

Reflections

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Social distancing, no closer to each other than six feet, has become part of everyday life during the COVID-19 pandemic. This may be effective in a grocery store or restaurant, but is it appropriate between a doctor and patient? As a retired physician, I have seen the continuing evolutions in the practice of medicine from private to corporate business models, to hospital-based practice, to abbreviated visits and no-touch medical exams, to telemedicine. Many of these changes increase the distance between patient and doctor.

In the 1950s and 1960s, the patient-physician relationship could be called a close encounter. While on clerkships we were assigned a new patient in the delivery ward and remained in close contact throughout the admission. This included the initial history and physical examination, lab work, and attendance during labor. In those days, there were no fetal or maternal electronic monitors or ultrasound. The doctor performed the role of a human monitor, sitting at the patient's bedside with his hand on the patient's abdomen. Each contraction was felt and recorded noting timing and intensity. Fetal heartbeats were heard with a metal-framed fetoscope worn and attached securely to the physician's forehead. Bedside monitoring ended only after the delivery. Prolonged labors intensified this experience. Sometimes the doctor remained at the patient's bedside for two or

three days. After delivery there were daily post partum visits until discharge. This was intense, sometimes very exhausting, and required close contact with the patient. Many nights were spent in the on-call room next to the delivery suite.

Throughout my career I delivered more than 1,000 babies, and have forgotten the details of each admission. There was one exception, however, a close encounter that became a clinical highlight of my professional life.

It started as a routine obstetrics (OB) case. A young healthy mother with normal prenatal visits and a term pregnancy in early labor at a small community hos-

pital. Complications began in the labor room with vaginal bleeding with each contraction. The baby's head could not be felt on examination. Within the center of the dilated cervix was a soft bleeding mass of tissue. This was the placenta previa diagnosis we were told about during our OB clerkships. Clinical professors assured us that this was a rare condi-

tion, and we would probably never see it!

This urgent situation at 3 a.m. became even more serious without a blood bank or in-house laboratory technician. Also, I was the only physician in the hospital. A quick call was made with the nurse holding the phone up to my masked face. My surgical colleague was fortunately only 15 minutes away. We rushed to the operating room and prepared for an emergency Cesarean section. Within the next hour a healthy newborn, a joyful mother, and two very relieved physicians welcomed a new day.

Years passed, and I continued to see this patient and

SOCIAL DISTANCING



care for her growing family. We had not spoken about that long night until one day I asked her what she remembered about the event. She said most of the details had been forgotten.

She clearly remembered, "You never left my side."

Her words have given purpose to years of training, the medical profession, and my personal concern for patients.

While social distancing is recommended and can be implemented within the scope of our daily lives, many aspects of medical care, such as establishing a history, listening to patients' concerns, responding to their questions, and prescribing medications can be carefully controlled and monitored within the limitations of six feet. However, the complete doctor-patient relationship requires a close encounter that should not be restricted by any measurement.

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SARS, COVID-19, and other emerging diseases

Richard L. Bynny, MD, FACP

The COVID-19 global pandemic has in six months created a major health, social, economic, and financial crises throughout the world. It has nearly paralyzed the planet and resulted in monumental illness and mortality.

Over the past 100 years, there have been numerous pandemics, epidemics and outbreaks around the world – Asian influenza, Hong Kong influenza, Russian influenza, Avian influenza, Swine A (H₁N₁) influenza, HIV/AIDS, Ebola virus, SARS, MERS.

Most of the emerging diseases are infectious viral diseases often caused by RNA viruses that are zoonotic. Viral hemorrhagic fevers are a complex group of animal and human diseases from the Arenaviridae, Filoviridae, Bunyaviridae, Flaviviridae, and Rhabdoviridae families. Some result in relatively mild illnesses, but others can cause severe, life-threatening disease, and death.

Other viral infectious diseases include yellow fever, dengue, Marburg virus, and West Nile virus. Smallpox is the only viral infection that has been eradicated through vaccination and public health efforts.

In 1969, I was Chief Resident in Medicine at Columbia Presbyterian Hospital and one of my responsibilities was the management of the isolation ward at the hospital. In early March, I was called by an attending physician requesting an isolation bed for a patient he was transferring by air transport from Jos, Nigeria, via Lagos, Nigeria. The female patient arrived on March 4 and was admitted to

the isolation ward of the hospital. We had no equivalent of the modern intensive care unit, or any type of personal protective equipment (we improvised with surgical gowns, masks, gloves, and frequent hand washing). I evaluated the patient and instructed the isolation ward nurses that she was likely infectious, but that the cause of her illness was yet unknown.

In January 1969, there was a nurse, who was working in a small mission hospital in Lassa, Nigeria, when she became ill with a backache and severe sore throat, followed by difficulty swallowing with mucosal ulcerations and a fever of 100 degrees. She had been treated with antibiotics and chloroquine, but the oral lesions increased, her neck swelled, and she became dehydrated. Her fever increased and she developed slurred speech and drowsiness with dyspnea. On exam her lungs seemed clear, but she had pedal and facial edema, a macular and petechial rash, and a hemorrhagic lesion on her left elbow. She was flown to a missionary hospital in Jos. She was hypotensive with an irregular heart rhythm and thought to be in heart failure. She died after several convulsions. An autopsy revealed gastrointestinal mucosal bleeding, lung congestion, with a normal heart except for thickening of the mitral valve.

One of the hospital nurses who cared for the nurse in Lassa, reported she had used a gauze dressing on her finger to clear secretions from the first patient's mouth and realized she had a small cut on her finger. She subsequently developed a chill, headache, severe back and leg pain, and sore throat. She took antimalarial medication and was hospitalized the next day with a fever of 102 - 103 degrees, and occasional nausea. She was leukopenic with immature granulocytosis. Tests were negative, but she was treated with hydroxychloroquine. She developed pharyngitis, tender posterior cervical lymph nodes, and tenderness over her right costal margin, and then developed a macular rash that became generalized with some petechiae. She developed more lymphadenopathy, worse pharyngitis, rales with cough, and right pleuritic pain. She was intermittently clouded mentally. She developed 4+ proteinuria. She developed facial swelling, dyspnea, tachycardia, and distant heart sounds, rales, rhonchi, stridor, and wheezing. She had 53 percent immature neutrophils with a normal WBC count. She became cyanotic, hypotensive, and died on the 11th day of the illness. An autopsy revealed pleural and ascetic fluid, and dark mottling of the lung surfaces with lung congestion, necrotic areas with polymorphonuclear infiltrates, and cell debris, and intestinal and splenic congestion.

DEATH TOLL [HIGHEST TO LOWEST]

200M

Black Death (Bubonic Plague)
1347-1351



The plague originated in rats and spread to humans via infected fleas.

↑ The outbreak wiped out 30-50% of Europe's population. It took more than 200 years for the continent's population to recover.

56M

Smallpox
1520



↑ **Smallpox** killed an estimated 90% of Native Americans. In Europe during the 1800s, an estimated 400,000 people were being killed by smallpox annually. The first ever vaccine was created to ward off smallpox.

40-50M

Spanish Flu
1918-1919



30-50M

Plague of Justinian
541-542



↑ The death toll of this plague is still under debate as new evidence is uncovered, but many think it may have helped hasten the fall of the Roman Empire.



25-35M

HIV/AIDS
1981-PRESENT



12M

The Third Plague
1855



5M

Antonine Plague
165-180



3M

17th Century Great Plagues
1600



1.1M

Asian Flu
1957-1958



1M

Russian Flu
1889-1890



1M

Hong Kong Flu
1968-1970



1M

Cholera 6 outbreak
1817-1923

A series of **Cholera** outbreaks spread around the world in the 1800s killing millions of people. There is no solid consensus on death tolls. ↓



1M

Japanese Smallpox Epidemic
735-737



848K*

COVID-19

2019-10:28AM PT, AUG 31, 2020 [ONGOING]

*Johns Hopkins University estimates



600K

18th Century Great Plagues
1700



200K

Swine Flu
2009-2010



100-150K

Yellow Fever
LATE 1800s



11.3K

Ebola
2014-2016



850

MERS
2012-PRESENT



770

SARS
2002-2003

These numbers are current as of press time.

Courtesy of Visual Capitalist. <https://www.visualcapitalist.com/history-of-pandemics-deadliest/history-of-pandemics-deadliest/>

The patient we received at Columbia was a 52-year-old missionary nurse, who had taken care of both patients who died, and assisted at the last autopsy. On February 20, she developed a fever of 100 degrees and stayed in bed with malaise, headache, nausea, and fever, followed by sore throat. She was admitted to the hospital with a small ulceration on the right buccal mucosa and slight tonsillar reddening with tenderness of the epigastrium and right costal margin. She had a low white blood cell (WBC) count with high immature polymorphonuclear cells and lymphopenia. She was treated with antibiotics and antimalarial medications without improvement and was flown to Lagos, and isolated. She was then flown to Columbia Presbyterian Hospital with an attendant missionary nurse.

When I first took her history and examined her, I made sure everyone gowned and scrubbed in and out, a procedure that I insisted on throughout her hospitalization. Further examination revealed dehydration, paravertebral muscle tenderness, right upper quadrant tenderness, and she was lethargic but fully oriented. She developed inspiratory rales at the left lung base. She was treated with fluids and tetracycline.

We had no idea what illness she had, but given the history it appeared to be a communicable disease that we did not recognize. Some of the findings suggested a possible viral hemorrhagic fever unrecognizable from the clinical or epidemiologic information.

She was treated empirically for many illnesses common in Africa without response and had a very extensive laboratory and other diagnostic work up for then recognizable diseases. Her portable chest X-rays revealed changes consistent with a left lower lobe pneumonitis with small pleural effusion and subsequently bibasilar infiltrates. She continued to be extremely weak and lethargic with continued fevers.

On March 11, her fever decreased, though she continued having difficulty swallowing, abnormal eye movements, and EEG demonstrated findings compatible with a diffuse encephalopathy with no focal defects. She slowly recovered, and I am certain that her recovery was entirely the result of the incredible, thoughtful, supportive nursing care she received that allowed her own defense mechanisms and immune response to cure her.¹

Serum from all three cases and serum and fluid from a thoracentesis done on March 6 had been sent to the Yale Arbovirus Research Unit (YARU). On March 23, the YARU laboratory reported that all specimens had grown a cytopathogenic agent on tissue culture and maintained in passages in tissue culture and reacted with

complement-fixation antibodies from the serum drawn from the last patient on March 20. They proposed the new disease be called "Lassa fever," after the community where the first case probably originated. They speculated that it was not unlike lymphocytic choriomeningitis infection with clinical manifestations that are pansystemic.¹

In June 1969, J.C., a doctor at the Yale University Arbovirus Research Laboratory, who was working with specimens from the Lassa fever patients, developed shivering that resolved with subsequent episodes alleviated by taking aspirin. He then noted severe pain in the lower portion of both thighs more in the muscles than joints and without swelling of the joints, but he had difficulty walking because of the weakness and pain. He then had increased malaise and Dr. Ed Leifer (AQA, Columbia University Vagelos College of Physicians and Surgeons, 1945), an attending physician at Columbia made a house call and J.C. was admitted to the isolation unit at Presbyterian Hospital. He had a temperature of 104 degrees, appeared acutely ill and was flushed with mild quadriceps muscle tenderness. His platelets and WBCs were low, and he had 2+ proteinuria.

He remained acutely ill with fever, a few petechiae on his pharynx and buccal mucosa with small vesicles on the buccal mucosa that ulcerated and cleared. His SGOT, SGPT, CPK, and LDH were elevated. His illness seemed progressive and presumptively was the Lassa fever virus.

Dr. Gocke, the infectious disease specialist, recommended that we contact the recovered patient and obtain convalescent plasma since she had demonstrated high titer of antibody prior to discharge.

During the infusion of convalescent plasmas J.C.'s temperature fell and he began to improve. He became afebrile and was discharged and subsequently recovered. In turn, the Lassa fever virus was recovered from J.C.'s blood, urine, and throat washings. The administration of convalescent plasma was beneficial.²

In November 1969, J.C. returned to the viral laboratory to work. In December, one of his laboratory technicians traveled to Philadelphia and became sick. He was admitted to a Philadelphia hospital and died from Lassa fever without receiving convalescent plasma. J.C. hence suspended experiments with live Lassa fever virus.

Subsequently, there was an outbreak of illness similar to Lassa fever in West Africa with 26 suspected cases and 10 deaths. Thirteen of the cases were confirmed as Lassa fever by complement-fixation tests. Fifty-three household contacts, some with minor illness, were evaluated and four were positive for Lassa fever.³

Lassa fever virus is an RNA virus, in the family arenaviruses and persists in Western Africa, including Nigeria, Liberia, Sierra Leone, and Guinea. The virus infects a host rodent called *Mastomys natalensis*, where it lives symbiotically. The virus is carried in the rodent's blood, urine, and stool. Fortunately, this rodent is not common in other geographic regions.

It is estimated that 100,000 to 300,000 people are infected each year with Lassa fever, that results in a high mortality rate. When suspected and if available the diagnosis can be made with ELISA test for antigen and IgM antibodies with 88% sensitivity and 90% specificity. Ribavirin when used early in the illness may be beneficial. Isolation and quarantine, personal protective measures, and hand washing are recommended for prevention of infection.

In 1989, an engineer in Chicago went to Nigeria to attend a family funeral and was ill when he returned to the U.S. He was hospitalized for fever of unknown origin.⁴ The diagnosis was not made at the time and he died from Lassa fever virus. Fortunately, others were not infected. Since then, there have been other cases in New Jersey, Pennsylvania, and Minnesota, and cases have been reported in the United Kingdom, Netherlands, Israel, and Japan.

This outbreak is a vivid reminder of the threat and consequences of infectious disease epidemics. Today, we recognize new infectious disease because of successful scientific research and methods, and through public health services and surveillance. When an epidemic initially begins, the health care systems are often inadequate, and the public health response can be limited.

The continuously emerging infectious epidemic threats emphasize that as physicians and health care providers, we must continue to learn, understand, and utilize our leadership, clinical skills, wisdom, and care to rapidly and responsibly act and to meet our social responsibilities including support of public health. In addition, the most effective approach to an infectious epidemic is to be able to control it at the source which means effective public health and epidemiologic measures and improved supportive care for infected people and health care workers.

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