

Terminal Ileal Photography or Biopsy to Verify Total Colonoscopy

Various modalities exist to document the extent of colonoscopy, including terminal ileum (TI) biopsy, which is considered the criterion standard by some authority. ASGE Guidelines concerning quality indicators for colonoscopy suggested visualization and photo documentation of the cecum to be performed in all colonoscopies. However, the ASGE concedes that "cecal photography may not be convincing in all cases."

The reliability of cecal landmarks to document the extent of colonic examination is corroborated by perfected studies.

A TI biopsy at the procedure is potentially hazardous and the detection of pathology in routinely acquired biopsy specimens of a macroscopically normal TI is limited. A safer, less costly alternative for documenting total colonoscopy was considered desirable.

To evaluate the effectiveness of TI photography in this circumstance, and to assess the diagnostic yield of TI biopsy in patients with a macroscopically normal TI, a prospective observational study was carried out in a district general hospital in the United Kingdom.

Two hundred thirty-two unselected patients undergoing colonoscopy, TI intubation, photography and biopsy were carried out with independent, experienced endoscopists, stating whether villi were "definitely, probably or definitely not" depicted in TI photographs. The diagnostic yield of specimens from macroscopically normal TI was determined.

Reviewers agreed that villi were "definitely present" in 96.8%, "probably present" in 5.9%, and "definitely not present" in 0.3% of cases with excellent interobserver agreement. TI photographs definitely depicting villi (93.8%) did not differ significantly from histology confirming TI mucosa (96.1%). Microscopic evidence of histology was only detectable in 2.3% of patients with an endoscopically normal TI.

It was concluded that TI photography is an effective, safe and cost effective means of documenting

total colonoscopy and that routine biopsy of the normal TI has a low diagnostic yield. (Powell M, Hayee BH, Yeo HZPK, et al. "Terminal Ileal Photography or Biopsy to Verify Total Colonoscopy: Does the Endoscope Agree With the Microscope?" *Gastrointest Endosc*, 2007; Vol. 66: 320-325.)

Diagnostic Yield of EUS-Guided FNA in Solid Pancreatic Masses

The diagnostic yield of EUS-FNA of solid pancreatic masses is a potential benchmark for EUS-FNA quality, because the majority of solid pancreatic masses should be diagnostic for malignancy. To determine the cytologic diagnostic rate of malignancy and to determine if variability exists among endoscopists and centers in evaluation of pancreatic masses, a multicenter, retrospective study was carried out with cytologic reports for all EUS-FNAs of solid, noncystic lesions, greater or equal to 10mm in diameter, solid pancreatic masses during a one year period. The main outcome measurement was cytologic malignancy.

A total of 1,075 patients underwent EUS-FNA at 21 centers. The median number of EUS-FNA of solid pancreatic masses performed during the year per center was 46 and per endoscopist, was 19. The mean mass dimensions were 32 × 27 mm with 73% located in the head. The mean number of passes was 3.5. Of the centers, 90% used a median cytologic evaluation. The overall diagnostic rate of malignancy was 71% with 5% suspicious for malignancy, 6% atypical cells and 18% negative for malignancy. The median diagnostic rate per center was 78%, first cortile 61% and per endoscopist 75% (first cortile 52%).

It was concluded in this retrospective study with participation bias and varying chronic pancreatitis prevalence, EUS-FNA cytology was diagnostic in malignancy in 71% of solid pancreatic masses. Endoscopists with a final cytologic diagnostic rate of malignancy for EUS-FNA of solid masses of less than 52% and in the lowest cortile, should evaluate reasons for their low yield. (Savides TJ, Donohue N, Hunt G, et al. "EUS-Guided FNA Diagnostic Yield of Malignancy in Solid Pancreatic Masses: A Benchmark for Quality Performance Management." *Gastrointest Endosc*, 2007; Vol. 66: 277-282.)

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Endotherapy for a Large Gastric Stoma After Bariatric Surgery

To determine the effect of endoscopic injection by using a sclerosant (sodium morrhuate) to induce stomal stenosis in patients who present with stomal dilation complicated by weight gain, 28 patients after bariatric surgery with GI bypass were referred with weight gain after initial weight loss. The weight gain was believed to be the result of a large gastric stoma.

Treatment included injection of sodium morrhuate (1 to 2 mL circumferentially), surrounding the stoma. A total of one-to-three injection sessions were performed in an attempt to achieve a stoma diameter of 1.2 cm. or smaller. Treatment success was defined as a decrease in stoma size to that level and a weight loss equal to or greater than 75% of the weight the patient gained after establishing a steady state post bariatric surgery weight.

Successful endotherapy was achieved in 18 of 28 patients (64%). One patient developed symptoms of stomal stenosis which required two separate balloon dilation sessions. No other complications were encountered.

In this retrospective case series, it was concluded that endoscopic injection of sodium morrhuate surrounding the dilated gastric stoma complicating bariatric surgery appeared to be a successful, less invasive therapeutic alternative to surgical revision. (Catalano MS, Rudic G, Anderson AJ, Chua TY. "Weight Gain After Bariatric Surgery as a Result of a Large Gastric Stoma: Endotherapy With Sodium Morrhuate May Prevent the Need for Surgical Revision." *Gastrointest Endosc*, 2007; Vol. 66: 240-245.)

Hepatic Venous Pressure Gradient (HVPG) in Evaluation of Compensated Cirrhosis

Two hundred thirteen patients with compensated cirrhosis and portal hypertension, but without varices, were included in a trial evaluating the use of B-blockers preventing varices. All had baseline laboratory tests and HVPG. They were followed prospectively every three months until development of varices, or variceal hemorrhage (VH), or end of the study.

Medical records were reviewed. Patients who underwent liver transplantation without decompensa-

tion wereensored at transplantation. Cox regression models were developed to identify the predictors of clinical decompensation. Receiver operating characteristic (ROC) curves were constructed to evaluate diagnostic capacity of HVPG.

Median follow-up time was 51.1 months. Sixty-two of 213 patients developed compensation (29%). Forty-six (21.6%) developed ascites. Six (3%) developed VH, 17 developed hepatic encephalopathy (8%). Ten patients received a transplant and 12 died without clinical decompensation.

Median HVPG at baseline was 11 mmHg. On multivariate analysis, three predictors of decompensation were identified. HVPG (Hazard ratio 1.11), MELD (HR 1.15) and albumin (HR 0.37). Diagnostic capacity of HVPG was greater for MELD or Child-Pugh Score.

It was concluded that HVPG, MELD and albumin independently predict clinical decompensation in patients with decompensated cirrhosis in patients with an HVPG less than 10 mmHg and a 90% probability of not developing clinical decompensation in a median follow-up of four years. (Ripoll T, Groszmann R, Garcia-Tsao G, et al, and the Portal Hypotension Collaborative Group. "Hepatic Venous Pressure Gradient Predicts Clinical Decompensation in Patients with Compensated Cirrhosis." *Gastroenterology*, 2007; Vol. 133: 481-488.)

Feasibility Trial of Narrow Band Imaging Endoscopy in GERD

Narrow band imaging (NBI) endoscopy system enhances visualization of microvasculature and mucosal patterns. In order to assess the utility of NBI in patients with GERD symptoms, patients with and without symptoms completed two validated GERD questionnaires prior to enrollment. The distal esophagus was examined by standard white light endoscopy, followed by NBI. Features seen only by NBI were compared between GERD patients and controls.

Eighty patients (50 GERD, 30 controls), were finally analyzed. A significantly higher proportion of patients with GERD had increased number (OR 12.6), dilation (OR 20), tortuosity of intracapillary loops (IPCLs)- OR 6.9, presence of microerosions, and increased vascularity at the squamocolumnar junction (OR 9.3), compared with controls.

On multivariate analysis, increased number (OR 5.5) and dilatation (OR 11.3), of IPCLs were the best predictors for diagnosis of GERD. The maximum, minimum and average number of IPCL yields were significantly greater in the GERD group, compared with controls. Although the interobserver agreement for the various NBI findings was very good, the intraobserver agreement was modest.

It was concluded that NBI endoscopy may represent a significant improvement over standard endoscopy with a diagnosis of GERD. Future prospective controls in blinded GERD trials will be required. (Sharma P, Wani S, Bansil A, et al. "A Feasibility Trial of Narrow Band Imaging Endoscopy in Patients with Gastroesophageal Reflux Disease." *Gastroenterology*, 2007; Vol. 133: 454-464.)

Mucosal Healing in IBD and Its Significance

In order to examine the possible predictors of mucosal healing and the impact of healing on subsequent course of disease in inflammatory bowel disease, 740 incident patients diagnosed with ulcerative colitis (UC) or Crohn's disease (CD) between 1990 and 1994, with no biologic therapy available, were recorded in reference to demographics and symptoms. Clinical and endoscopic evaluations were done at baseline before treatment and repeated after one and five years in 495 patients.

In UC patients, education longer than 12 years and extensive disease and diagnosis were significant pre-

dictors of mucosa healing (MH) after one year. MH was significantly associated with the low risk of future colectomy. In patients with CD, fever at diagnosis and medical treatment without steroids were significant predictors for MH. MH was significantly associated with less inflammation after five years and decreased future steroid treatment.

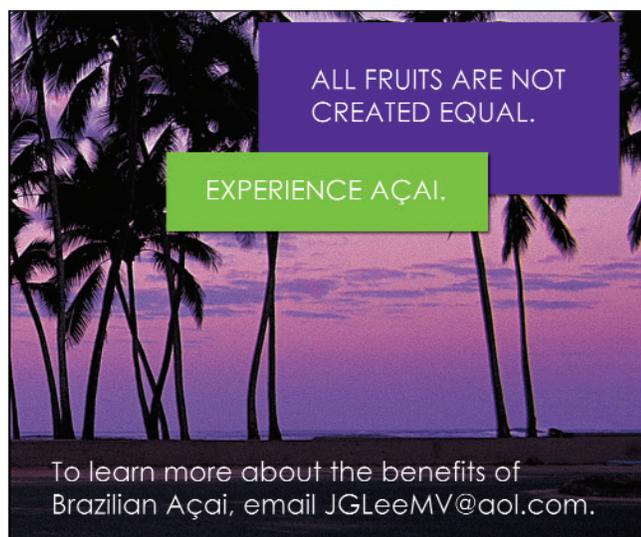
It was concluded that several factors predicted subsequent MH. Education as a predictor may implicate the importance of coping, compliance or lifestyle. MH after one year of treatment is predictive of reduced subsequent disease activity and decreased need for active treatment. Present results give further strength to the use of mucosal healing as a clinical indicator and treatment goal in inflammatory bowel disease. (Froslie KF, Jahnsen J, Moum BA, Vatn MH, and the IPSEN GROUP. "Mucosal Healing in Inflammatory Bowel Disease: Results from a Norwegian, Population-Based Cohort." *Gastroenterology*, 2007; Vol. 133, 412-422.)

Central Adiposity and Barrett's Esophagus

A case-controlled study investigating body mass index (BMI), central adiposity and cigarette smoking was carried out in reference to the risk of Barrett's esophagus (BE). Patients newly diagnosed with specialized intestinal metaplasia on at least one of four esophageal biopsy specimens taken at Community Gastroenterology Clinic (N = 193), were compared with match population controls (N = 211). Case subgroups included those with any visible columnar epithelium and those with at least 2 cm. of that epithelium (long segment BE/LSBE). Interviewers conducted personal interviews and took anthropometric measurements.

All measurements of central adiposity were strongly related to BE risk, particularly for LSBE. For the high category of waist to hip ratio (WHR), the adjusted odds ratios were 2.4 for all cases, 2.8 for visible BE, and 4.3 for LSBE. In contrast, the associations with BMI were weaker. When BMI and WHR were modeled simultaneously, the associations with BMI were greatly attenuated, whereas those with WHR remained strong. Further adjustments for frequency of heartburn did not change these results. Cigarette smoking moderately increased risk, but with no evidence of

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a dose-dependent response for increasing strength by case group.

It was concluded that these observations indicate the importance of identifying the mechanisms underlying obesity's role in BE and esophageal adenocarcinoma suggests that weight loss might be a fruitful approach to the prevention of these diseases. (Edelstein ZR, Farrow DC, Bronner MP, Rosen SM, Vaughan TL. "Central Adiposity and Risk of Barrett's Esophagus." *Gastroenterology*, 2007; Vol. 133: 403-411.)

Statins and NSAIDs in Colorectal Cancer Risk

A nested case-controlled study was conducted within 454 general practices in the United Kingdom, using the QRESEARCH database. Cases of colorectal cancer were diagnosed between 1995 and 2005. The effects of statins, nonsteroidal anti-inflammatory drugs, cyclooxygenase-II inhibitor and aspirin on colorectal cancer were estimated with conditional logistic regression adjusted for morbidity, smoking status, body mass index and socioeconomic status.

Five thousand six hundred and eighty-six cases and 24,982 matched controls with four or more years of records were analyzed. The adjusted odds ratio for colorectal cancer associated with any statin prescription was 0.93, with no trend in duration of use or number of prescriptions. For any nonsteroidal anti-inflammatory drug prescription, the adjusted odds ratio was 0.94 with a significant decrease in risk with the increasing number of prescriptions and adjusted odds ratio of 0.76, for greater or equal to 25 prescriptions. Prolonged use of Cox-II inhibitors was minimal, but for those receiving 25 or more prescriptions, the adjusted odds ratio was 0.34. Results were similar in the subset of participants with eight or more years of records. The adjusted odds ratio for greater or equal to 61 months of statin prescriptions was 1.00.

It was concluded in this large, population-based, case-controlled study that prolonged use of NSAIDs and Cox-II inhibitors was associated with a reduced colorectal cancer risk, but prolonged statin use was not. (Vinogradova Y, Hippisley-Cox J, Couplan DC, Logan R. "Risks of Colorectal Cancer in Patients Prescribed Statins, Nonsteroidal Anti-inflammatory Drugs, and Cyclooxygenase-II Inhibitors: Nested, Case-Controlled Study." *Gastroenterology*, 2007; Vol. 133: 393-402.)

Findings Suggesting Crohn's Disease in Diagnosed Ulcerative Colitis

Some patients diagnosed with UC undergo a change in diagnosis to CD. Predictors of diagnostic change could potentially impact the management of patients with chronic inflammatory bowel disease. In order to characterize the clinical and serologic predictors of change in diagnosis from UC to CD, a nested, case-controlled study was performed to compare individuals with a change in diagnosis from UC to CD, with age-matched UC and CD controls.

Primary analysis compared cases with UC controls. Subjects underwent chart review for clinical "red flags," identified by gastroenterologists with expertise in IBD. Serum collected at the time of database enrollment was tested for antibodies (ASC), pseudomonas fluorescens-related protein, *E. coli* outer membrane porin (CBIir1 flagellin), and pANP.

Twenty-one cases, 52 UC controls and 56 CD controls were assessed. Three red flags, but no serologic markers differed between cases and UC controls. At initial colonoscopy, cases were more likely to have extensive colonic involvement than UC controls. Multivariate regression identified non-bloody diarrhea at initial presentation and weight loss (greater than 10 percent) at presentation, as independent predictors of diagnostic change. Serologic markers did not add to the contribution of these two clinical factors in predicting a change in diagnosis from UC to CD.

Diagnostic change was evident in 6 of 6 patients with both predictors compared, with 8 of 50 with neither of these factors.

It was concluded that patients with diagnosis of UC with initial non-bloody diarrhea or weight loss have an increased likelihood of subsequent change in diagnosis to CD and might thus warrant further diagnostic workup. (Melmed GY, Elashoff R, Chen GC, et al. "Predicting a Change in Diagnosis from Ulcerative Colitis to Crohn's Disease: A Nested, Case-Control Study." *Clin Gastroenterol Hepatol*, 2007; Vol. 5: 602-608.)

Murray H. Cohen, D.O., editor of "From the Literature" is a member of the Editorial Board of *Practical Gastroenterology*.

Oligoclonal T-Cells in Biliary Atresia

Biliary atresia is one of the most common causes of liver transplant in infancy, and its cause remains unknown. One theory of its etiology is a viral-induced T-cell reaction with oligoclonal expansion leading to inflammation and fibrosis of the biliary epithelium. The authors of this elegant study evaluated for similar T-cell receptor (TCR) variable regions in patients with biliary atresia, indicating oligoclonal infiltration of the liver and biliary structures.

Over a two-year study period, peripheral blood mononuclear cells, liver tissue, and bile duct remnant tissue were collected from six infants with biliary atresia. Similar tissue was collected from six pediatric patients with other causes of cholestasis, which would serve as a control group. Monoclonal antibodies were used for immunofluorescence and cell sorting in order to identify TCR variable regions, and gene expression was determined in all patients. Liver tissue from patients with biliary atresia had a larger number of T-cells compared to the control patients. The majority of immune cells in the livers of biliary atresia patients were CD3⁺, and no such change was seen in blood lymphocytes and biliary tissue. Significantly more CD8⁺ T-cells were seen in the liver of biliary atresia patients, and significantly more CD4⁺ T-cells were seen in the bile duct remnant tissue of patients with biliary atresia. Interestingly, the TCR variable regions of the CD4⁺ and CD8⁺ T-cells in the biliary atresia patients were limited to a small number of variable regions, suggesting oligoclonal expansion. Since oligoclonal expansion generally is seen in certain antigenic stimulations (especially autoimmune disease and infections), this study points to evidence of a specific antigenic stimulation. Recent studies have suggested a viral process, possibly Rotavirus, leading to fibrosis and obliteration in biliary atresia, and this study adds to the evidence of an infectious cause of biliary atresia.

(Mack C, Falta M, Sullivan A, Karrer F, Sokol R, Freed B, Fontenot A. "Oligoclonal Expansions of CD4⁺ and CD8⁺ T-Cells in the Target Organ of Patients with Biliary Atresia." *Gastroenterology*, 2007; Vol. 133: 278-287).

Oral Tacrolimus for Ulcerative Colitis in Children

There are limited medical options for children with steroid-dependent and steroid-resistant ulcerative colitis, and colectomy is often necessary in this population. This study evaluated oral tacrolimus usage for children with steroid-dependent and steroid-resistant ulcerative colitis. A retrospective analysis was performed on 18 pediatric patients with ulcerative colitis who were unable to be weaned from steroids or had no response to five days of intravenous steroid therapy after presenting with bloody diarrhea. The average age of the patients was 11.5 ± .3 years, and 56% of the patients were female. Tacrolimus was started at an initial dose of 0.2 milligrams per kilograms divided twice daily, with a trough goal of 7-12 nanograms per milliliter. All patients received trimethoprim-sulfamethoxazole prophylaxis.

The study showed that 17 patients (94%) responded to oral tacrolimus with a mean time of 8.5 ± 6.7 days before a clinical response was noted. Of these patients, 89% of the steroid-resistant patients and 100% of the steroid-dependent patients responded to oral tacrolimus for a mean length of 260 days. However, 11 patients (60%) eventually required colectomy.

This study suggests that tacrolimus may be an effective therapy in preventing colectomy in difficult-to-treat ulcerative colitis patients; however, as with many pediatric studies involving high-risk patients, the sample size is small, and the data is retrospective. Further studies are needed to verify these findings. (Ziring D, Wu S, Mow W, Martin M, Mehra M, Ament M. "Oral Tacrolimus for Steroid-Dependent and Steroid-Resistant Ulcerative Colitis in Children." *J Ped Gastroenterol Nutr*, 2007; Vol. 45: 306-311).

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Liver Disease in Children, 3rd edition

Suchy FJ, Sokol RJ, Balistreri WF, Eds
 Cambridge University Press, New York, NY, 2007
 ISBN: 978-0-521-85657-7; \$185

Over the last decade, pediatric hepatology has evolved to become a distinct subspecialty within the field of Pediatric Gastroenterology with specialized areas of study even within this field. In 2007 the American Board of Pediatrics, offered the first board certifying examination that provided board certified pediatric gastroenterologists an opportunity to obtain a certificate of added qualification in pediatric transplant hepatology. It is possible, and in fact likely, that in the future additional certifying examinations may be required in other areas of special interest within pediatric gastroenterology including pediatric inflammatory bowel disease, pediatric endoscopy, and pediatric gastrointestinal motility testing among others.

The text review of *Liver Disease in Children* is one of a limited number devoted to the study of the pathogenesis, complications, and treatment of liver disease in pediatric patients. The multi-authored text has 41 chapters, is 993 pages in length, and is divided into five sections: pathophysiology, cholestatic liver disease, hepatitis and immune disorders, metabolic

liver disease, and other conditions. The last section includes a chapter on infections of the liver, hepatic tumors and liver transplantation.

The chapters on cystic fibrosis, Alagille syndrome, viral hepatitis, medical and nutritional management of the infant with cholestasis and drug induced liver disease are particularly well written and easy to read. The chapter on medication-induced liver disease would benefit from some additional tables and an expanded discussion of the effects of anti-tumor agents on the pediatric liver, as this is a frequent reason for consultation from the pediatric oncology service. There is some duplication of material between chapters, and some important topics such as hepatic transplantation are allocated only a relatively small percentage of space while others such as disorders of bile acid synthesis and metabolism and mitochondrial hepatopathies although well written receive a disproportionate amount of the text based on the relative frequency of these conditions. With the increasing complexity of pediatric liver transplantation, this topic warrants a larger fraction of the text in a subsequent edition and should be a distinct section of the text with chapters devoted to issues in immunosuppression, surgical options, post-transplant management and perhaps a chapter on combined hepatic and small intestinal transplant. Additional space should also be devoted to the topic of autoimmune hepatitis based on relative disease prevalence and importance of this condition in pediatric patients.

The text is accompanied by an excellent selection of color plates on a diverse number of topics, and is generally well illustrated throughout. The text would also benefit from the addition of images of newer imaging techniques such as magnetic resonance cholangiopancreatography (MRCP) in the chapter on sclerosing cholangitis among others. The authors are to be congratulated for tapping the writing skills of some of the foremost experts in pediatric hepatology for the text. With future editions of the text an increased emphasis on liver transplantation and a strong clinical focus will make this text even more valuable to pediatric and adult gastroenterologists and to those in training.

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BOOK REVIEWS

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GI Epidemiology

Talley NJ, Locke GR III, and Saito YA, Editors
Blackwell Publishing, Inc, 2007
ISBN: 9781405149495; \$99.95

The editors' purpose for this book is to establish a field of epidemiology of gastrointestinal [GI] diseases. The previous, and apparently only attempt, at such an effort was a book published in 1979 entitled *The Epidemiology of Chronic Digestive Disease*, written by M. J. S. Langman while he was Professor of Therapeutics at the University of Nottingham Medical School. Lacking a subsequent book of similar character the editors of the present book undertook to focus and expand interest and awareness of epidemiology for current practices in the fields of gastroenterology. Their attempt is commendable and worthy of attention not only by established gastroenterologists but also by all clinicians and clinical investigators of the future in gastroenterology and related fields of medicine and surgery.

As presented in its three parts, this publication is, in effect, three related books within one cover. Part 1, *Gastrointestinal Diseases and Disorders: The Public Health Perspective*, provides the rationale in its two chapters for interests in the importance and burden of diseases within the scope of gastroenterology. The next 14 brief chapters in Part 2, *Methodological Issues in GI Epidemiology*, address essential techniques for conducting epidemiologic studies in the field of gastroenterology. Part 3, *Epidemiology of Specific GI Diseases*, offers 19 chapters that address specific diseases and disorders prevalent in the field of practice

for gastroenterology. Throughout the book the chapters consolidate current information about knowledge of the clinical problem or research technique based on extensive reviews of the published medical literature. Each chapter is written concisely and clearly.

I found Part 3 of particular interest because each chapter had a useful summary about important developments over about the last three decades. Such summaries would provide current practitioners of gastroenterology useful reminders applicable to their practices. Certainly persons preparing to take examinations in internal medicine or gastroenterology would find the information useful. The book intends to encourage future investigators in public health and gastroenterology by providing helpful guides and suggestions about significant clinical problems that need to be addressed using the tools of epidemiology in several ways.

The editors have achieved their purposes of assembling information to support future developments in epidemiology for the field of gastroenterology. In the process they have compiled a valuable, concise reference book for broadening understanding of the public health elements of gastrointestinal diseases. Now the editors must wait to see if their promotion of interests and research will activate epidemiologists for gastrointestinal diseases.

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Help Wanted: Doctors and Nurses; PricewaterhouseCoopers Calls the Current Medical Workforce Model Broken in New Report On Health Care Staffing Shortage

There are more doctors and nurses today than ever before, but they are not being trained, distributed or deployed efficiently, according to PricewaterhouseCoopers' Health Research Institute in an analysis of the changing medical workforce and how it will affect the quality and delivery of health care in coming years. According to PwC, a majority of doctors and nurses is nearing retirement just as the American public will need them most, and health care organizations are left with a diminishing pipeline of primary care physicians, new competition for nurses and a generation of young clinicians who have different expectations about work-life balance than their predecessors.

The federal government is projecting a shortage of one million nurses and 24,000 doctors in the U.S. by 2020, but the PwC report "What Works: Healing the Healthcare Staffing Shortage" asserts that these projections are built around a broken, dysfunctional medical workforce model. It calls for major changes in the way doctors and nurses are trained, formation of public-private partnerships to promote and redeploy physician and nursing programs and new thinking about how, where and by whom health care will be delivered in the future.

Highlights of PwC's analysis of the changing medical workforce include:

- The total number of registered nurses has increased by 75 percent since 1980, but will begin to decline in 2010, the first decrease in decades. The absolute number of physicians also has increased steadily over the years, but there are serious maldistributions of physicians by specialty and geography. Only 20 percent of internal medicine residents are now choosing primary care internal medicine, and the rest are pursuing higher-paying subspecialties. Meanwhile, 20 percent of Americans live in areas with a shortage of primary medical care.
- The roles of nurses and physicians are blurring in primary care. According to PwC's research, hospitals in the U.S. now rely more heavily on physician extenders, such as nurse practitioners and physician assistants. Competition for these clinicians is

increasing, particularly with the advent of retail health clinics, which heavily employ physician extenders. By 2009, 1,500 retail clinics staffed by registered nurses are scheduled to be open.

- Hospital nurses will be elevated in stature from "overhead" to "rainmaker." Whereas physicians have traditionally been the revenue rainmakers in hospitals while nurses were considered overhead, new pay-for-performance programs that focus on clinical quality and patient satisfaction will give nurses significant ability to influence the measures that drive revenue.
- Technology is shifting what is done and by whom. Radiologists are now doing work that cardiologists used to do, and cardiologists are now replacing surgeons in some procedures as more people choose less-invasive treatments such as stents instead of coronary bypasses.
- Physicians are moving toward hospital employment while nurses are moving away. Two-thirds of hospital executives surveyed recently by PwC said their physicians now want to be employed by the hospital and nearly 75 percent say physicians are asking for on-call pay. On the other hand, the percentage of nurses working in hospitals has been dropping steadily. Nurse vacancy rates are running between 7 percent and 10 percent, and hospitals are now regularly turning to temporary workers to fill nursing shoes.
- International recruitment has been filling the gaps in the U.S. medical workforce. In 2005, approximately 13 percent of all new licensed nurses in the U.S. and 25 percent of all practicing physicians were international recruits.

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