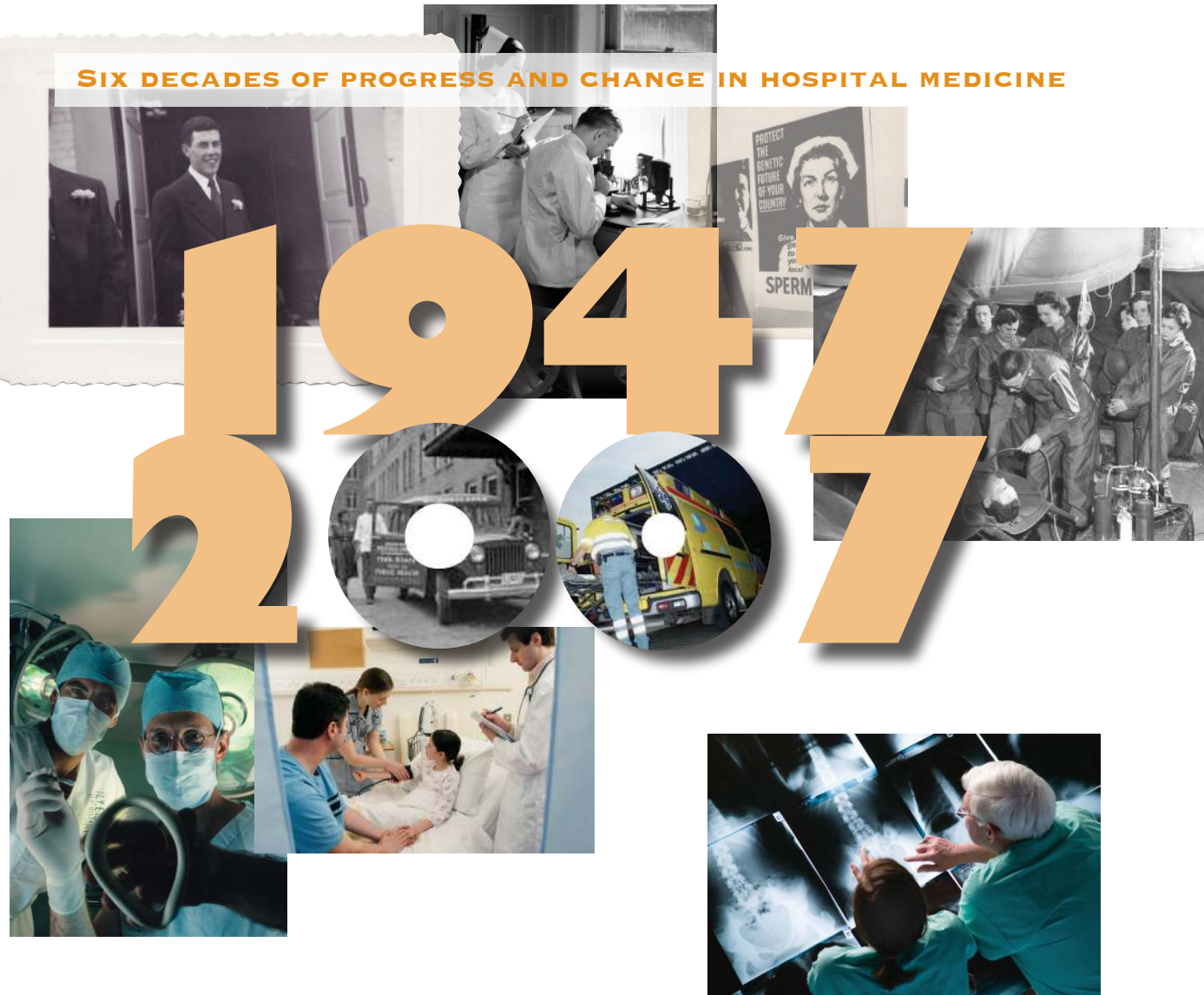


SIX DECADES OF PROGRESS AND CHANGE IN HOSPITAL MEDICINE



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The past sixty years have brought about extraordinary progress and innovation in the fields of medical science and biomedical technology. High-resolution imaging with computed tomography and magnetic resonance, echocardiography, flexible endoscopies, increasingly potent antimicrobial agents, pharmaceutical design based on genetics and molecular biology, interventional radiology, organ transplantation, coronary and intensive care units, computerized records, and access via the Internet to an ever-expanding collection of information are examples. We have also faced unanticipated

challenges, some related to this great progress, including antibiotic resistance and skyrocketing health care costs. Some are unrelated, including the emergence of unforeseen pathogens such as human immunodeficiency virus (HIV) and avian influenza A (H₅N₁), and an escalating malpractice crisis.

Although much has been written about the progress and challenges over the past half-century, little of its enormous impact has been recorded by the generation of physicians who witnessed this medical revolution. Dr. Sleisenger is among the remaining teachers of those senior physicians who still practice and teach over a half-century after their medical residencies.

To illustrate this revolution, we describe the case histories of two patients with common medical diseases—myocardial infarction and gastrointestinal bleeding—and outline the sharp contrast between their hospital courses in 1947 and 2007. The management of the patients in 1947 accords with the protocol published in the 1947 Seventh Edition of the

Cecil-Loeb *Textbook of Medicine*,¹ the authoritative medical textbook of the time in the United States. We also consider how these changes in care have affected the education of residents and their relationships with their patients.

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**CASE HISTORIES—
ACUTE MYOCARDIAL INFARCTION**

Six hours before the ambulance arrived, a fifty-two-year-old man, with six months of transitory chest pressure associated with exercise, developed his familiar chest discomfort while reading the newspaper. It gradually increased in intensity, radiated to his left neck and shoulder, and was not relieved by an antacid. He is a heavy cigarette smoker, obese, and had hypertension.



His wife called for an ambulance, which transported him to the local emergency room. The ambulance had an oxygen tank and an attendant. In the emergency room, a four-lead electrocardiogram (three leads from the limbs, one from the anterior chest wall) showed ST-segment depressions in lead one and in the anterior chest wall lead. (Official reading of the EKG appeared on the following morning after the strip was “fixed and ironed.”) The physical exam showed no evidence of congestive heart failure.

With a working diagnosis of myocardial infarction, 10 mg of morphine sulfate was administered intramuscularly for pain and anxiety, along with oxygen by face mask. The patient was transferred to the open ward, with vital signs recorded at half-hour intervals. The medical team debated whether to administer atropine or aminophylline intravenously to prevent spasm of the coronary arteries, a reflex believed to attend cardiac ischemia. Ultimately both medicines were withheld and the patient’s pain was managed with intramuscular injections of morphine. Additional sedation was achieved with sodium phenobarbital.

While the second-year resident wrote orders, the intern drew blood for the admission laboratory work. After hours and on weekends, interns were responsible for analyzing the complete blood counts, sedimentation rates, urinalyses, and electrocardiograms, as well as typing and cross-matching blood for transfusions.

Admission orders for the man included a low residue diet to minimize gaseous distension. The nursing staff was specifically instructed to feed the patient for ten days while he remained recumbent or seated in bed. After two weeks he would be permitted to dangle his legs over the sides of the bed and

feed himself a regular diet.

Over the following twenty-four hours, the patient’s mild anxiety was well controlled with 15 mg of phenobarbital every six hours. Periodic lead two electrocardiograms began to show occasional runs of premature ventricular beats. Two hours after he was given 0.2 grams of quinidine by mouth to suppress the premature contractions, his heart rate suddenly accelerated to 170 beats per minute, and he reported a “pounding” in his chest and a sense of impending death. The lead two tracing revealed ventricular tachycardia. His blood pressure dropped to 115/70 mm Hg and respirations increased to twenty-four per minute. Intravenous quinidine had no effect on the tachycardia. Shortly thereafter, he developed rales at both lung bases and his neck veins became distended. His congestive heart failure was treated with intramuscular morphine and mercurhydrin (a mercurial diuretic). Tourniquets were applied to three limbs in rotating fashion.

Repeated attempts to break the tachycardia with intravenous injections of quinidine were unsuccessful. He subsequently lost consciousness. His lead two tracing showed ventricular fibrillation. Shortly thereafter he was pulseless. The EKG lead revealed no electrical activity, and his respirations ceased. An injection of epinephrine beneath the sternum into the ventricle had no effect. He was pronounced dead.

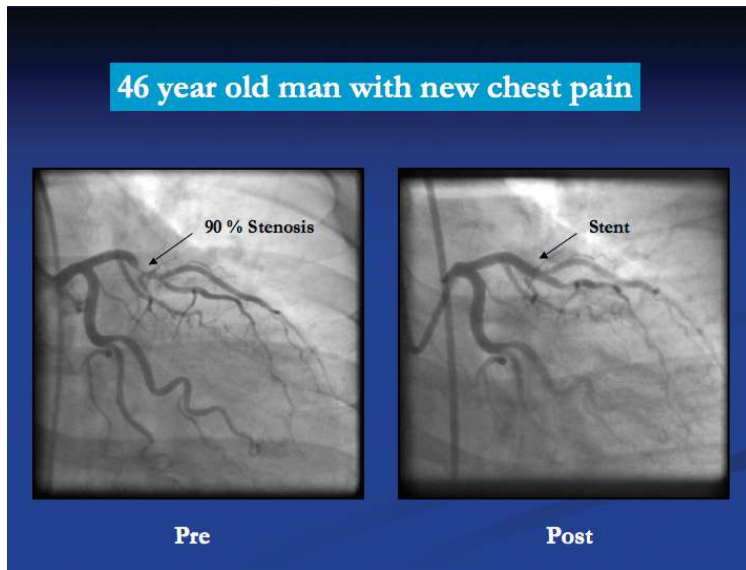


After a 911 call an ambulance with cardiac monitoring staffed by trained paramedics transports the patient to the nearest emergency room. Oxygen, aspirin, and sublingual nitroglycerin are given en route to the hospital. In the emergency department, the twelve-lead electrocardiogram reveals ST-segment depressions in the anterior leads. While being monitored on telemetry, an intravenous beta blocker, sublingual nitroglycerin, and intravenous morphine sulfate are administered, and his chest pain diminishes. The serum creatine kinase-MB and troponin-I are slightly elevated.

The patient is admitted to the coronary care unit with the diagnosis of acute coronary syndrome. His oral medications are aspirin, clopidogrel, metoprolol, and an HMG-CoA-reductase inhibitor. Low-molecular-weight heparin is given subcutaneously along with an intravenous glycoprotein-IIb/IIIa inhibitor. Intravenous nitroglycerin is given for recurrent chest pain.

Within one hour the patient becomes acutely short of breath and experiences severe substernal pressure. A rapid ventricular tachycardia is observed on the monitor, his blood pressure drops to 50/30 mm Hg, and he loses consciousness as the rhythm quickly degenerates into ventricular fibrillation. A code blue team arrives and resuscitates the patient using intravenous vasopressin, epinephrine, amiodarone, and direct electrical cardioversion.

The patient is immediately taken to the cardiac



Courtesy of Fred St. Goar, Director of Interventional Cardiology, Cardiovascular Institute, Mountain View, California.

catheterization laboratory, where a high-grade stenosis of the left anterior descending artery is visualized on angiography and treated with balloon angioplasty followed by deployment of a drug-eluting coronary stent.

A transthoracic echocardiogram done on the day of discharge demonstrates dyskinesis of the anterior wall and an ejection fraction of thirty percent. He is discharged on aspirin, clopidogrel, simvastatin, metoprolol, and lisinopril. Over the ensuing months his simvastatin dose is increased to bring his LDL cholesterol concentration below 70 mg/dL. Because of his coronary artery disease and low ejection fraction, he will receive an implantable automated cardioverter defibrillator to arrest potentially fatal arrhythmias.

**CASE HISTORIES—ACUTE
GASTROINTESTINAL BLEEDING**



A fifty-six-year-old man vomited an unknown volume of bright red blood twice during the previous twelve hours. Following the second episode he rapidly became weak, felt “faint,” and began to perspire profusely. He is a heavy drinker, drinking approximately one pint of vodka daily since his twenties, with considerably more on weekends. He smokes about fifty packs of cigarettes per year. (“I’d Walk a Mile for a Camel,” and “Your Doctor Recommends Camels” were two Reynolds Tobacco Company advertisements in 1947.)

In the emergency room, the patient appeared acutely ill, with a blood pressure of 100/50 mm Hg and a heart rate of 100 beats per minute. His sclerae were icteric. His abdomen was neither tender nor distended, but both his liver and spleen were enlarged. Neurological examination showed a gross tremor of his outstretched hands, but no asterixis. There was no evidence of focal weakness or cerebellar damage, although he had mild wasting of the muscles of both upper and lower extremities.

The intern drew blood for a complete blood count, type and cross-match, and blood chemistries (the latter were reported by the laboratory on the following morning). The patient’s hemoglobin was 7.5 gm/dL and hematocrit twenty-four percent; platelets appeared decreased in number on the blood smear.

Gastric lavage with cold normal saline through an Eder nasogastric tube returned coffee ground fluid. Lavage was continued until washings were clear.

Intravenous infusion of saline was administered via a #16 needle and sterilized rubber tubing. With a presumptive diagnosis of alcoholic cirrhosis and portal hypertension, esophageal or gastric varices were the suspected source. Two units of whole blood were administered. The patient’s blood pressure increased to 115/70 mm Hg, and his pulse rate decreased to 90 beats per minute. He was admitted to an open ward. The following morning during transport to the X-ray department for a barium study of his esophagus, he vomited a large amount of blood. His blood pressure fell to 85/50 mm Hg and his pulse rose to 120 beats per minute. Over the next hour two liters of normal saline were infused, followed by two additional units of whole blood. The patient became somnolent, restless, and increasingly diaphoretic as his temperature increased to 102°F (38.9°C). He also began hallucinating, seeing large insects crawling on the walls, floor, and bed. Intramuscular paraldehyde was given for alcohol withdrawal, and the Eder tube was again passed into his stomach with manual aspiration of a continuing stream of bright red blood. His pulse and blood pressure remained unchanged and his urine output diminished. After two additional units of whole blood were infused over four hours, the bleeding again stopped and his blood pressure stabilized at 95/50 mm Hg.

With apparent diminution of bleeding, a plan was made to have the surgical service follow closely for possible exploration of his upper abdomen and shunt procedure to decompress the portal venous system. The attending physician considered contacting Dr. Blakemore in New York for his new gastric tube that applied compression to a ruptured varix with an inflated balloon; however, it was thought very unlikely that the device could reach Boston in less than two days. Later that evening, the patient suddenly vomited a large amount of bright red blood and rapidly went into shock with his blood pressure falling to 50/30 mm Hg. He lost consciousness and died before he

could be transferred to the operating room.

Autopsy revealed a large ulcer in the antrum of the stomach, with an exposed eroded artery at its base, an engorged fatty liver, and splenomegaly. His wife provided additional history: the patient had experienced years of intermittent epigastric distress, at times relieved by sodium bicarbonate. He frequently took aspirin for “stress headaches” during which time he also increased his intake of alcohol.



The patient arrives in the emergency room and is monitored on telemetry. Two large-bore intravenous peripheral lines are established for rapid saline infusion. A nasogastric tube aspirate with bright red blood confirms ongoing upper gastrointestinal bleeding. Within an hour, basic laboratory tests results show the patient’s hemoglobin level is 7 mg/dL, his platelet count is 50,000, international normalized ratio for prothrombin activity is 1.6, and bilirubin and aminotransferases are elevated.

The patient is admitted to the intensive care unit, where packed red blood cells, platelets, fresh frozen plasma, and vitamin K are administered intravenously, along with continuous infusions of a proton pump inhibitor and octreotide. His blood pressure stabilizes at 110/70 mm Hg. The gastroenterologist performs a flexible esophagogastroduodenoscopy that reveals a 2.5 centimeter gastric ulcer in the antrum, with an actively bleeding vessel at its base. Epinephrine is injected around the

ulcer and a heated probe passed through the endoscope coagulates the vessel. The patient has no further bleeding over the next forty-eight hours, although plans are in place for a repeat endoscopy in the event of recurrent hemorrhage. (In the event of bleeding from the initially suspected cause—esophageal varices—he would have undergone variceal banding, and recurrent bleeding would have been treated with an emergent transjugular intrahepatic portosystemic shunt by the interventional radiology service.)

Antibodies to *Helicobacter pylori* are present in the serum. Omeprazole, clarithromycin, and amoxicillin are prescribed for two weeks. The patient is discharged after four days in the hospital. Alcohol cessation is advised and a referral is made to an alcohol rehabilitation program. A repeat esophagogastroduodenoscopy six weeks later rules out the possibility of gastric cancer. The ulcer has healed. The patient’s prescription for a proton pump inhibitor is continued indefinitely.

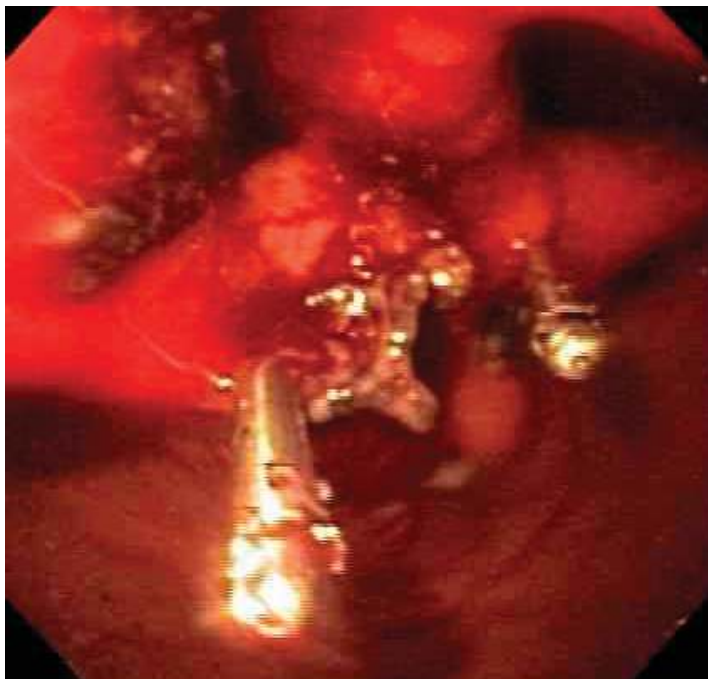


THEN AND NOW

Although these two case histories illustrate remarkably different outcomes in 1947 compared with 2007, we are not suggesting that all such patients as we have described would have had those outcomes. While very good care was provided in 1947 with relatively limited knowledge and resources, the in-hospital mortality for these diseases has significantly declined in subsequent decades.^{2,3,4}

Myocardial infarction

Diagnosis of myocardial infarction in 1947 depended on the patient’s history and the four-lead electrocardiogram, which often was sufficiently diagnostic, but also was falsely negative in patients with few or no relevant findings on the tracings. Not yet available was the measurement of aspartate aminotransferase (AST), which may be elevated in myocardial infarction, but the value of which is reduced by its elevation with hepatic vascular congestion or other diseases of the liver. Creatine phosphokinase (CPK) measurements were years in the future. The lead two tracing could reveal arrhythmias, but was connected only when symptoms or dramatic changes in pulse rate required it. Little pharmacological help existed for rhythm disturbance. Atropine was available for heart block, digoxin for atrial fibrillation, and quinidine for ventricular tachycardias, but often they were ineffective. The anatomical extent of coronary artery disease could not be determined, and there were no means to improve blood flow or to dissolve a thrombus in the course of an infarction. Coumadin as prophylaxis for “clot extensions” was used after the diagnosis was established,



A clipped ulcer after bleeding. Courtesy of Lauren B. Gerson, MD, MSc, Director, Esophageal and Small Bowel Disorders Center, Stanford University.

with questionable benefit and an unacceptable incidence of bleeding complications. In 1947 the principal components of management were absolute bed rest for ten days followed by restricted physical activity for some weeks after discharge, without dietary restrictions or medical regimens thereafter. Phenobarbital was sometimes prescribed for hypertension (if it was treated at all), but its efficacy was marginal at best.

Today the perspective on coronary artery disease—linked to genes, lifestyle, inflammation, and thrombosis—has changed as remarkably as have the accuracy of diagnosis and efficacy of treatment. The routine administration of aspirin and beta blockers, the monitoring of patients in coronary care units, and revascularization with thrombolysis, angioplasty, and stents have significantly decreased the mortality of this disease. Antiplatelet, antiarrhythmic, antihypertensive, and lipid-lowering agents are now the foundation of acute and long-term management. The advent of randomized, double-blinded, placebo-controlled clinical trials has contributed much to validating these therapies.^{5,6}

Gastrointestinal bleeding

The patient with acute upper gastrointestinal bleeding was always a puzzle in 1947, particularly if he or she had liver disease. The most common cause of upper gastrointestinal bleeding was, and remains, peptic ulcer disease. In patients thought to have a peptic ulcer, the standard therapy was to feed them milk and antacids as soon as they could tolerate this rather nauseating regimen. Some gastroenterologists even recommended continuous intragastric drips of milk, despite milk's known failure to buffer gastric acid.

Transfusions of whole blood were given for a fall in blood pressure or rise in pulse, or decreased hematocrit, which was done frequently. Packed red blood cell units and platelet or plasma concentrates were not available. Intravenous infusions were always via a peripheral vein and the largest bore needle was usually a #16. The needles were steel and, along with rubber tubing, were part of an "intravenous set" that was autoclaved after each use and saved for the next patient. The needles were periodically sharpened by hand, sometimes producing barbs that required discarding. Sudden fevers were common in the minutes after infusions began. A spike in temperature, often preceded by a shaking chill, mandated immediate withdrawal of the needle and replacement with a new needle and infusion set.

Accurate diagnosis and location of the bleeding lesion were nearly impossible. Occasionally, after cessation of bleeding, an upper gastrointestinal barium study might reveal an ulcer, varices, or a mass but could not prove the source of bleeding. Although guidelines for surgical intervention to stop the bleeding existed, based on patient age, number of units transfused, refractory hypotension or brisk hemorrhages, the decision to intervene largely depended on local experience

and availability of skilled surgeons.

The causes of upper gastrointestinal bleeding have not changed significantly over the past half century, but its management has been revolutionized. Understanding of peptic ulcer disease has been radically transformed by the discovery of *Helicobacter pylori* as an etiologic agent. A disease once thought to be mediated by "stress" stimulating acid production and managed with antacids, ulcer disease is now treated with highly effective antimicrobial agents along with potent inhibitors of acid production. The development of flexible fiberoptic endoscopy now permits rapid diagnosis and, often, treatment of the bleeding source. (Parenthetically, the increased ability to make diagnoses during illness coincides with an increasing inability to confirm diagnoses after death due to the decline in the number of autopsies that are performed in the twenty-first century, even in teaching hospitals.) End-stage liver disease with variceal bleeding still carries a high mortality rate, but banding of directly observed varices, the use of intravenous somatostatin analogues that decrease portal pressure, and the ability to connect the portal and systemic venous circulations have greatly enhanced our ability to halt profound hemorrhage.

Changes in medical education

These startling advances in medical diagnosis and treatment have affected both the tasks and duties of the medical resident, and thereby have significantly altered some aspects of the educational programs of trainees in internal medicine.

Although the basic formulae for teaching residents have remained constant over the past sixty years, dominated by daily resident rounds, attending rounds, medical grand rounds, morbidity and mortality conferences, morning report, and journal clubs, their formats and emphasis have changed substantially. For example, in 1947, daily rounds were conducted in a formal fashion at the bedside. The intern or clinical clerk presented patients with great emphasis on brevity, clarity, cogency, and sensitivity. After asking additional questions, the attending completely examined the patient. In 2007 attending physicians rarely perform complete physical examinations on rounds.⁷ Grand rounds presentations, formerly centered on a patient brought to the auditorium are now frequently didactic lectures.⁸ Morbidity and mortality conferences frequently do not focus on medical errors or adverse outcomes.^{9,10} Regrettably, each of these exercises are increasingly difficult for residents to attend given their increased duties and decreased work hours.

In 1947 house staff teams were on duty every other night and every other weekend. Residents were virtually omnipresent in the hospital (hence the name "resident"). With comparatively few diagnostic and therapeutic tools, and no limits on a patient's length of stay, the housestaff spent many hours at the bedside, examining, assessing, conversing with, and

thereby getting to know their patients. Continuity of care by the intern and assistant resident was both helpful and reassuring to patients.

During a typical hospitalization of 2007, the diagnostic and therapeutic process unfolds at a hectic pace, partly as a result of an explosive increase in available tests and interventions.¹¹ The inpatient physician is charged with executing the diagnostic and therapeutic plan, consulting subspecialists, coordinating the care with allied health professionals, and discussing diagnosis, care, and prognosis with the patient and family. In teaching hospitals these responsibilities often fall to the residents. Their innumerable daily tasks include collecting data from charts or computer screens and arranging consultations, procedures, imaging studies, and appointments. There is little time remaining in which to understand the social, familial, occupational, and psychological context of the illness. Concomitant with all these changes has been a logarithmic increase in the cost of hospitalization.¹²



It is particularly striking how few diagnostic and therapeutic options were available to physicians in 1947 compared with today. While the technological advances are obvious improvements, some of the most basic elements of everyday practice today were unimaginable over a half-century ago—a time when penicillin was just coming into widespread use and the double helix of deoxyribonucleic acid was yet to be discovered. Undoubtedly, sixty years from now, future generations of physicians will look back to medicine in 2007 and marvel at how primitive it was.

The improved outlook and prognosis of the patients we have discussed extends to many diseases. The progress has been astonishing. Everyone—trainees, physicians in practice, and most importantly patients—recognizes and are grateful for this progress. This change, however, has brought with it a nearly frenetic pace of activity that has discernibly altered aspects of resident-patient interactions.

In retrospect, our senior author, Dr. Sleisenger, asks himself: With such limited diagnostic tools and therapeutic armamentarium, what was he doing for a hundred or more hours each week in the hospital in 1947? The answer is that he spent many hours with patients, and learning from the attendings about how to take care of them. That educational environment has long given way to the exigencies of modern hospital practice, which, while affecting bedside teaching and residents' interpersonal relations with patients, must be accepted as an affordable price for the great advances made in diagnosis and care in these past six decades.

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References

1. Cecil RL, editor. *A Textbook of Medicine*. Seventh Edition. Philadelphia: W. B. Saunders; 1947.
2. Boersma E, Mercado N, Poldermans D, et al. Acute myocardial infarction. *Lancet* 2003; 361: 847–58.
3. Lewis JD, Bilker WB, Brensinger C, et al. Hospitalization and mortality rates from peptic ulcer disease and GI bleeding in the 1990s: Relationship to sales of nonsteroidal anti-inflammatory drugs and acid suppression medications. *Am J Gastroenterol* 2002; 97: 2540–49.
4. Higham J, Kang J-Y, Majeed A. Recent trends in admissions and mortality due to peptic ulcer in England: Increasing frequency of haemorrhage among older subjects. *Gut* 2002; 50: 460–64.
5. Antman EM, Armstrong PW, Anbe DT, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to revise the 1999 guidelines for the management of patients with acute myocardial infarction). *J Am Coll Cardiol* 2004; 44: E1–E211.
6. Braunwald E, Antman EM, Beasley JW, et al. ACC/AHA 2002 guideline update for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction: A report of the American College of Cardiology/American Heart Association task force on practice guidelines (Committee on the Management of Patients with Unstable Angina). *J Am Coll Cardiol* 2002; 40: 1366–74.
7. LaCombe MA. On bedside teaching. *Ann Intern Med* 1997; 126: 217–20.
8. Herbert RS, Wright SM. Re-examining the value of medical grand rounds. *Acad Med* 2003; 78: 1248–52.
9. Orlander JD, Barber TW, Fincke BG. The morbidity and mortality conference: The delicate nature of learning from error. *Acad Med* 2002; 77: 1001–06.
10. Pierlusissi E, Fischer MA, Campbell AR, Landefeld CS. Discussion of medical errors in morbidity and mortality conferences. *JAMA* 2003; 290: 2838–42.
11. Budetti PP. 10 years beyond the Health Security Act failure. *JAMA* 2004; 292: 2000–06.
12. Ginsburg PB. Controlling health care costs. *N Engl J Med* 2004; 351: 1591–93.

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