisillä ei-diabeetikoilla hyperglykemian yleisyys on 50 %[1]. Hyperglykemia altistaa monille haitoille, jotka lisäävät sairastavuutta ja kuolleisuutta. Tavanomaisia komplikaatioita ovat mm. infektiot, etenkin sepsis sekä monielinvaurio, laihtuminen, heikkous ja tehohoitopolyneuropatia (CIP). Tuore tutkimustulos antaakin aihetta suhtautua tehohoitopotilaan verensokerin korjaamiseen totuttua aktiivisemmin[4]. Insuliini-infuusiolla aikaansaatu normoglykemia parantaa potilaiden ennustetta ja vähentää

sekä komplikaatioita että hoitokustannuksia[4]. Seuraavassa tarkastellaan kyseistä, yhtenä viime vuosikymmenten merkittävimmistä tehohoitotutkimuksista pidettyä työtä.

Leuvenin yliopistosairaala on vaikeimpien potilaiden hoitoon keskittynyt ns. tertiäärisairaala. Noin 30 % potilaista viipyy sairaalan aikuisten kirurgisella teho-osastolla yli 5 vrk ja teho-osastokuolleisuus on noin 20 %. Van den Berghe ym. tutkivat tehostetun verenglukoosin hoidon vaikutuksia tavanomaiseen hoitoon verrattuna 1548:lla tälle osastolle saapuneella potilaalla seuraavassa tutkimusasetelmassa.

Teho-osastolla potilaat satunnaistettiin kahteen ryhmään. Intensiivisen insuliinihoidon ryhmässä tavoitteena oli normoglykemia (verensokeri 4,4–6,1 mmol/l), kun taas perinteisessä hoitoryhmässä insuliini-infuusiota käytettiin vain, jos verenglukoosi ylitti 11,8 mmol/l ja tavoitteena oli verenglukoosin pitäminen välillä 10–11,1 mmol/l. Alkuvaiheessa kaikki potilaat olivat mekaanisesti ventiloituja. Kahdelle kolmesta potilaasta oli tehty sydänkirurginen toimenpide. Potilaiden keski-ikä oli 62 vuotta, BMI keskimäärin 26 kg/m^2 ja 15 %:lla oli maligniteetti. 13 %:lla potilaista oli edeltävästi diabetes ja 12 %:lla verensokeri yli 11,1 mmol/l. Ensimmäisen 24 tunnin APACHE II-pisteluku oli keskimäärin 9 ja TISS-pisteluku 43.

Potilaat saivat standardoitua ravitsemusta aluksi 8 kcal/kg/vrk glukoosi-infuusiona ja siitä portaittain viikon kuluessa lisättynä glukoosi-, proteiini- ja rasvakombinaationa ad 25 kcal/kg/vrk. Keskimääräinen energiansaanti koko tehohoitojakson aikana oli 19 kcal/kg/vrk. Enteraaliseen ravitsemukseen pyrittiin mahdollisimman pian. Hoitotulos ei kuitenkaan ollut riippuvainen siitä, saiko potilas enteraalista, parenteraalista vaiko näiden yhdistelmäravitsemusta. Insuliini annosteltiin ruiskupumpulla (50 ky lyhytvaikutteista insuliinia 50 ml:ssa Na 0,9). 39 % perinteisen hoidon ryhmästä sai insuliinia (keskimäärin 33 ky/vrk) ja 99 % intensiiviryhmästä (keskimäärin 71 ky/vrk). Intensiiviryhmässä hoito aloitettiin infuusionopeudella 2 ky/h, jos verenglukoosi oli koholla. Jos verenglukoosi ylitti 12,2 mmol/l, aloitusnopeudeksi valittiin 4 ky/h. Alkuvaiheen jälkeen energiansaantiin suhteutettu insuliinitarve väheni, sillä insuliiniresistenssin arveltiin vähentyvän. Intensiiviryhmän tehohoitojakson keskimääräinen infuusionopeus oli 3–4 ky/h. Enimmillään sallittiin jopa 50 ky/h! Valtaosa potilaista saavutti hoitotavoitteen vuorokauden kuluessa. Aamuverensokeri oli intensiiviryhmässä 5,7 ± 1,1 mmol/l ja perinteisessä ryhmässä 8,5 ± 1,8 mmol/l. Huomattavaa on, että perinteisessäkin ryhmässä saavutettiin selvästi alle 10 mmol/l:n verenglukoositaso.

Infuusioprotokollan avulla hoito toteutui ongelmitta sairaanhoitajien ja lääkäreiden toteuttamana eikä vakavia hypoglykemioita ilmennyt. Verenglukoosia seurattiin 1–4 tunnin välein verikaasunäytteen oton yhteydessä. Lyhytkestoisia hypoglykemioita ilmeni 5,2 %:lla intensiiviryhmässä ja 0,8 %:lla perinteisessä ryhmässä.

Tutkimuksen primaaripäätetapahtumana oli teho-osastokuolleisuus ja tutkimus keskeytettiin suunniteltua aiemmin selvän ryhmien välisen eron vuoksi. Erikseen tarkasteltuna kuolleisuusero ilmeni yli 5 vuorokautta hoidettujen alaryhmässä (20,2 % vs. 10,6 %, p = 0,005). Vastaavasti myös sairaalakuolleisuus aleni intensiiviryhmässä (kuva 1). Suurin kuolleisuuden aleneminen todettiin monielinvauriopotilailla, joilla todettiin septinen fokus. Intensiivihoidetuilla todettiin vähemmän veriviljelypositiivisia infektioita, pitkittyvän antibioottihoidon tarvetta, dialyysihoitoja, CIP:ia, pitkittynyttä mekaanista ventilaatiota ja pitkittynyttä tehohoitoa (kuva 2). Potilaiden hoitoisuus oli matalampi intensiivihoitoryhmässä, ja tehohoitojakson kesto väheni keskimäärin kolme vuorokautta, joten hoidon kustannusvaikutus oli edullinen.

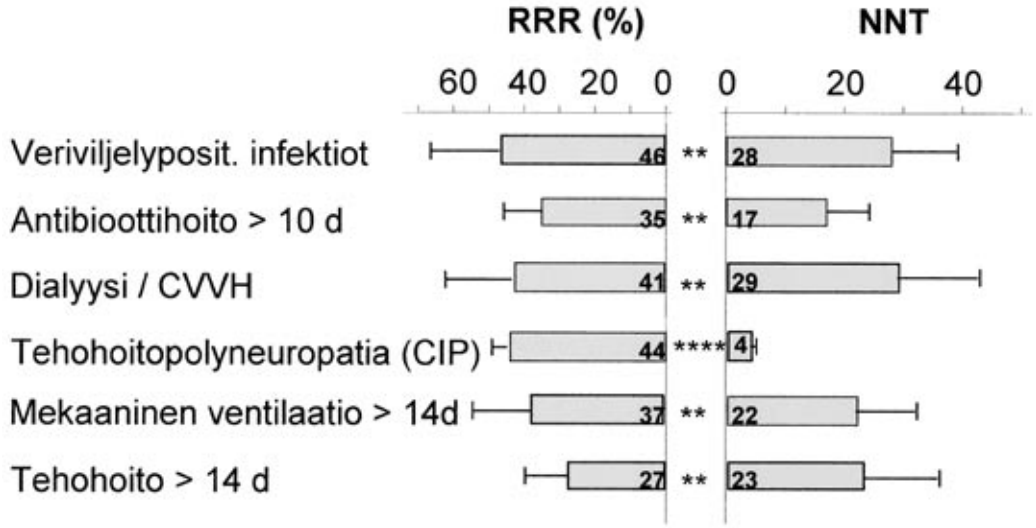
Yhteenvetona voidaan tämän tutkimuksen osalta todeta, että normoglykemian saavuttaminen portaittain nostettavalla insuliini-infuusiolla on helppoa ja onnistuu valtaosalla potilaista vuorokauden sisällä. Hoidolla voidaan ainakin kirurgisessa tehohoitoaineistossa vähentää kuolleisuutta ja kompli-

Kuva 1. Teho-osasto- ja sairaalakuolleisuus konventionaalisessa ja intensiiviryhmässä (van den Berghe 2002).

Insuliinihoito	**Perinteinen (n = 783)**	**Intensiivi (n = 765)**	**p**
Teho-osastokuolleisuus	8,0 %	4,6 %	0,005
5 päivän kuolleisuus	1,8 %	1,7 %	0,9
Kuolleisuus yli 5 vrk hoidetuilla*	20,2 %	10,6 %	0,005
Sairaalakuolleisuus	10,9 %	7,2 %	0,01
Kuolleisuus yli 5 vrk hoidetuilla*	26,3 %	16,8 %	0,01

*n = 451

Kuva 2. Sairastavuuden suhteellinen väheneminen (RRR) ja number needed to treat (NNT) intensiivihoitoryhmässä (van den Berghe 2002).



(error bars: 95 % luottamusväli)

RRR = suhteellinen riskin alenema, NNT = number needed to treat

kaatioita. Lisäksi hoito on kustannustehokasta. Suurin hyöty saavutetaan pitempää tehohoitoa vaativilla potilailla, jotka luonnollisesti ovat alttiimpia tehohoidon komplikaatioille. Monilla teho-osastoilla insuliini-infuusiohoito on jo nyt tuttua, joten tarvittaisiin enää uusien tavoitteiden omaksumista. Hoitoprotokollan avulla voidaan parantaa hoidon toteutumista ja turvallisuutta. Jo nyt tuntuisi järkeenkäyvältä hoitaa tehohoitopotilas normoglykeemiseksi eli plasman glukoosi neljän ja kuuden mmol/l välille (viitealue 4,0–6,1 mmol/l). Suurella mielenkiinnolla voidaan odottaa tämän merkittävän tutkimuksen tulosten vahvistamista myös muissa tehohoitopotilasaineistoissa sekä postoperatiivisilla potilailla tehon ulkopuolella. ❐

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Anesthesia for undiagnosed muscle and metabolic disorders

Robert J. Friedhoff

MITOCHONDRIA ARE THE PRINCIPAL source of energy metabolism within the cell. It is here where we find the enzymes for the Krebs cycle, fatty acid synthesis, beta and oxidative phosphorylation. The enzymes for these processes are processed after transcription and translation of genes within the DNA of the cell nucleus. The enzymes are then formed in the cytoplasm and transported to the mitochondria. Uniquely some of the enzymes for oxidative phosphorylation are synthesized within the mitochondria. Mutations in mitochondria genomes (from mitochondrial DNA exposed to free radicals formed in the electron transport chain) lack DNA repair mechanisms can produce energy failure in those cells that maintain a high metabolic rate such as muscle and nerve. Mitochondria genomes are inherited solely from the egg. Mutations are thus heteroplasmic. The cell itself will be affected only when a critical threshold of mutant DNA is present so that oxidative phosphorylation is disrupted. Typical symptoms of mitochondrial failure include myopathy, pigmentary retinopathy, sensorineural hearing loss, stroke, metabolic encephalopathy and seizures. Mitochondrial genome defects increase with age and

can be caused by autosomal mutations or exposure to toxins. Patients with these primary disorders of metabolism are prone to disruption at times of high metabolic stress.

Mitochondrial cytopathies are an increasingly recognized cause of human illness. They comprise a group of uncommon disorders with variable clinical presentations with multiple systems involvement. First reported in 1962 by Luft, they have very diverse manifestations. All have a lack of adequate energy to meet the cells needs. They vary considerably from disease to disease and from case to case. Post mitotic tissues at birth (non-dividing cells) such as the brain, muscles, nerves, retina and kidneys are more vulnerable because of there high demand for energy and these diseased cells cannot be replaced. Symptoms are usually vague, such as fatigue or weakness, muscle pain or cramping, SOB. No single test is diagnostic. Laboratory values often, but not always show elevation of lactic acid and an increased lactic acid / pyruvate ratio. Glucose levels can be variable. Muscle biopsy shows "ragged red fibers" that is most pronounced with a Gomori trichrome stain. Treatment is symptomatic, striving to maintain homeostasis and to avoid stress and infection. Patients are often given vitamins and co-factors such as Coenzyme Q_{10}, L-carnitine, creatinine phosphate, B vitamins and antioxidants.

Providing anesthesia for patients already diagnosed with or to diagnose Mitochondrial myopathy can be challenging. Since this disease has such a high variability, the anesthetic plan should be based on the individual patient. Several case reports have recently been published in the literature. Patients often have hypotonia, bulbar dysfunction and poor pulmonary function. They are at increased risk for post-op pulmonary complications such as aspiration (this is especially seen in patients with Leigh syndrome). Their response and sensitivity to muscle relaxants is often unpredictable and prolonged. The possibility of a myopathy can put them at risk for developing malignant hyperthermia. Any additional stress can cause deteriation and worsening of symptoms. Patients with Kearns-Sayre syndrome are at high risk for developing bradycardia or cardiac conduction abnormalities. Halothane should be avoided in patients with a history of conduction defects. It is advised to avoid drugs that may depress cardiac function. Careful attention to preventing infection and maintaining glucose: homeostasis should be sought. Prolonged use of propofol should be avoided. Lactated ringers solution is contraindicated in these patients due to elevating the lactic acid level. Since general anesthetics can inhibit mitochondrial respiration in skeletal muscle (this is not due to a direct effect on complexes of electron transport or uncoupling of oxidative phosphorylation), regional anesthesia is preferred over general anesthesia when providing anesthesia for muscle biopsy and diagnosis.

Patients diagnosed with Duchenne's muscular dystrophy are at increased risk of malignant hyperthermia and developing succinylcholine induced hyperkalemic cardiac arrest. Mean age of diagnosis of Duchenne's muscular dystrophy is 5.2 years with virtually 100 % diagnosed by 9 years of age. They are predominantly male. These patients suffer from not only a myopathy, but also commonly have reduced cardiac function, increased incidence of cardiac arrythmias and reduced pulmonary function that continues to deteriorate with increasing age despite surgery. Non-triggering anesthetics should be provided when these patients come for surgery. ❐

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Uutta lasten perioperatiivisesta paastosta

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AIKAISEMMIN OLETETTIIN PITKITTYNEEN paaston varmistavan mahan tyhjenemisen ja suojaavan siten potilasta aspiraatioriskiltä. Toisaalta anestesian jälkeen erityisesti PÄIKI-potilaita kehotettiin juomaan ennen kotiutusta sillä ajatuksella, että juominen vähentäisi oksentelua ja dehydraation vaaraa. Viimeaikaiset tutkimukset ovat muuttaneet käsityksemme kummastakin oletuksesta, ja uudet liberaalim-

mat paasto-ohjeet ovat nopeasti levinneet rutiinikäytännöksi lapsipotilaiden anestesiatoiminnassa. Lyhyempi paastoaika on paitsi inhimillisempää lapselle ja vanhemmille, myös vähentää dehydraation ja hypoglykemian riskiä. Retrospektiivisissä analyyseissa lapsipotilaan aspiraatiosyndrooman on osoitettu olevan harvinainen komplikaatio (1:10 000–10,2: 10 000), johon lähes aina liittyy jokin erityisriskitekijä (päivystysleikkaus, ruokatorven alasfinkterin alentunut tonus tai GER, gastrointestinaalikanavan tukos tai ahtauma, palleatyrä, obesiteetti, pitkittynyt intubaatio ym.).

Eri-ikäisillä lapsilla tehdyt mahanesteen volyymin mittaukset ovat osoittaneet, että kirkas neste tyhjenee mahalaukusta exponentiaalisella nopeudella (puoliintumisaika 10–20 min). Kiinteän ruokamassan osalta tyhjenemiskäyrä on lineaarinen, paljon hitaampi ja vaihtelee huomattavasti eri yksilöiden välillä. Monet eri tutkimukset ovat yhteneväisesti näyttäneet toteen, että pitkä (yli 8 t) totaalipaasto ei vähennä mahanesteen volyymia verrattuna 2–3 tuntia kirkkaan nesteen nauttimisesta mitattuihin arvoihin. Mahanesteen pH voi jopa hapantua paaston kestäessä. Täten kirkkaan nesteen osalta paasto-ajaksi on suositeltu 2 tuntia. Kiinteän ruuan osalta entinen 6–8 tunnin paastosuositus on edelleen perusteltu.

Suurta hämmennystä on aiheuttanut kysymys rintamaidon, lehmänmaidon ja äidinmaidon vastikkeen luokittelusta kirkkaaksi nesteeksi tai ruuaksi. Tulokset mahan tyhjenemistä mittaavista tutkimuksista ovat antaneet hieman eriäviä tuloksia, joiden perusteella on päädytty varovaiseen konsensukseen siten, että suositeltava paastoaika rintamaidon jälkeen on 4 t ja lehmänmaidon sekä vastikkeen jälkeen 6 t. Uusimmat tutkimukset tosin viittaavat siihen, että 4 t paasto riittäisi myös vastikkeen jälkeen.

Monien eri tutkimusten mukaan nopeasti anestesian jälkeen tapahtuva lapsipotilaan juottaminen lisää pahoinvoinnin ja oksentelun insidenssiä. Tästä syystä ennen kotiutusta vaaditusta ”pakkojuomisesta” on syytä luopua, ja juotavaa tulee antaa vain niille lapsille, jotka sitä haluavat. ❐

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The surgical treatment of childhood strabismus

Brian G. Mohney

Purpose: Strabismus is a disorder of ocular alignment that affects 5 % of the pediatric population and commonly requires surgical intervention. The purpose of this paper is to provide an overview of the surgical management of childhood strabismus.

Methods: An overview of the various forms of childhood strabismus, the indications, timing, and goals of surgery, as well as the different surgical techniques including adjustable sutures, and postoperative complications will be presented.

Results: Childhood strabismus is a common disorder that requires surgical correction in approximately two-thirds of patients. Surgery is recommended soon after the deviation develops to improve alignment, promote stereoacuity, and enhance self-esteem and coordination. Weakening or strengthening of the extraocular muscles are the most common surgical procedures. Eighty to 85 % of patients will be successfully treated with 1 surgical procedure while 95 % will successfully respond to 2 procedures.

Conclusions: Surgical intervention continues to be an important modality of treatment for children with strabismus. A careful preoperative assessment and thorough understanding of the various surgical techniques will lead to a successful result in the majority of patients. ❐

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Keuhkojen digitaaliakustiikka

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KEUHKOJEN DIGITAALIAKUSTIIKALLA TARKOITETAAN keuhkojen akustisten ominaisuuksien mittaamista ja rekisteröintiä. Perusmenetelmiä on kaksi; ne eroavat toisistaan käytettävän signaalin alkuperän suhteen. Aktiivinen digitaaliakustiikka rekisteröi luonnollisia hengitysääniä ja on verrattavissa tavanomaiseen keuhkojen auskultaatioon, paitsi että käytössä on signaalin sähköinen rekisteröinti, käsittely, muokkaus ja analyysi. Passiivinen digitaaliakustiikka käyttää ulkoista, keinotekoista signaalia, jonka muuttuminen hengityselimistössä on menetelmän informaatioarvon perusta. Edellinen on teknisesti helpompi toteuttaa, vähimmillään tarvitaan vain mikrofoni, perusäänikortilla varustettu kannettava tietokone ja äänisignaalin editointiohjelmisto. Passiivinen digitaaliakustiikka edellyttää lisäksi laitteistoa, jolla signaali muodostetaan, vahvistetaan ja syötetään hengitysteihin. Keinotekoista signaalia on toisaalta helpompi muokata, eikä se riipu hengityselimistön toiminnasta sinänsä. Seuraavassa lyhyt katsaus molempiin perusmenetelmiin.

Aktiivinen digitaaliakustiikka

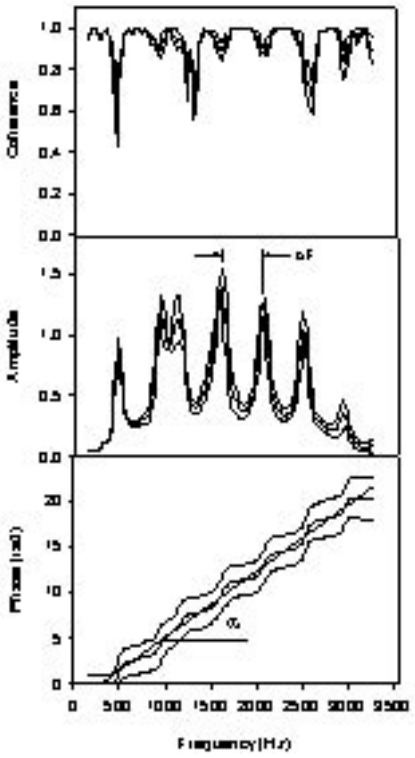
Fysiologian perusteella luonnollisten hengitysäänten kuuntelun pitäisi olla hyödyllinen keuhkovaurion seurantamenetelmä. Ploysongsang työtovereineen rekisteröi hengitysäänten tehojakauman viideltä koiralta, joista kahdella oli interstitielli ja kolmella alveolaarinen korkeapaineinen keuhkopöhö.[1] He totesivat jakauman siirtyvän kohti korkeita taajuuksia tavalla, joka oli verrannollinen ylinesteytyksen asteeseen ja sekä keuhkomekaniikan että kaasujenvaihdon huononemiseen. Vaikka keuhkojen nestemäärän lisääntyminen ja kaasupitoisuuden väheneminen aiheuttaisivatkin edelläkuvatun kaltaisia muutoksia, ne eivät todennäköisesti ole tarpeeksi suuria tai spesifejä jotta niistä saataisiin vauriota paikantavaa tietoa. Pienten ilmateiden aukeaminen aiheuttaa kuitenkin epänormaalien keuhkoäänten, rahinoiden, muodostumista, jotka rahinat voidaan rekisteröidä ja laskea. Ilmateiden aukeamisen aiheuttamien äänten toistuminen hengitysvaiheen mukana edellyttää sitä, että keuhkoissa on jaksoittaisesti kasaan painuvia ja aukeavia alueita. Tietyltä keuhkon alueelta kuuluvien rahinoiden kvantitointi saattaa olla käyttökelpoinen menetelmä keuhkojen paikallisen kaasutilavuuden seurannassa. Keuhkojen auskultaatiota stetoskoopilla käytetään päivittäin teho-osastoilla keuhkopatologian paikallistamiseen. Stetoskoopilla on kuitenkin huomattavia akustisia rajoituksia ja sillä kuullun informaation käyttöarvo riippuu huomattavasti kuuntelijasta. Sitäpaitsi kokenutkaan kliinikko ei kykene erottamaan ja laskemaan rahinoiden kaltaisia nopeasti toistuvia äänitapahtumia. Rahinoilla on kuitenkin tyypillinen ja helposti todettava aaltomuoto, jonka perusteella ne voidaan rekisteröidä automaattisesti ja laskea tarkkaan. Useita validoituja rahina-algoritmeja on kehitetty ja niitä on käytetty jo kohtalaisen pitkään kroonisten keuhkosairauksien diagnostiikassa ja seurannassa.[2,3]

Passiivinen digitaaliakustiikka

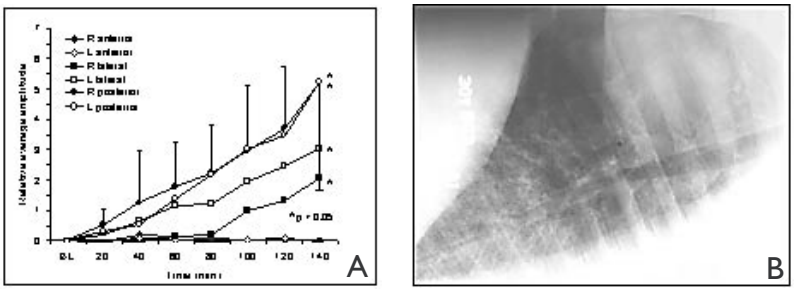
Keuhkot ja rintakehä sisältävät kaasua, nestettä ja kiinteää kudosta vaihtelevissa määrin; näiden rakenneosien suhteellinen osuus vaihtelee myös sairauden aiheuttamien muutosten seurauksena. Koska äänisignaali johtuu eri tavalla kaasun, nesteen ja kiinteän aineen läpi, äänienergiaa voidaan teoriassa käyttää patologisten keuhkomuutosten diagnosointiin ja seuraamiseen tarkastelemalla tunnetun äänisignaalin suodattumista sen kulkiessa hengityselimistön läpi. Perusajatus on siis se, että rintakehän pinnalta rekisteröity äänisignaali (S_n) on hengitysteihin syötetyn, tunnetun äänisignaalin (S_0) funktio (ns. siirtofunktio f):

$$S_n = f(S_0)$$

Signaalit S_0 ja S_n on rekisteröitävä samanaikaisesti siten, että niitä mittaavien antureiden välille jää mahdollisimman vähän tutkimuksen kannalta tarpeettomia tai häiritseviä elementtejä. Siirtofunktio on spesifi ja erilainen kullekin $S_0 – S_n$ rekisteröintiparille ja edustaa tiettyä, antureiden sijainnin määräämää signaalin kulkureittiä hengityselimistön läpi. Sen muutokset kuvaavat äänisignaalin suodattumisen muutoksia esim. keuhkovaurion kehittyessä. Siirtofunktion f ominaisuuksia luonnehtivia muuttujia on tavallisesti kolme: koherenssi, vaihe, ja amplitudi. Nämä esitetään yleensä graafisesti taajuusjakaumana (kuva 1). Koherenssi on validointimuuttuja, joka kuvaa sitä, miten hyvin signaalin S_0 amplitudivaihtelut heijastuvat signaalissa S_n kullakin taajuudella. Jos koherenssi jollakin taajuudella on 1, em. signaalien amplitudivaihtelut vastaavat täysin toisiaan ja S_n:n voidaan katsoa olevan S_0:n 'variantti'. Jos koherenssi on matala, S_n ei todennäköisesti ole peräisin S_0:sta vaan edustaa jotakin muuta signaalia. Vaihe tietyllä taajuudella ilmoittaa sen, missä kulmassa radiaaneina mitattuna tätä taajuutta vastaava ääniaalto on kun se saapuu mittauskohtaan n. Koska ly-



Kuva 1. Intubaatioputken siirtofunktio äänisignaalille. Kuvaajat edustavat neljän leikkaamattoman 8,0 mm putken keskiarvoja (paksu viiva) ja 95 % luottamusväliä (ohuet viivat). Koherenssi (yläpaneli) on yleensä hyvä, mutta heikkenee niillä taajuuksilla, joilla syötetty ja putken päästä takaisin heijastunut signaali pyrkivät kumoamaan toisensa. Näillä taajuuksilla amplitudi on matala (antiresonanssi). Vaiheen taajuusjakauman (alapaneli) kaltevuus riippuu putken pituudesta ja äänen nopeudesta.



Kuva 2. (A) Keskimääräinen (n = 5) keuhkojen ja rintakehän äänisignaalin siirtofunktion amplitudi verrattuna perustasoon (BL), mitattuna kolmelta rintakehän alueelta molemmin puolin äkillisen öljyhapon aiheuttaman keuhkovaurion kehittymisen aikana. (B) Yhden koe-eläimen translateraali-keuhkokuva, jossa painovoiman mukainen keuhkovaurion jakautuminen vastaa hyvin todettuja akustisia muutoksia.

hyen (korkeataajuuksisen) ääniaallon kulma muuttuu kuljetun matkan mukana nopeammin, vaihekulma kasvaa taajuuden mukana, kun matka on vakio. Vaiheen taajuusjakauma on siksi suora, jonka kulmakerroin riippuu signaalin kulkeman matkan pituudesta ja äänen nopeudesta. Amplitudi on S_n:n suhteellinen äänenvoimakkuus S_0:aan verrattuna. Se on usein erilainen eri taajuuksilla; S_n:n amplitudi voi olla suurempi kuin S_0:n jos järjestelmässä on resonansseja (kuva 1). Keuhkojen siirtofunktion osalta tämä kuitenkin on harvinaista, koska rintakehän sisäpinta muodostaa kohtalaisen voimakkaan akustisen rajapinnan, jossa huomattava osa äänienergiasta heijastuu takaisin, eikä johdu ihon pinnalla olevalle anturille.

Terveillä koehenkilöillä suoritettujen tutkimusten perusteella äänisignaali kulkee suuontelosta rintakehän pinnalle 1,5–5 millisekunnissa, mikä vastaa nopeutta 60–80 m/s – siis huomattavasti äänen ilmanopeutta (350 m/s) hitaammin. Taajuuden kasvaessa äänen siirtymisnopeus vähenee asteittain. Äänen kulkureittiä hengitysteissä ei tunneta tarkkaan, mutta matalien taajuuksien uskotaan poistuvan keuhkoputkien ilmatilasta ja siirtyvän kiinteitä kudoksia pitkin; korkeat taajuudet todennäköisesti pysyvät ilmateissä pitempään ja kulkeutuvat keuhkojen ääriosiin asti. Baughman ja Loudon tutkivat koehenkilön muodostaman äänen taajuussiirtymiä sen johtuessa rintakehän pinnalle ja havaitsivat ”i”-äänteen menettävän korkeataajuuksisia osioita kulkeutuessaan ilmattoman keuhkon läpi, jolloin se alkaa muistuttaa ”a”-äännettä.[4] Vastaavanlaista auskultaatiotekniikkaa olivat kliinikot jo aikaisemmin käyttäneet atelektaasien paikantamiseksi.

Passiivista keuhkoakustiikan käytöstä äkillisen keuhkovaurion toteamiseen on toistaiseksi julkaistu vain muutama kokeellinen työ. Donnerberg työtovereineen seurasi äänisignaalin voimakkuuden suhteellista muutosta koirilla joille aiheutettiin kokeellinen hydrostaattisen keuhkopöhö.[5] Keuhkovaurion vaikeusasteen ja äänen siirtofunktion välillä oli suora riippuvuus, jonka perusteella keuhkopöhön vaikeusaste voitiin kohtalaisen tarkasti arvioida. Omissa ARDS eläinmallilla suoritetuissa tutkimuksissamme olemme todenneet, että äänisignaalin siirtofunktion amplitudi kasvaa voimakkaasti keuhkovaurion kehittymisen aikana. Muutos on suurin niillä keuhkon alueilla joilla vaurio on pahin (kuva 2).[6] Vaikuttaa siis siltä, että keuhkovaurion asteen seuraaminen ja itse vaurion paikantaminen noninvasiivisin akustisin keinoin on mahdollista.

Leikkaussaleissa ja teho-osastoilla suurin osa potilaan tilan seurantaan liittyvästä tiedosta saadaan ja rekisteröidään sähköisesti. Keuhkojen auskultaatio on huomattava poikkeus: siinä käytettävä metodiikka ja instrumentointi on muuttunut noin 180 vuoden aikana vain hyvin vähän. Hengityselimistön tilan seurantaan voidaan käyttää sekä passiivisia että aktiivisia akustiikkaan perustuvia, sähköisiä menetelmiä, joista molemmista on odotettavissa tutkimustuloksia ja mahdollisesti uusia kliinisiä sovellutuksia lähiaikoina. ❐

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Neurocognitive disorders following cardiopulmonary bypass

Gregory J Schears

Introduction

The current application of cardiopulmonary bypass allows for the safe repair of nearly all forms of congenital heart disease, valve replacement, and revascularization. Over the last 10–15 years the surgical mortality has undergone a dramatic reduction. With improvement in survival more attention has been paid to reducing the incidence of adverse neurologic sequelae. This presentation will highlight some of the observations made over the last 10–15 years and discuss some of the directions for research in the future.

Etiology

Potential causes of neurologic injury related to cardiac surgery are many. It is generally agreed that the two major causative factors for neuropsychological dysfunction are global brain hypoperfusion and cerebral emboli. It is important to recognize that they are interrelated such that by increasing cerebral perfusion, one would potentially deliver more emboli. Other factors such as the inflammatory response, patient predisposing factors, and cardiopulmonary bypass time play a role but to a lesser degree. Particulate emboli arising from atherosclerotic material in the aorta may be a predominant cause of embolic stroke seen in adults from cardiopulmonary bypass. These are referred to a macroemboli. Air and fat emboli introduced into the circuit are generally categorized as microemboli. The rate of perioperative strokes have dramatically reduced over the last several decades due to increasing attention to some of these known causes.

Neurocognitive injury

Neurocognitive or neuropsychological injury deals with the more subtle injury that can occur and requires some sort of formal testing ideally pre and postoperatively. Until relatively recently there has been no effort to standardize this testing so that inter-institutional comparisons could be made. These tests include assessment for memory disorders, speed of processing deficits, and motor skill abnormalities. Concurrent assessment of depression and anxiety are always necessary as they can impact test performance.

In 1995 a consensus statement was initiated to standardize assessment and agree upon definitions. Level 1 injury including stroke, coma, TIA, and transient defects are to be used by all investigators reporting neurologic outcomes for consistency in the Society of Thoracic Surgery database. Level 2 disorders provide for more comprehensive evaluation with standardization of the neuropsychological assessment. The high numbers of patients with neurocognitive decline have been known for some time. Shaw in 1985 reported a 30–79 % rate of cognitive decline in a group of 312 patients. A recent New England Journal of Medicine article (volume 344(6): 395–402, 2001) adds new insight into the issue of neurocognitive dysfunction in that it associated those patients with initial impairment documented following discharge were more likely to have significant impairment at five years post surgery. Conventional wisdom had been that the initial impairment continued to improve and was not thought to be an issue for the future.

Neuroprotection

In an attempt to reduce the amount of embolization occurring during cardiopulmonary bypass, a number of techniques have developed including the use of epi-aortic scanning, Y-grafting of radial arteries off the internal mammary and the avoidance of cardiopulmonary bypass completely using beating heart strategies for coronary revascularization.

These strategies and studies associated with them have been able to demonstrate some reductions in neurocognitive injury though it is clearly not the full answer.

Perfusion pressure

During cardiopulmonary bypass cerebral blood flow is determined by the complex interactions of factors such mean arterial pressure, pump flow rate, cerebral metabolic rate, blood gas management strategy, and hemoglobin level. The common practice of maintaining perfusion pressures at 50 mmHg (for adults) is based on the observation that most patients seem to tolerate it. Some studies suggest that maintaining means greater than 50 mmHg significantly reduce the incidence of neurologic complications associated with cardiopulmonary bypass and help to reduce intensive care and hospital stays.

Brain temperature

Hypothermia remains the main form of neuroprotection for cardiopulmonary bypass. With decrease in temperature the tight coupling between cerebral metabolic rate and cerebral blood flow is gradually lost such that cerebral blood flow exceeds cerebral metabolic rate and thus provides a margin of protection. It has been shown that at lower perfusion temperatures that autoregulation is lost and flow becomes pressure dependent. The rate of cooling and pH regulation strategy can impact uniformity of cerebral cooling and hence, neuroprotection.

Similarly, rate and degree of rewarming can dramatically impact the degree of neurologic injury associated with ischemia. Several studies have suggested that a subgroup of patients who otherwise appear totally normal, have evidence of inadequate oxygen delivery for their metabolic demand post bypass.

Summary

There have been dramatic reductions in mortality and morbidity associated with cardiac surgery and cardiopulmonary bypass. We have identified a number of factors that we believe to be causal for neurologic injury and continue to try to find ways to reduce their influence. Much additional work is needed to better understand those factors that impair and improve the matching of cerebral perfusion, oxygen delivery, and metabolic demand. ❐

Gregory J Schears, MD.

The shared airway in pediatrics

Dana Thompson and Randall Flick

ABNORMALITIES OF THE AIRWAY PRESENT an extraordinary challenge to all those who care for children in the operating room setting. The child with a difficult or potentially difficult airway presents with a set of problems and solutions that are for the most part completely different from that seen in the adult patient. However, as in adult airway abnormalities it is particularly important for the surgeon and anesthesiologist to work in close communication whenever the airway must be shared.

This discussion is intended to provide an overview of some of the common (and not so common problems) encountered in the anesthetic care of the pediatric patient with an airway anomaly. Although some of the problems presented are quite rare hopefully they will be nonetheless interesting, entertaining and more importantly offer some insight into the management of the difficult airway in pediatrics. I have tried to include a variety of patients with abnormalities of both the upper and lower airway occurring in children ranging in age from neonates to teenagers. The setting is not always the operating room as we are often called on to provide assistance with airway management in a variety of setting including the delivery room, the intensive care units, the emergency room, and the radiology and endoscopy suites.

Rather than describe each patient for you here I have chosen to maintain the patients as unknowns. This, I hope, will make for a more entertaining discussion. The patients will be presented in the following format:

1. Brief history with photo or video clip.
2. Question with addition photos or video
3. Answer to the question and information about the condition or problem.

The etiologies of airway abnormalities in children are almost infinite in number and include congenital malformations of the airway (Pierre-Robin), metabolic abnormalities affecting the airway (Hurlers Syndrome), infections (laryngeal papillomatosis), traumatic injuries to the airway (iatrogenic tracheal rupture), malignant airway involvement (lymphoma), foreign bodies of the airway and many others.

The types of airway abnormalities encountered vary markedly with age. Obviously most, but not all, of the congenital anomalies appear in the neonatal

period. For example, malacia of the larynx, trachea or bronchi virtually always presents in the neonatal period and for the most part resolves by the end of the first year. However, occasionally, congenital malformations worsen with age. The best example of this may be subglottic hemangioma. These lesions are asymptomatic at birth but worsen as they enlarge during the first year or so. Each age group has problems that are typical such as foreign bodies of the aero-esophageal tract that occur most commonly in toddlers and anterior mediastinal masses that are most typical in adolescents with lymphoma.

Emphasis will be on the anatomy, presentation, and management, both surgical and anesthetic, of each of the conditions. Following the discussion I will make available copies of the slides for those who wish them. I have also included a list of references that I found to be useful in the preparation of this talk and in the care of these and many other children with airway abnormalities. ❐

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Dana Thompson, MD and Randall Flick, MD, MPH

Prevention of adverse effects of preoperative fasting

Anders Thorell

INSULIN RESISTANCE DEVELOPS as a response to virtually all types of surgical stress. There is an increasing body of evidence that suggests that insulin resistance in surgical stress is not beneficial for outcome. A recent large study in intensive care patients showed that aggressive treatment of insulin resistance using iv insulin reduced mortality and morbidity substantially. Similarly, in burn patients, intensive insulin and glucose treatment has been shown to improve nitrogen economy and enhance skin graft healing. In surgical patients insulin resistance has been characterized in some detail and has been shown to have many similarities with metabolic changes seen in patients with type 2 diabetes. This may be important since insulin resistance has been shown to be one independent factor that influences length of stay. When patients about to undergo elective surgery have been treated by glucose iv or a carbohydrate rich drink instead of overnight fasting, insulin resistance was reduced by about half. A small meta analysis showed that when postoperative insulin resistance was reduced by preoperative carbohydrates, length of hospital stay was shortened. Overnight iv glucose in high doses improved postoperative nitrogen economy. This type of treatment has also repeatedly been shown to reduce cardiac complications after open heart surgery. Furthermore, if the carbohydrates are given as a drink preoperatively, preoperative thirst, hunger and anxiety is markedly reduced. In summary, preventing or treating insulin resistance in surgical stress influences outcome. Fasting over night is not an optimal way to prepare patients for elective surgery. Instead, preoperative carbohydrates have clinical benefits. ❐

Anders Thorell, docent

Pediatric postoperative pain management

Robert T. Wilder

ACUTE PAIN MANAGEMENT must first begin with pain assessment. For pediatric patients, this is more complex that with adults as developmentally appropriate pain scales are required. At least three scales will be needed. First is a behavioral and or physiologic scale to use for patients who are unable to give any self-report on their pain. This includes infants and toddlers up to about three years of age as well as severely developmentally delayed patients. Potential examples include the Facial Action Coding Scale[1], the Observational Pain Scale[2] or FLACC[3]. Examples of the OPS and FLACC are appended. Children in the 3 to 7 years age range need a scale with few points (one point per year of age has been suggested.) A common strategy is to use a faces scale, either pho-

tographs or drawn pictures. Examples are appended. Of these the scale best verified for use in children is the Bieri faces scale[4]. This is anchored with a neutral face and the no pain end since lack of pain does not necessarily translate to happy. The drawings of increasing pain were adapted from responses of children who were asked whether a face looked like someone in pain. The most severe end does not show tears, as for some patients tears are not socially acceptable. Interestingly, the facial features used in this scale are the same as those in the facial action coding scale. Most children seven years of age or older are able to use a variant of the visual analog scale. A popular example is the Chromatographic Visual Analog Pain Scale or Pain Slide Rule (picture appended).

The primary classes of medications used for acute pain management include NSAIDS, including acetaminophen/paracetamol and the COX2 inhibitors, opioids, and regional anesthetics. More recently, low doses of ketamine have been used to improve overall analgesia as have dextromethorphan and clonidine.

The problem is to minimize medication side effects while maximizing analgesia. There are several methods to do this. One is appropriate dosing. In general this means using small doses frequently to minimize peak concentrations that would tend to promote increased side effects. Patient controlled analgesia (PCA) is a good example of this. By allowing the patient access to self-dosing as soon as needed, PCA allows us to keep the delivered dose small, e.g., 20 microg/kg of morphine. A continuous infusion also works well in this regard, but it does not allow rapid changes in blood levels in response to changes in pain. Finally, methadone, by virtue of its long half-life, allows small incremental doses as intervals that are short in relation to the half-life. In-

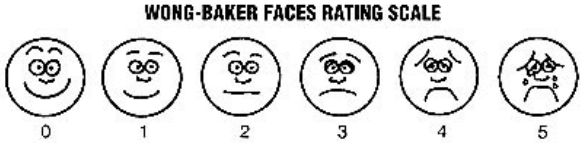
Categories	Scoring		
	0	1	2
Face	No particular expression or smile	Occasional grimace or frown; withdrawn, disinterested	Frequent to constant frown, clenched jaw, quivering chin
Legs	Normal position or relaxed	Uneasy, restless, tense	Kicking or legs drawn up
Activity	Lying quietly, normal position, moves easily	Squirming, shifting back and forth, tense	Arched, rigid, or jerking
Cry	No cry (awake or asleep)	Moans or whimpers, occasional complaint	Crying steadily, screams or sobs; frequent complaints
Consolability	Content, relaxed	Reassured by occasional touching, hugging, or being talked to; distractable	Difficult to console or comfort
Each of the five categories is scored from 0–2, resulting in a total score between 0 and 10.			

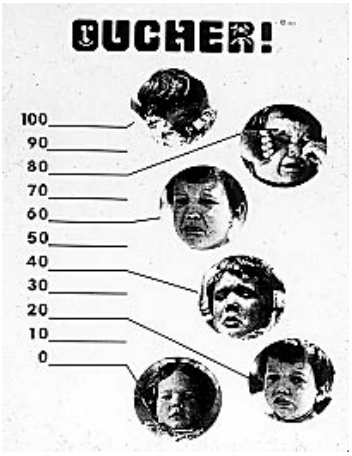
Reprinted with permission: Merkel SI, et al. The FLACC: a behavioral scale for scoring postoperative pain in young children. Pediatr Nurs 1997;23(3):293-7. The FLACC scale was developed by Sandra Merkel, MS, RN, Terri Voepel-Lewis, MS, RN, and Shobha Malviya, MD, at C. S. Mott Children's Hospital, University of Michigan Health System, Ann Arbor, MI.

Observational Pain Scale (Broadman)

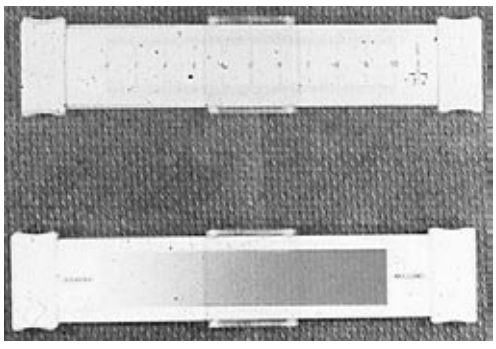
Observation	0	1	2
BP	<120% preop	120-130% preop	>130% preop
Crying	Not	Responds to TLC	Not consolable
Movement	None	Restless	Thrashing
Agitation	Calm	Mild	Hysterical
Verbal Evaluation	States "No pain"	Mild pain	Moderate or severe

Bieri Faces Scale





Chromatographic Visual Analog Pain Scale (Pain Slide Rule)



tramuscular injections are the worst possible choice. Large doses are given to maximize the interval between injections. This increases side effects immediately after the injection. Also children hate shots and will under-report pain to avoid them. This leads to complications from untreated pain.

A second possibility is to use medications with fewer potential side effects. As an example, the COX2 inhibitors decrease gastric irritation and bleeding relative to traditional NSAIDs. They have no benefit in terms of renal and cardiac toxicity, however. They are also very slow in onset, so need to be started preoperatively if they are to be effective for postoperative analgesia.

The other major strategy is to use combinations of different classes of medications. Drug combinations provide additive or synergistic analgesia. This allows lower dosing of each individual medication, again minimizing side effects. Examples include combining regional anesthetics with clonidine, opioids or both for regional blocks, or combining an NSAID, e.g., ketorolac or acetaminophen, with opioids for systemic analgesia. Clonidine and dextromethorphan given preoperatively have been shown improve analgesia overall as has low dose ketamine used in conjunction with other pain regimens. ❐

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Mitä uutta horroskoopista?

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ANESTESIATILAA ON TOTUTTU arvioimaan potilaan liikkumisen, sydämen syketaajuuden, verenpaineen ym. "anestesian kliinisten merkkien" perusteella. On osoitettu, että mikään näistä merkeistä ei kuvaa potilaan tajuttomuuden astetta, ja anestesian hypnoottisen komponentin mittaaminen onkin ollut viimeaikaisen tuotekehittelyn suuri haaste. Jotta hypnoottista komponenttia voidaan mielekkäästi mitata, täytyy aivosähkökäyrästä (EEG) eristää piirteitä, jotka liittyvät lääkkeellä aikaansaatuun tajuttomuuteen.

Viimeksi kuluneiden kymmenen vuoden aikana markkinoille on tullut useita anestesian hypnoottisen komponentin mittareita. Tunnetuin näistä on bispektraali-indeksi, BIS, jossa anestesian "syvyyttä" mitataan EEG:sta ja se esitetään asteikolla 0–100. Tällä asteikolla 90–100 vastaa hereilläoloa, 40–60 mielekästä kirurgista anestesiasyvyyttä, ja 0 vastaa "suora-viiva -EEG:aa". Muitakin anestesian hypnoottisen komponentin mittareita on sittemmin kehitetty sekä EEG:n että kuuloherätevasteiden pohjalle. Yhteistä näille kaikille on, että mitattavasta signaalista eristetään yksittäinen tunnusluku nollan ja 100 välillä; suuret luvut kuvaavat hereilläoloa ja pienet luvut tajuttomuutta.

Entropia on alun perin termodynamiikassa käytetty mitta, jolla kuvataan tutkittavan systeemin epäjärjestyksen määrää. Sitä on viime vuosina sovellettu mm. anestesian aikaisen EEG:n tutkimukseen. Korkea entropialuku (EEG:n suuri "epäjärjestys") kuvaa kevyttä anestesiaa tai hereillä oloa. Anestesian syvetessä EEG:n järjestyksen määrä kasvaa ja entropialuku pienenee.

Kotimainen laitevalmistaja (Instrumentarium OYJ/Datex-Ohmeda Ryhmä) aloitti anestesian hypnoottisen komponentin monitoroimiseen tähtäävän tuotekehityksen vuonna 1998. Tässä projektissa kerättiin EEG:aa potilaiden ollessa hereillä ja vaihtelevan syvyisissä propofoli- tai sevofluraanianestesioissa, ja tutkittiin eri mahdollisuuksia prosessoida EEG:aa näyttämään luotettavasti anestesian hypnoottista komponenttia. Analyysitapa, johon lopulta päädyttiin, oli spektraalinen entropia, koska se on nopeasti ja luotettavasti laskettavissa eikä edellytä laskenta-algoritmin vaihtamista anestesiasyvyyden muuttuessa.

Datex-Ohmedan entropiamonitoroinnissa EEG-signaali kerätään potilaan otsalta, vahvistetaan, analysoidaan ja esitetään monitorin ruudulla. Entropia-arvo esitetään kahtena lukuna. State Entropy (SE) lasketaan taajuuskaistalta 0,8–32 Hz, ja se kuvaa lähinnä EEG:ssa tapahtuvia muutoksia. Response Entropy (RE) lasketaan 0,8–47 Hz:n kaistalta, joka sisältää sekä EEG:aa että mahdollista otsalihaksen EMG-toimintaa. Koska otsalihaksen EMG aktivoituu kivun aistimisen yhteydessä anestesian aika-

na ja ennakoi potilaan heräämistä anestesian lopussa aiemmin kuin muut merkit, voidaan sitä käyttää arvioitaessa anestesian ”adekvaattisuutta”. SE- ja RE-lukemat ovat anestesian kestäessä hyvin lähellä toisiaan. Jos nämä luvut alkavat erota otsalihaksen aktivoituessa (RE kohoaa, SE ei muutu), voidaan tehdä vallitsevasta tilanteesta riippuvia johtopäätöksiä. Tyypillisiä tilanteita, joissa RE ja SE näyttävät eroavan toisistaan, ovat suora laryngoskopia, intubaatio ja tajuisuuden palautumista edeltävä tilanne anestesian lopussa.

Kun anestesian hypnoottista komponenttia mitataan entropialla, SE:n arvot 40–60 kuvaavat riittävää kirurgista anestesiaa. Jos anestesiaa kevennetään niin, että SE:n arvot nousevat huomattavasti yli 60, tajuisuuden palaamisen todennäköisyys kasvaa ja tätä ennakoi tilanne, jossa SE ja RE alkavat erkaantua toisistaan. Vastaavasti alle 40 entropia-arvot kuvaavat syvää tajuttomuustilaa, josta toipuminen kestää pitkään.

Spektraalinen entropia on osoittautunut BIS:n veroiseksi hereilläolo- ja tajuttomuustilan luokittelijaksi. Entropiamonitorointi näyttää myös liittyvän nopeampaan toipumiseen propofoli-alfentaniili-typpioksiduulianestesian jälkeen. Entropia toimii luotettavasti ainakin sevofluraania, desfluraania, isofluraania, propofolia ja tiopentaalia käytettäessä. Entropia-arvot pienenevät reagoimattomuuden yhteydessä myös opioidivoittoisen sydänanestesian aikana, mutta luotettava entropiamonitorointi edellyttää hypnoottisen lääkityksen eli yleisanesteettien käyttöä[1]. Entropian käyttäytymisestä typpioksiduulia, etomidaattia tai ketamiinia yksinään käytettäessä ei toistaiseksi ole riittävästi tietoa. Entropian käyttökelpoisuutta pitkäaikaisen tehosedaation monitoroinnissa tutkitaan parhaillaan.

Spektraalinen entropia, joka tarjoaa anestesiologin käyttöön kaksi anestesiatilan tunnuslukua, näyttää luokittelevan potilaan tajuttomuus- ja tajuisuustilan luotettavasti, ja auttaa monitoroimaan ja ymmärtämään puutteellisesta analgesiasta johtuvia muutoksia EEG-indeksissä. ❐

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1. Datex-Ohmeda, julkaisematonta aineistoa.

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