Identifying and reporting risk factors for adverse events in endoscopy. Part II: noncardiopulmonary events

Joseph Romagnuolo, MD, FRCPC, MSc, Peter B. Cotton, MD, FRCP, Glenn Eisen, MD, MPH, John Vargo, MD, MPH, Bret T. Petersen, MD

Charleston, South Carolina; Portland, Oregon; Cleveland, Ohio; Rochester, Minnesota, USA

The risks of endoscopy procedures are likely affected by the competence of the endoscopist and the team (nursing, anesthesia, and technicians), the details of the specific procedure being performed, and the patient's anatomy, demographics, and health status. In 2008, the American Society for Gastrointestinal Endoscopy (ASGE) convened a workshop to recommend a lexicon to define and describe the adverse events (AEs) (previously commonly referred to as complications) that can result from endoscopy procedures.[1] One additional goal of this workshop was to standardize the reporting of factors that may predict AEs, in clinical practice and in research. This list of such factors might enable the creation of risk strata (allowing comparison of AE rates by risk groups). In addition, AE rates among different groups of endoscopists and different groups of patients might be more appropriately compared by strata. Last, prospective risk assessment might enhance the quality of informed consent and facilitate decisions regarding procedural appropriateness.

Abbreviations: AE, adverse event; ASA, American Society of Anesthesiologists; ASGE, American Society for Gastrointestinal Endoscopy; CHF, congestive heart failure; MI, myocardial infarction; OR, odds ratio.

DISCLOSURE: *The following authors disclosed financial relationships relevant to this publication: Dr. Romagnuolo: consultant to Olympus, honoraria from Cook endoscopy; Dr. Cotton: board member for Olympus Charitable Medical Foundation, honoraria from Cook Endoscopy, member of the advisory board of Barosense Inc, consultant to and equity in Apollo Endosurgery, consultant to Olympus; education grant from Boston Scientific; Dr. Vargo, consultant to Olympus America. The other authors disclosed no financial relationships relevant to this publication. This article resulted from presentation and discussion at an ASGE-sponsored AE Workshop.*

0016-5107/$36.00
doi:10.1016/j.gie.2010.11.023

Received August 30, 2010. Accepted November 16, 2010.

Current affiliations: Medical University of South Carolina (J.R., P.B.C.), Charleston, South Carolina, Oregon Health and Science University (G.E.), Portland, Oregon, Cleveland Clinic (J.V.), Cleveland, Ohio, Mayo Clinic (B.T.P.), Rochester, Minnesota.

Reprint requests: Joseph Romagnuolo, MD, MUSC GI and Hepatology, 25 Courtenay Drive, ART 7100, MSC 290, Charleston, SC 29425.

As stated in part I, the aim of this 2-part article is to summarize the body of work that has been published on this topic and to suggest the types of risk factors that need to be considered for inclusion in endoscopy reporting/database systems. Possible, but unproven, risk factors were also considered to guide further research into candidate factors and their relationship with AEs. Establishing and recording the competency of the team are beyond the scope of this article.

AEs are often organized by type of event (eg, cardiopulmonary, bleeding, perforation). However, their frequency and type clearly vary by procedure (the risks of colonoscopy are different than those of ERCP), and some events (pancreatitis, infection) rarely or never apply to some procedures. Equally, the factors that predict those AEs also vary among procedures (eg, demographics may predict post-ERCP pancreatitis, but may have little influence on EGD complications).

Because many noncardiopulmonary AEs are closely linked to the type of intervention (eg, bleeding or perforation, related to polypectomy), each procedure (eg, colonoscopy, upper endoscopy) would have to be discussed separately under each AE heading. For a more efficient presentation of the data, we elected to discuss them organized by procedure type rather than by AE type. In contrast, most predictors of cardiopulmonary AEs are patient-centered factors and do not vary significantly from procedure to procedure (although admittedly procedural complexity and duration may influence event rates). In addition, comorbidities do not generally predict the noncardiopulmonary events. Therefore, the cardiopulmonary AEs and their predictors, which are relatively constant across procedure types and which require a discussion of the various comorbidity indices, are discussed separately from the other AEs and their potential predictors in part I of this 2-part article. Part II discusses predictors of noncardiopulmonary events and contains a summary and final recommendations for documentation of predictors or risk factors for both types of events.

METHODS

As described in part I, a comprehensive PubMed search to June 1, 2010, yielded more than 2000 articles by using

the following criteria: EGD/endoscopy, complications (n = 256); colonoscopy, complications (n = 692); EUS, complications (n = 175); ERCP, complications (n = 573); esophageal dilation, complications (n = 293); comorbidity index, endoscopy (n = 276); sedation risks, endoscopy (n = 246). A search of the Cochrane library uncovered one additional systematic review.[2] Abstracts from these articles were read to select articles pertaining to predictors or risk factors of AEs in endoscopy. The majority of the articles listed in the PubMed search were unfortunately not relevant to the prediction of AEs in endoscopy; 106 articles were selected as potentially relevant and critically reviewed. In addition, we reviewed references to several comorbidity indices, and citations from reviews on risk assessment in endoscopy and noncardiac surgery. The data and conclusions were presented at the ASGE Adverse Events Workshop (September 5-6, 2008, Chicago, Ill). Further in-person and online discussion occurred, and the article draft was then reviewed by members of the Workshop and approved by the ASGE Quality Committee.

RISK FACTORS FOR NONCARDIOPULMONARY EVENTS

Most predictors for noncardiopulmonary events are procedure-specific rather than generic (Tables 1-3). The AEs that we want to predict are different for each procedure (eg, perforation vs pancreatitis); even procedures that overlap in terms of types of certain AEs (eg, bleeding) have different instruments and techniques that lead to the AEs. For most, it is unlikely that a common list of predictors will be possible or would prove to be very helpful.

Colonoscopy

Independent predictors of bleeding after colonoscopy include advanced age (odds ratio [OR] 1.4-1.6),[3-6] male sex (somewhat unexplained, perhaps caused by unreported/unaccounted medications, but found to be an independent predictor in 4 studies) (OR 1.2-9.2),[4,6-8] nonscreening/nonsurveillance indication (OR 1.3),[6] cardiovascular or renal disease (OR 2.1-3.3),[3] polypectomy (OR 10.3 vs no polypectomy)[4,6,7,9,10] (especially large-polyp [>10 mm] polypectomy [OR 2.4-5.6 vs small polypectomy]),[3,6,7,9,11-13] and coagulopathy (OR 3.7-13.4)[3,8,12,14] or resumption of anticoagulation within 1 week (OR 5.2).[14] A risk increase has not been seen with aspirin (although some studies stopped aspirin 3 days or more before colonoscopy).[9,12,14,15] Until recently, studies had not shown an increase in bleeding risk with clopidogrel or ticlopidine, but the power was limited because of the small numbers of patients with events on these medications. A study has now shown that there does not seem to be an increased risk with clopidogrel, but that the combination of clopidogrel with either aspirin or a nonsteroidal anti-inflammatory drug does increase risk (OR 3.7).[5] A recent study of overall AEs within 30 days found clopidogrel and warfarin use to increase risk, but most of the clopidogrel users were likely also taking aspirin.[16] The risk of bleeding AEs in general may also have been decreasing over the past decades.[7,10] Technique may also play a role, as pure cutting current may increase risk,[3] and pure coagulation current may trade off a decrease in immediate bleeding for an equal increase in delayed bleeding.[17] Detachable snares and clips may be helpful for larger polyps.[18] Other studies show no difference in risk with respect to technique.[11] Perforation is rare, and polypectomy, age, and comorbidities seem to be significant risk factors.[4,19] In 3 studies, when perforation and bleeding were considered together, endoscopist volume was found to be a predictor when considering all endoscopists.[4,20] However, this relationship between volume/experience and AEs did not seem to exist among gastroenterologists[4] and was not seen in other studies.[3] The perforation rate may be higher among nongastroenterologists.[20] Infection and splenic injury[21,22] are extremely rare. Right-sided polypectomy may increase overall complications.[13]

Upper endoscopy

For diagnostic EGD, the predominant risk is that which is related to the sedation, but therapeutic procedures add other risks (Table 2). Bleeding risks are increased by polypectomy (especially EMR),[23] variceal therapy (sclerotherapy more than banding),[24] and hemostatic procedures. For perforation, polypectomy (especially EMR),[25-29] dilation (achalasia[30-32] more than nonachalasia[33-38] dilations), sclerotherapy of varices,[24] foreign body removals,[39,40] nonvariceal hemostasis procedures,[41-43] and stenting[44] procedures are associated with increased risk. Risk of stricture formation is increased by ablation procedures,[44] variceal therapy,[24,25] and EMR.[25,46] Infection is very rare after upper endoscopy. Metal expandable stents, natural orifice transluminal endoscopic surgery, and percutaneous procedures (such as PEG and percutaneous endoscopic jejunostomy) are not discussed. However, peristomal infection does seem to be increased (OR 3.2) after PEG if antibiotics are not given.[47]

ERCP

Table 3 summarizes predictors of AEs after ERCP. Infection is more common after ERCP than after upper or lower endoscopy, but it remains very rare. However, it is higher in liver transplant patients (OR 5.2),[48] hilar/intrahepatic strictures,[49,50] jaundiced patients (OR 1.4)[51] (although this probably applies only if obstruction is not relieved by the procedure), and in small centers (OR 1.4).[51] It is also likely higher in those with leaks/fistulae (biliary and pancreatic) and ductoscopy. Bleeding can occur after therapeutic procedures; sphincterotomy (OR 4.7)[52] and performance of procedures in small centers (OR 1.1)[51] are predictors. In the sphincterotomy cases,[53] coagulopathy (OR 3.3), resumption of anticoagulants within 3 days (OR 5.1), cholangitis (OR 2.6), low case volume (OR

TABLE 1. Colonoscopy and predictors of noncardiopulmonary adverse events

Adverse event	Modifying factor	Risk magnitude (OR)	References	Comments
Infection	—			Very rare
Bleeding*	Polypectomy (vs no polypectomy)	10.3	4, 6, 7, 9, 10	Very rare (1.6/1000) without polypectomy; uncommon with polypectomy (<1%); 1 study found higher risk with biopsy alone but may have included self-limited bleeding[6]
	Age	1.4 (>65 y); 1.6 (>60 y); 1.03/y	3-6	
	Year/era	1.7/5 y period in past	7, 10	Bleeding seems to be decreasing with time
	Male sex	1.2-9.2	4, 6-8	Unexplained, and possibly due to uncorrected confounders
	Nonscreening/ nonsurveillance indication	1.3	6	May have included minor self-limited bleeding
	Polyp size	2.4-28 for >10 mm; 1.1/1 mm increase; 12 for 5-10 mm vs 5 mm	3, 6, 7, 9, 11-13	
	Polyp number	1.1-1.3	5, 6, 8	
	Anticoagulation after polypectomy	3.7-13.4 (current), 5.2 (resumed within 1 week)	3, 8, 12, 15	One study showed greater relative risk increase with small polyps[12]
	Antiplatelets	3.7 for clopidogrel plus aspirin or NSAID	5, 9, 12, 14, 15	No increase risk with aspirin[5,9,12,14,15] or clopidogrel alone,[5,14] but the combination increases bleeding[5]
	Cardiovascular disease†	2.1	3	Hypertension, ischemic or valvular heart disease for >6 mo, an arrhythmia requiring medication, or cerebrovascular disease
	Renal disease†	3.3	3	Serum creatinine level of >3 mg/dL for >6 mo
	Polyp morphology†	1.4 for sessile in one study; 1.5 for peduncular in another	3, 6	Sessile, semipedunculated, or lateral spreading vs others; other studies did not see a difference[11,12]
	Pure cutting current†	7.0	3, 17	Also inadvertent cold polypectomy had higher risk (OR 7.2); pure coagulation current may trade off a decrease in immediate for an increase in delayed bleeding[17]
	Poor bowel prep†	1.5	3	Adequate or poor higher risk than good or excellent
Perforation	Polypectomy	2.3-3.0	4, 6, 10, 16, 19, 97	Rare (~0.1%) without polypectomy, uncommon (0.5%) after polypectomy; reference 19 summarizes multiple studies compiling >50,000 polypectomy cases
	Age	2.1 (>60 y)	4, 6	
	Comorbidities	3.0	4	Deyo score of ≥3 based on Charlson index
	Renal failure, on dialysis	19.2	98	
Splenic injury	Female sex	—	21	Very, very rare (0.001%)[22]; predictors reported based on literature review
	Difficult procedure	—	21	

NSAID, nonsteroidal anti-inflammatory drug; *OR*, odds ratio.
*Apart from polypectomy, other risk factors for bleeding are only within the postpolypectomy group and do not necessarily apply to cases without polypectomy; the odds ratios for these other factors are with respect to the lower risk category of polyps, not versus no polypectomy at all.
†Many of these factors were not found by other studies, and this study was mainly of immediate bleeding (4% bleeding rate, most stopping within 60 seconds) rather than clinically important delayed bleeding.

TABLE 2. EGD and predictors of noncardiopulmonary adverse events

Adverse event	Modifying factor	Risk magnitude (OR)	References	Comments
Infection	—			Very rare
Bleeding (delayed)	Polypectomy	—	23	Very rare without polypectomy; 5%-15% with polypectomy; OR 8.1 if immediate bleeding at gastric EMR
	Variceal therapy	—	24	Sclerotherapy (25%) greater than banding (6%)
	Hemostasis (nonvariceal)	—		
Perforation	Polypectomy		25-29	Rare (~0.5% of EMR; higher [about 5%-10%] with ESD)
	Dilation (of achalasia)	—	30-32	2%-3% in meta-analyses of pneumatic dilation
	Dilation (nonachalasia)	—	33-37	Higher with Maloney, 10% perforation in anastomotic strictures in children, 5% anastomotic strictures in adults, 15%-20% post-caustic ingestion, 2% radiation stricture
	EGD in eosinophilic esophagitis (with or without dilation)	—	38, 99	Can occur even with passage of an endoscope, but mucosal tears are much more common than true perforations, which seem rare
	Variceal therapy	—	24	Sclerotherapy, 2%
	Foreign body	—	39, 40	No perforation in 2 series of food impactions
	Stent	—	44	Review, 7%-15%
	Hemostasis (nonvariceal)	—	41-43	Low risk (<1%) unless sclerosants used; very rare with nonsclerosant monotherapy
Stricture	Ablation	—	44	20% after photodynamic therapy (n = 102)
	Variceal therapy	—	24, 45	Sclerotherapy, 26%; much greater than banding 2%
	EMR	—	25, 46	5%-25% of esophageal EMR (especially if circumferential resection, ≤70%); 3% for peripyloric

ESD, endoscopic submucosal dissection; *OR*, odds ratio.

2.2), and bleeding during the procedure (OR 1.7) predict a significant bleeding AE. Antiplatelet agents do not seem to substantially increase the risk of postsphincterotomy bleeding,[53,54] but data on clopidogrel are limited. Perforation is associated with surgically altered anatomy (OR 2.5),[51,52] sphincterotomy (precut [OR 2.0] more than conventional),[51] and intramural contrast injection (OR 1.9, although this may just be a marker of a difficult cannulation).[51] There are many studies looking at predictors of post-ERCP pancreatitis, and not all agree (eg, normal-caliber bile duct, bilirubin level, and trainee involvement are predictors in some but not others). Established and independent predictors include female sex (OR 1.8-3.5),[55-57] suspected sphincter of Oddi dysfunction (OR 1.9-9.7),[52,55,58,59] pancreatic injection (OR 1.04-1.5)[51,55,58,60-62] (especially filling to the tail),[62] pancreatic sphincterotomy (OR 1.5-3.8),[52,55,58,60] difficult cannulation (OR 1.8-9.4),[55-57,59] and lack of pancreatic stenting in high-risk cases (OR 1.4-3.2).[52,63-65] Biliary sphincterotomy does not seem to increase the risk of pancreatitis, and it is not clear whether precut/needle-knife sphincterotomy adds risk beyond that of the difficulty of cannulation itself; early precut sphincterotomy may even reduce risk versus persistence according to meta-analyses of randomized trials.[66,67] Although comorbidities, including obesity, do not seem to significantly increase the risk, they may influence the severity, morbidity, and mortality of pancreatitis when it occurs; the same may be true for other events such as perforation. Other related specialized procedures such as pseudocyst drainage, percutaneous cholangioscopy, and laser or electrohydraulic lithotripsy each have unique risks, but data are limited and they are not discussed here.

EUS

EUS, although typically a longer procedure than an EGD, has a risk profile similar to that of EGD, unless therapy (EMR, dilation) or FNA is performed (Table 4). Even after FNA procedures, infection seems to be very

TABLE 3. ERCP and predictors of noncardiopulmonary adverse events

Adverse event	Modifying factor	Risk magnitude (OR)	References	Comment(s)
Infection	Liver transplant	5.2	48	Very rare (0.25%-0.5%) (risk decreasing with time: OR 0.9/y)
	Fistulae, nondrainable ducts (eg, hilar, intrahepatic strictures)		49, 50	
	Ductoscopy			
	Jaundice	1.4	51	
	Small center	1.4	51	
Bleeding (delayed)	Sphincterotomy	4.7	52	Very rare; can also occur with large balloon sphincteroplasty; no increased risk with antiplatelet agents[54]
	Small center	1.1	51	
	Intraprocedure bleeding*	1.7	53	
	Coagulopathy*	3.3	53	Defined as >2-s prothrombin time, hemodialysis or platelet count <80,000/mm^3
	Anticoagulation within 3 days*	5.1	53	
	Cholangitis*	2.6	53	
	Small-volume endoscopist*	2.2 (≤1/wk)	53	
Perforation	Postsurgical anatomy	2.5	51, 52	
	Precut sphincterotomy	2.0	51	
	Intramural contrast	1.9	51	
	Sphincterotomy			Rare
Pancreatitis†	Suspected SOD	1.9-9.7	52, 55, 58, 59	Biliary sphincterotomy is not a risk factor[51,56,58]
	Female	1.8-3.5	55-57, 59	
	Post-ERCP pancreatitis (prior)	5.4	55	
	Younger age	1.1/5-y decrease[60]; 1.1 (age <70 y)[55]; 1.6 (age <60 y)[62]	51, 56, 58	
	Normal bile duct	1.05	51	
	Normal bilirubin	1.9	55	
	No chronic pancreatitis	1.9	55	
	Nonuniversity center	2.4	51	
	Difficult cannulation	1.8-9.4	55-57, 59	Early precut sphincterotomy may reduce risk vs persistence[66,67]
	Pancreatic sphincterotomy	1.5-3.8	Standard,[52,55,60] minor papilla[52,58]	
	Pancreatic injection	1.04-1.5	51, 55, 58, 60-62	Most studies define as any injection; in Cheng et al,[58] defined as ≥2; extent of filling important[62]
	No pancreatic stenting	1.4-3.2	52, 63-65	Significant in high-risk ERCP, especially SOD
	Trainee involvement	1.5	58	
	Balloon sphincteroplasty	2.0	[100]	For stone disease, heterogeneous studies

OR, Odds ratio; *SOD,* Sphincter of Oddi dysfunction.
*These risk factors were determined only in sphincterotomy subgroup and do not necessarily predict bleeding in all ERCPs.
†As is evident in this section, some factors have more consensus than others on their role as a risk factor for post-ERCP pancreatitis; factors only found significant in 1 of many studies may or may not be true risk factors.

TABLE 4. EUS and predictors of noncardiopulmonary adverse events

Adverse event	Modifying factor*	References	Comments
Infection	FNA	68-74	Very rare with FNA, rare with FNA
	Pancreatic cysts	74	Rare (0.2%), especially with antibiotic prophylaxis; risk factors thought to include incomplete drainage, multiple punctures
	Mediastinal cysts	75-78	Common and can be severe; FNA best avoided
	Transrectal/ colonic FNA	79, 80	Uncommon (4%); good prep critical
	Celiac block	81-84	Rare
Bleeding (delayed)	FNA	74, 85, 86	Very rare (1%-4%), mostly intraluminal
Perforation	Age, difficult intubation, inexperience	90	0.03% cervical esophageal perforation (older patient, history of difficult esophageal intubation, inexperienced operator)
Pancreatitis	FNA	87-89	Very rare
	FNA of pancreas mass	88, 91, 92	Very rare without FNA, rare with FNA (1%-2%); placement of fiducials can be associated with pancreatitis[101]
	FNA for nonfocal chronic pancreatitis	93	Seems much higher than for masses, especially if Trucut is used (13%); intracystic brushing may increase risk[94]
Bile leak	Gallbladder FNA	95	High risk (>50%), not recommended
Pneumothorax	Lung mass FNA	77, 96	Very rare

*Adjusted odds ratios not clear for any of these risk factors as they have not been subject to multivariate analysis.

rare.[68-74] Risk of infection is heightened by FNA of pancreatic cysts,[74] FNA of mediastinal cysts,[75-78] FNA via a transcolonic route,[79,80] and performance of celiac plexus block or neurolysis.[81-84] Bleeding is very rare, even with FNA.[74,85,86] However, there are limited data on the influence of anticoagulants or antiplatelet agents on the risk of bleeding after FNA. Perforation is rare,[87-89] but cervical esophageal perforation from endoscope insertion has been reported with older patients with histories of difficult esophageal intubation, especially for inexperienced operators.[90] It may also potentially occur with altered surgical anatomy or traversing strictures, but data are limited on these settings. Pancreatitis is rare,[88,91,92] unless pancreatic FNA is done for reasons other than a suspected tumor[93] and is uncommon in cysts unless Trucut or intracystic brushing is performed.[94] Bile leak is very rare but has been associated with gallbladder FNA[95] (for collection of bile) and reported for complex procedures such as EUS-guided transluminal cholangiography with or without stenting. Pneumothorax can potentially occur with FNA of lung masses, but is very rare.[77,96] EUS-guided natural orifice transluminal endoscopic surgery, EUS-guided ductography, and EUS-guided intracystic or intratumoral therapeutic injections are not discussed.

SUMMARY

Our goal was to identify or develop a relatively small data set that would be useful in predicting the risk of endoscopic procedures. This review shows that this is not an easy task because there are a wide range of factors and a variety of risks. Risk assessment may also be complex when more than 1 risk factor is present because the way in which different factors interact is variable.

To summarize what was concluded in part I, for the cardiopulmonary events, it seems that documentation should include age, inpatient versus outpatient status (perhaps also clarify for inpatients where the procedure was performed [endoscopy suite vs other] and when [routine hours vs off-hours, elective vs urgent/emergent]), trainee involvement and type of sedation or anesthesia, supplemental oxygen use, and major comorbidities (especially cardiopulmonary compromise and recent myocardial infarction [MI]). The American Society of Anesthesiologists (ASA) class is most frequently used as a broad measure of fitness and inherently combines severity of comorbidities with functional status, but there are problems with interobserver agreement and lack of detail. Unfortunately, some widely recorded factors, such as the Mallampati score (because it reflects potential difficulty in intubation), are not discussed in this review because their predictive value for AEs after endoscopy have not been validated.

The literature on cardiac risk status could be condensed for endoscopy. Relevant items include age, previous or recent MI, previous congestive heart failure (CHF), current/recent CHF, rhythm other than sinus, diabetes, renal failure, uncontrolled hypertension, previous stroke

or other neurologic impairment, inability to walk up a flight of stairs (or equivalent activity), dependency (on someone to help with activities of daily living), sleep apnea, or severe chronic obstructive pulmonary disease. Although comorbidities do not seem to increase the risk of most noncardiopulmonary events, eg, pancreatitis and perforation, they likely do influence the morbidity and mortality of those events if they occur.

In terms of noncardiopulmonary events, Tables 1 through 4 show that the main predictors are the demographics and indications (eg, suspected sphincter of Oddi dysfunction) and the actual procedure performed (eg, EMR). These are already well documented in most endoscopy reporting systems. Items such as liver transplant status and surgically altered anatomy should be noted even if not technically part of the indication for the procedure. So-called intraprocedural incidents (eg, bleeding that stops spontaneously or with endoscopic treatment) that do not meet the threshold for an AE are worth documenting because it is possible that some may predict real AEs. There are limited data proving that monotherapy with antiplatelet agents is related to AEs; however, combination antiplatelet therapy should be noted because it seems to predict bleeding, and drugs such as clopidogrel are often held for 3 to 7 days before and/or after large-polyp polypectomy, sphincterotomy, or FNA. It would be useful to note the amount of time that these drugs are held before and after endoscopy. The rate of balancing events (such as cardiovascular and cerebrovascular events) that may occur as a result of antiplatelet agents being held, especially in patients with coronary stents, needs further study. Active anticoagulation is a risk factor for bleeding after therapeutic or FNA procedures, but anticoagulation is generally held (for >4-5 days) when those procedures are done, essentially eliminating that immediate bleeding risk. Therefore, it likely is not worth noting anticoagulant medications for the purpose of predicting bleeding risk, unless the medication use is current (ie, uncorrected coagulopathy). What may be more worthwhile to note, however, because bleeding can be delayed by as long as 10 to 14 days, is whether anticoagulation is expected to be resumed within the next 14 days (and if so, when). Noting this in the recommendations of the procedure report is suggested.

RECOMMENDATIONS FOR CARDIOPULMONARY (PART I) AND NONCARDIOPULMONARY (PART II) EVENTS

Table 5 summarizes a list of factors that either are known (basic use, recommended for routine practice) or are suspected (intermediate/advanced use, for more advanced reporting and research) to predict AEs. Table 6 represents a proposal of the menu items that might be used in an endoscopic database to record the factors that are listed in Table 5.

TABLE 5. Summary of the recommended adverse event predictive factors to be reported for basic, intermediate, and advanced/investigational use

Basic
Demographics
ASA class
Outpatient/Inpatient/intensive-care unit/other setting
Diagnosis/indication/context (jaundice, pancreatic cyst aspiration, surgically altered anatomy, gastric retention)*
Procedure therapeutic details†
Type of anesthesia, and who administered it
Procedure duration
Current anticoagulant/antiplatelet therapy
Antibiotic prophylaxis
Intermediate
Basic, plus:
Recent anticoagulation/antiplatelet therapy (or holding thereof)‡
Immunosuppression
Major comorbidity
(e.g., coma, cardiomyopathy, dialysis, cirrhosis with or without ascites, oxygen-requiring chronic pulmonary disease)
Trainee involvement
Advanced
Intermediate, plus:
Cardiac status (previous/recent angina, myocardial infarction, heart failure, or arrhythmia)
Coronary interventions (previous/recent angioplasty/stent, bypass surgery)
Pulmonary status (resting hypoxia, severe chronic pulmonary disease, sleep apnea)
Neurologic status (stroke with residual deficit, decreased level of consciousness, or coma)
Functional status (inability to walk up a flight of stairs [or equivalent activity], dependency on someone to help with activities of daily living)
Nutritional status (including malnutrition and obesity)
Other comorbidities (including immunosuppression)

ASA, American Society of Anesthesiologists.
Including type, duration held before procedure, duration to hold after procedure.
*With special attention to the factors in Tables 1 through 4.
†Such as size/type of polypectomy, type of sphincterotomy, type of hemostasis/variceal treatment.
‡Including type, duration held before procedure, duration to hold after procedure.

TABLE 6. Proposed menu items for endoscopy reporting software for the factors listed in Table 2*

- Demographics
  - Age
  - Sex
- Clinical status
  - BMI
    - <20
    - 20-30
    - 30-40
    - 40-45
    - >45
  - ASA grade
  - Independence (KPS scale)
    - Able to carry on normal activity and to work; no special care needed (KPS score 80-100)
    - Unable to work; able to live at home and care for most personal needs; varying amount of assistance needed (KPS score 50-70)
    - Unable to care for self; requires equivalent of institutional or hospital care; disease may be progressing rapidly (KPS score 0-40)
  - Coagulation issues
    - Current anticoagulation (therapeutic INR or PTT)
    - Current antiplatelet effect (last dose within 48 hours)
    - Current prophylactic (low dose) low-molecular weight heparin (last dose within 24 hours)
    - Recently stopped
      - Aspirin: specify number of days since last dose
      - Coumadin and related nonheparin anticoagulant agents: specify number of days since last dose
      - Unfractionated heparin: specify number of hours since last dose
      - Low-molecular weight heparin: specify number of half-days since last dose
      - Nonaspirin antiplatelet agents: specify number of days since last dose
      - NSAIDs: specify number of days since last dose
    - Plan to restart anticoagulant or antiplatelet agents <10 days: specify anticipated delay to restart
  - Cardiac
    - Previous coronary intervention: bypass surgery, angioplasty without stent, drug-eluting stent, bare metal stent
    - Timing of coronary intervention (for each intervention)
      - <6 weeks
      - 6 weeks to 12 months
      - >12 months
    - Previous MI
    - Recent MI
      - <1 week
      - 1 week to 1 month
      - within 6 months
      - 6-12 months
    - Previous heart failure
    - Recent heart failure (within 1 month)
    - Severe cardiomyopathy (ejection fraction <25%)
    - Atrial flutter/fibrillation
    - Ventricular arrhythmia
  - Pulmonary
    - Resting hypoxia (Sao_2 <90% on room air)
    - Severe chronic pulmonary disease
      - Previous admission
      - Oxygen at home
    - Sleep apnea
  - Cardiopulmonary functional status
    - No limitations
    - Limited but able to climb 1 flight of stairs (or equivalent)
    - Unable to climb a flight of stairs (or equivalent)
  - Neurologic status
    - Previous stroke, no/minimal residual deficit
    - Previous stroke with residual deficit
    - Recent stroke (within 1 month)
    - Decreased level of consciousness
    - Coma
  - Renal/liver
    - Significant renal insufficiency (creatinine >2 mg/dL) but not on dialysis
    - Dialysis or awaiting dialysis
    - Portal hypertension or ascites

(*continued on next page*)

TABLE 6. (continued)

Other comorbidity (eg, diabetes)
Poor nutritional status
Albumin <2.5 g/dL
Weight loss >10%
Limited oral intake for >7 days
Other
Procedure
Status/setting
Outpatient
Inpatient
Endoscopy unit
Intensive care unit
Operating room
Emergency department
Urgent, during working hours
Urgent, outside of working hours
Trainee involvement
Hands on: 0%, 1%-25%, 25%-50%, 50%-75%, 75%-100%
Administrator/type of sedation/anesthesia
Endoscopist, moderate/conscious sedation
Endoscopist, involving propofol (deep sedation or general anesthesia without intubation)
Anesthesia team, moderate/conscious sedation
Anesthesia team, involving propofol (deep sedation or general anesthesia without intubation)
General anesthesia with intubation
Nonendoscopist/nonanesthesia sedation
Procedure duration (time from endoscope in to endoscope out)

ASA, American Society of Anesthesiologists; *BMI*, body mass index; *INR*, international normalized ratio; *KPS*, Karnofsky Performance Status; *MI*, myocardial infarction; *NSAID*, nonsteroidal anti-inflammatory drug; *PTT*, partial prothrombin time.

On current evidence, to fulfill the basic use criteria, we recommend that endoscopy reporting systems allow documentation of, in addition to demographics (age, sex), details of the procedure sufficient to describe the elements highlighted in Tables 1 through 4 (eg, size of polyp removed at colonoscopy, type of sphincterotomy, extent of pancreatic injection at ERCP, context of dilation at EGD [achalasia vs nonachalasia]), inpatient/outpatient status, and the ASA class. The ASA classification covers a broad range of reasons for unfitness, although it has known limitations. Uncorrected coagulopathy (intrinsic or medicated) and current antiplatelet use should also be recorded.

Some endoscopists, especially those in research facilities, will want to record more details of cardiac risk (especially previous/recent MI, CHF, and arrhythmia), pulmonary status (especially resting hypoxia or severe chronic obstructive pulmonary disease), details of other severe comorbidities (eg, renal failure requiring dialysis, severe liver disease), serious neurologic disease (eg, stroke with residual deficit, current coma), functional status, and severe immunosuppression (Table 5). In addition, any expected resumption (including in how many days resumption has been suggested) of halted antiplatelet agents or anticoagulants within 14 days of the procedure should be recorded because it may contribute to delayed complications, including both bleeding and vascular thrombosis, in high-risk patients or procedures. The influence of malnutrition, obesity, body mass index, and associated sleep apnea on procedural and cardiopulmonary event rates is unclear, but they are suspected to be increasingly important issues. More research is needed to establish which of these additional factors, or others, are worth documenting on a routine basis. Procedure difficulty scales for each of the major procedures (EGD, colonoscopy, EUS, ERCP) may also be useful to record to help with risk stratification, and such scales are in the process of being developed by the group.

Routine documentation of a basic or an advanced set of modifying factors will help identify or clarify the role of various patient-, operator-, and procedure-related variables in predicting AEs. Although the role of several factors is already known, many others require further research.

REFERENCES

1. Cotton PB, Eisen GM, Aabakken L, et al. A lexicon for endoscopic adverse events: report of an ASGE workshop. Gastrointest Endosc 2010; 71:446-54.
2. Singh H, Poluha W, Cheung M, et al. Propofol for sedation during colonoscopy. Cochrane Database Syst Rev 2008;(8):CD006268.
3. Kim HS, Kim TI, Kim WH, et al. Risk factors for immediate postpolypectomy bleeding of the colon: a multicenter study. Am J Gastroenterol 2006;101:1333-41.
4. Rabeneck L, Paszat LF, Hilsden RJ, et al. Bleeding and perforation after outpatient colonoscopy and their risk factors in usual clinical practice. Gastroenterology 2008;135:1899-906, 1906 e1.
5. Singh M, Mehta N, Murthy UK, et al. Postpolypectomy bleeding in patients undergoing colonoscopy on uninterrupted clopidogrel therapy. Gastrointest Endosc 2010;71:998-1005.
6. Crispin A, Birkner B, Munte A, et al. Process quality and incidence of acute complications in a series of more than 230,000 outpatient colonoscopies. Endoscopy 2009;41:1018-25.
7. Paspatis GA, Vardas E, Theodoropoulou A, et al. Complications of colonoscopy in a large public county hospital in Greece. A 10-year study. Dig Liver Dis 2008;40:951-7.

8. Witt DM, Delate T, McCool KH, et al. Incidence and predictors of bleeding or thrombosis after polypectomy in patients receiving and not receiving anticoagulation therapy. J Thromb Haemost 2009;7:1982-9.
9. Shiffman ML, Farrel MT, Yee YS. Risk of bleeding after endoscopic biopsy or polypectomy in patients taking aspirin or other NSAIDS. Gastrointest Endosc 1994;40:458-62.
10. Macrae FA, Tan KG, Williams CB. Towards safer colonoscopy: a report on the complications of 5000 diagnostic or therapeutic colonoscopies. Gut 1983;24:376-83.
11. Watabe H, Yamaji Y, Okamoto M, et al. Risk assessment for delayed hemorrhagic complication of colonic polypectomy: polyp-related factors and patient-related factors. Gastrointest Endosc 2006;64:73-8.
12. Sawhney MS, Salfiti N, Nelson DB, et al. Risk factors for severe delayed postpolypectomy bleeding. Endoscopy 2008;40:115-9.
13. Heldwein W, Dollhopf M, Rösch T, et al. The Munich Polypectomy Study (MUPS): prospective analysis of complications and risk factors in 4000 colonic snare polypectomies. Endoscopy 2005;37:1116-22.
14. Hui AJ, Wong RM, Ching JY, et al. Risk of colonoscopic polypectomy bleeding with anticoagulants and antiplatelet agents: analysis of 1657 cases. Gastrointest Endosc 2004;59:44-8.
15. Yousfi M, Gostout CJ, Baron TH, et al. Postpolypectomy lower gastrointestinal bleeding: potential role of aspirin. Am J Gastroenterol 2004; 99:1785-9.
16. Ko CW, Riffle S, Michaels L, et al. Serious complications within 30 days of screening and surveillance colonoscopy are uncommon. Clin Gastroenterol Hepatol 2010;8:166-73.
17. Van Gossum A, Cozzoli A, Adler M, et al. Colonoscopic snare polypectomy: analysis of 1485 resections comparing two types of current. Gastrointest Endosc 1992;38:472-5.
18. Kouklakis G, Mpoumponaris A, Gatopoulou A, et al. Endoscopic resection of large pedunculated colonic polyps and risk of postpolypectomy bleeding with adrenaline injection versus endoloop and hemoclip: a prospective, randomized study. Surg Endosc. Epub 2009 May 9.
19. Misra T, Lalor E, Fedorak RN. Endoscopic perforation rates at a Canadian university teaching hospital. Can J Gastroenterol 2004;18:221-6.
20. Singh H, Penfold RB, DeCoster C, et al. Colonoscopy and its complications across a Canadian regional health authority. Gastrointest Endosc 2009;69:665-71.
21. Saad A, Rex DK. Colonoscopy-induced splenic injury: report of 3 cases and literature review. Dig Dis Sci 2008;53:892-8.
22. Kamath AS, Iqbal CW, Sarr MG, et al. Colonoscopic splenic injuries: incidence and management. J Gastrointest Surg 2009;13:2136-40.
23. Okano A, Hajiro K, Takakuwa H, et al. Predictors of bleeding after endoscopic mucosal resection of gastric tumors. Gastrointest Endosc 2003;57:687-90.
24. Schmitz RJ, Sharma P, Badr AS, et al. Incidence and management of esophageal stricture formation, ulcer bleeding, perforation, and massive hematoma formation from sclerotherapy versus band ligation. Am J Gastroenterol 2001;96:437-41.
25. Kantsevoy SV, Adler DG, Conway JD, et al. Endoscopic mucosal resection and endoscopic submucosal dissection. Gastrointest Endosc 2008;68:11-8.
26. Gotoda T, Yamamoto H, Soetikno RM. Endoscopic submucosal dissection of early gastric cancer. J Gastroenterol 2006;41:929-42.
27. Singer M, Busse R, Seib HJ, et al. Endoscopic polypectomy of the upper gastro-intestinal tract: results and clinical features (author's translation) [German]. Dtsch Med Wochenschr 1975;100:2313-6.
28. Spinelli P, Cerrai FG, Casella G, et al. Endoscopic treatment of polyps in the resected stomach. Minerva Chir 1994;49:393-6.
29. Alexander S, Bourke MJ, Williams SJ, et al. EMR of large, sessile, sporadic nonampullary duodenal adenomas: technical aspects and long-term outcome (with videos). Gastrointest Endosc 2009;69:66-73.
30. Leyden JE, Moss AC, MacMathuna P. Endoscopic pneumatic dilation versus botulinum toxin injection in the management of primary achalasia. Cochrane Database Syst Rev 2006;(4):CD005046.
31. Campos GM, Vittinghoff E, Rabl C, et al. Endoscopic and surgical treatments for achalasia: a systematic review and meta-analysis. Ann Surg 2009;249:45-57.
32. Wang L, Li YM, Li L. Meta-analysis of randomized and controlled treatment trials for achalasia. Dig Dis Sci 2009;54:2303-11.
33. Hernandez LV, Jacobson JW, Harris MS. Comparison among the perforation rates of Maloney, balloon, and Savary dilation of esophageal strictures. Gastrointest Endosc 2000;51:460-2.
34. Ko HK, Shin JH, Song HY, et al. Balloon dilation of anastomotic strictures secondary to surgical repair of esophageal atresia in a pediatric population: long-term results. J Vasc Interv Radiol 2006;17:1327-33.
35. Kim HC, Shin JH, Song HY, et al. Fluoroscopically guided balloon dilation for benign anastomotic stricture after Ivor-Lewis esophagectomy: experience in 62 patients. J Vasc Interv Radiol 2005;16:1699-704.
36. Swaroop VS, Desai DC, Mohandas KM, et al. Dilation of esophageal strictures induced by radiation therapy for cancer of the esophagus. Gastrointest Endosc 1994;40:311-5.
37. Karnak I, Tanyel FC, Buyukpamukcu N, et al. Esophageal perforations encountered during the dilation of caustic esophageal strictures. J Cardiovasc Surg (Torino) 1998;39:373-7.
38. Kaplan M, Mutlu EA, Jakate S, et al. Endoscopy in eosinophilic esophagitis: "feline" esophagus and perforation risk. Clin Gastroenterol Hepatol 2003;1:433-7.
39. Vicari JJ, Johanson JF, Frakes JT. Outcomes of acute esophageal food impaction: success of the push technique. Gastrointest Endosc 2001; 53:178-81.
40. Weinstock LB, Shatz BA, Thyssen SE. Esophageal food bolus obstruction: evaluation of extraction and modified push techniques in 75 cases. Endoscopy 1999;31:421-5.
41. Chung SC, Leung JW, Sung JY, et al. Injection or heat probe for bleeding ulcer. Gastroenterology 1991;100:33-7.
42. Gevers AM, De Goede E, Simoens M, et al. A randomized trial comparing injection therapy with hemoclip and with injection combined with hemoclip for bleeding ulcers. Gastrointest Endosc 2002;55:466-9.
43. Marmo R, Rotondano G, Piscopo R, et al. Dual therapy versus monotherapy in the endoscopic treatment of high-risk bleeding ulcers: a meta-analysis of controlled trials. Am J Gastroenterol 2007;102:279-89; quiz 469.
44. Newcomer MK, Brazer SR. Complications of upper gastrointestinal endoscopy and their management. Gastrointest Endosc Clin N Am 1994; 4:551-70.
45. Guynn TP, Eckhauser FE, Knol JA, et al. Injection sclerotherapy-induced esophageal strictures. Risk factors and prognosis. Am Surg 1991;57: 567-71; discussion 571-2.
46. Seewald S, Ang TL, Gotoda T, et al. Total endoscopic resection of Barrett esophagus. Endoscopy 2008;40:1016-20.
47. Lipp A, Lusardi G. Systemic antimicrobial prophylaxis for percutaneous endoscopic gastrostomy. Cochrane Database Syst Rev 2006;(4): CD005571.
48. Cotton PB, Connor P, Rawls E, et al. Infection after ERCP, and antibiotic prophylaxis: a sequential quality-improvement approach over 11 years. Gastrointest Endosc 2008;67:471-5.
49. Bangarulingam SY, Gossard AA, Petersen BT, et al. Complications of endoscopic retrograde cholangiopancreatography in primary sclerosing cholangitis. Am J Gastroenterol 2009;104:855-60.
50. Ertugrul I, Yuksel I, Parlak E, et al. Risk factors for endoscopic retrograde cholangiopancreatography-related cholangitis: a prospective study. Turk J Gastroenterol 2009;20:116-21.
51. Loperfido S, Angelini G, Benedetti G, et al. Major early complications from diagnostic and therapeutic ERCP: a prospective multicenter study comment]. Gastrointest Endosc 1998;48:1-10.
52. Cotton PB, Garrow DA, Gallagher J, et al. Risk factors for complications after ERCP: a multivariate analysis of 11,497 procedures over 12 years. Gastrointest Endosc 2009;70:80-8.
53. Freeman ML, Nelson DB, Sherman S, et al. Complications of endoscopic biliary sphincterotomy. N Engl J Med 1996;335:909-18.

54. Hussain N, Alsulaiman R, Burtin P, et al. The safety of endoscopic sphincterotomy in patients receiving antiplatelet agents: a case-control study. Aliment Pharmacol Ther 2007;25:579-84.
55. Freeman ML, DiSario JA, Nelson DB, et al. Risk factors for post-ERCP pancreatitis: a prospective, multicenter study. Gastrointest Endosc 2001;54:425-34.
56. Williams EJ, Taylor S, Fairclough P, et al. Risk factors for complication following ERCP; results of a large-scale, prospective multicenter study. Endoscopy 2007;39:793-801.
57. Wang P, Li ZS, Liu F, et al. Risk factors for ERCP-related complications: a prospective multicenter study. Am J Gastroenterol 2009;104:31-40.
58. Cheng CL, Sherman S, Watkins JL, et al. Risk factors for post-ERCP pancreatitis: a prospective multicenter study. Am J Gastroenterol 2006; 101:139-47.
59. Bailey AA, Bourke MJ, Kaffes AJ, et al. Needle-knife sphincterotomy: factors predicting its use and the relationship with post-ERCP pancreatitis (with video). Gastrointest Endosc 2010;71:266-71.
60. Romagnuolo J, Hilsden R, Sandha GS, et al. Allopurinol to prevent pancreatitis after endoscopic retrograde cholangiopancreatography (ERCP): a randomized placebo-controlled trial. Clin Gastroenterol Hepatol 2008;6:465-71.
61. Ho KY, Montes H, Sossenheimer MJ, et al. Features that may predict hospital admission following outpatient therapeutic ERCP. Gastrointest Endosc 1999;49:587-92.
62. Cheon YK, Cho KB, Watkins JL, et al. Frequency and severity of post-ERCP pancreatitis correlated with extent of pancreatic ductal opacification. Gastrointest Endosc 2007;65:385-93.
63. Andriulli A, Forlano R, Napolitano G, et al. Pancreatic duct stents in the prophylaxis of pancreatic damage after endoscopic retrograde cholangiopancreatography: a systematic analysis of benefits and associated risks. Digestion 2007;75:156-63.
64. Fazel A, Quadri A, Catalano MF, et al. Does a pancreatic duct stent prevent post-ERCP pancreatitis? A prospective randomized study. Gastrointest Endosc 2003;57:291-4.
65. Singh P, Das A, Isenberg G, et al. Does prophylactic pancreatic stent placement reduce the risk of post-ERCP acute pancreatitis? A meta-analysis of controlled trials. Gastrointest Endosc 2004;60:544-50.
66. Cennamo V, Fuccio L, Zagari RM, et al. Can early precut implementation reduce endoscopic retrograde cholangiopancreatography-related complication risk? Meta-analysis of randomized controlled trials. Endoscopy 2010;42:381-8.
67. Gong B, Hao L, Bie L, et al. Does precut technique improve selective bile duct cannulation or increase post-ERCP pancreatitis rate? A meta-analysis of randomized controlled trials. Surg Endosc 2010;24:2670-80.
68. Eloubeidi MA, Jhala D, Chhieng DC, et al. Yield of endoscopic ultrasound-guided fine-needle aspiration biopsy in patients with suspected pancreatic carcinoma. Cancer 2003;99:285-92.
69. Barawi M, Gottlieb K, Cunha B, et al. A prospective evaluation of the incidence of bacteremia associated with EUS-guided fine-needle aspiration. Gastrointest Endosc 2001;53:189-92.
70. Levy MJ, Jondal ML, Clain J, et al. Preliminary experience with an EUS-guided trucut biopsy needle compared with EUS-guided FNA. Gastrointest Endosc 2003;57:101-6.
71. Janssen J, Konig K, Knop-Hammad V, et al. Frequency of bacteremia after linear EUS of the upper GI tract with and without FNA. Gastrointest Endosc 2004;59:339-44.
72. Williams DB, Sahai AV, Aabakken L, et al. Endoscopic ultrasound guided fine needle aspiration biopsy: a large single centre experience. Gut 1999;44:720-6.
73. O'Toole D, Palazzo L, Arotcarena R, et al. Assessment of complications of EUS-guided fine-needle aspiration. Gastrointest Endosc 2001;53: 470-4.
74. Lee LS, Saltzman JR, Bounds BC, et al. EUS-guided fine needle aspiration of pancreatic cysts: a retrospective analysis of complications and their predictors. Clin Gastroenterol Hepatol 2005;3:231-6.
75. Wildi SM, Hoda RS, Fickling W, et al. Diagnosis of benign cysts of the mediastinum: the role and risks of EUS and FNA. Gastrointest Endosc 2003;58:362-8.
76. Ryan AG, Zamvar V, Roberts SA. Iatrogenic candidal infection of a mediastinal foregut cyst following endoscopic ultrasound-guided fine-needle aspiration. Endoscopy 2002;34:838-9.
77. Storch I, Shah M, Thurer R, et al. Endoscopic ultrasound-guided fine-needle aspiration and Trucut biopsy in thoracic lesions: when tissue is the issue. Surg Endosc 2008;22:86-90.
78. Diehl DL, Cheruvattath R, Facktor MA, et al. Infection after endoscopic ultrasound-guided aspiration of mediastinal cysts. Interact Cardiovasc Thorac Surg 2010;10:338-40.
79. Fais S, Kaplan R, Hawes RH, et al. Role of endoscopic ultrasound with guided fine needle aspiration (EUS-FNA) in the diagnosis of pelvic masses [abstract]. Gastrointest Endosc 2005;61:AB276.
80. Schwartz DA, Harewood GC, Wiersema MJ. EUS for rectal disease. Gastrointest Endosc 2002;56:100-9.
81. Hoffman BJ. EUS-guided celiac plexus block/neurolysis. Gastrointest Endosc 2002;56(4 Suppl):S26-8.
82. Gress F, Schmitt C, Sherman S, et al. Endoscopic ultrasound-guided celiac plexus block for managing abdominal pain associated with chronic pancreatitis: a prospective single center experience. Am J Gastroenterol 2001;96:409-16.
83. Gress F, Schmitt C, Sherman S, et al. A prospective randomized comparison of endoscopic ultrasound- and computed tomography-guided celiac plexus block for managing chronic pancreatitis pain. Am J Gastroenterol 1999;94:900-5.
84. Levy MJ, Wiersema MJ. EUS-guided celiac plexus neurolysis and celiac plexus block. Gastrointest Endosc 2003;57:923-30.
85. Voss M, Hammel P, Molas G, et al. Value of endoscopic ultrasound guided fine needle aspiration biopsy in the diagnosis of solid pancreatic masses. Gut 2000;46:244-9.
86. Gress FG, Hawes RH, Savides TJ, et al. Endoscopic ultrasound-guided fine-needle aspiration biopsy using linear array and radial scanning endosonography. Gastrointest Endosc 1997;45:243-50.
87. Bournet B, Migueres I, Delacroix M, et al. Early morbidity of endoscopic ultrasound: 13 years' experience at a referral center. Endoscopy 2006; 38:349-54.
88. Eloubeidi MA, Tamhane A, Varadarajulu S, et al. Frequency of major complications after EUS-guided FNA of solid pancreatic masses: a prospective evaluation. Gastrointest Endosc 2006;63:622-9.
89. Tournoy KG, Burgers SA, Annema JT, et al. Transesophageal endoscopic ultrasound with fine needle aspiration in the preoperative staging of malignant pleural mesothelioma. Clin Cancer Res 2008;14: 6259-63.
90. Das A, Sivak MV Jr, Chak A. Cervical esophageal perforation during EUS: a national survey. Gastrointest Endosc 2001;53:599-602.
91. Gress F, Michael H, Gelrud D, et al. EUS-guided fine-needle aspiration of the pancreas: evaluation of pancreatitis as a complication. Gastrointest Endosc 2002;56:864-7.
92. Adler DG, Jacobson BC, Davila RE, et al. ASGE guideline: complications of EUS. Gastrointest Endosc 2005;61:8-12.
93. DeWitt J, McGreevy K, LeBlanc J, et al. EUS-guided Trucut biopsy of suspected nonfocal chronic pancreatitis. Gastrointest Endosc 2005;62: 76-84.
94. Al-Haddad M, Gill KR, Raimondo M, et al. Safety and efficacy of cytology brushings versus standard fine-needle aspiration in evaluating cystic pancreatic lesions: a controlled study. Endoscopy 2010;42:127-32.
95. Jacobson BC, Waxman I, Parmar K, et al. Endoscopic ultrasound-guided gallbladder bile aspiration in idiopathic pancreatitis carries a significant risk of bile peritonitis. Pancreatology 2002;2:26-9.
96. Varadarajulu S, Hoffman BJ, Hawes RH, et al. EUS-guided FNA of lung masses adjacent to or abutting the esophagus after unrevealing CT-guided biopsy or bronchoscopy. Gastrointest Endosc 2004;60:293-7.
97. Mai CM, Wen CC, Wen SH, et al. Iatrogenic colonic perforation by colonoscopy: a fatal complication for patients with a high anesthetic risk. Int J Colorectal Dis 2010;25:449-54.

98. Imai N, Takeda K, Kuzuya T, et al. High incidence of colonic perforation during colonoscopy in hemodialysis patients with end-stage renal disease. Clin Gastroenterol Hepatol 2010;8:55-9.
99. Dellon ES, Gibbs WB, Rubinas TC, et al. Esophageal dilation in eosinophilic esophagitis: safety and predictors of clinical response and complications. Gastrointest Endosc 2010;71:706-12.
100. Weinberg BM, Shindy W, Lo S. Endoscopic balloon sphincter dilation (sphincteroplasty) versus sphincterotomy for common bile duct stones. Cochrane Database Syst Rev 2006;(4):CD004890.
101. Sanders MK, Moser AJ, Khalid A, et al. EUS-guided fiducial placement for stereotactic body radiotherapy in locally advanced and recurrent pancreatic cancer. Gastrointest Endosc 2010;71:1178-84.