

# AMERICAN JOURNAL OF CLINICAL MEDICINE®

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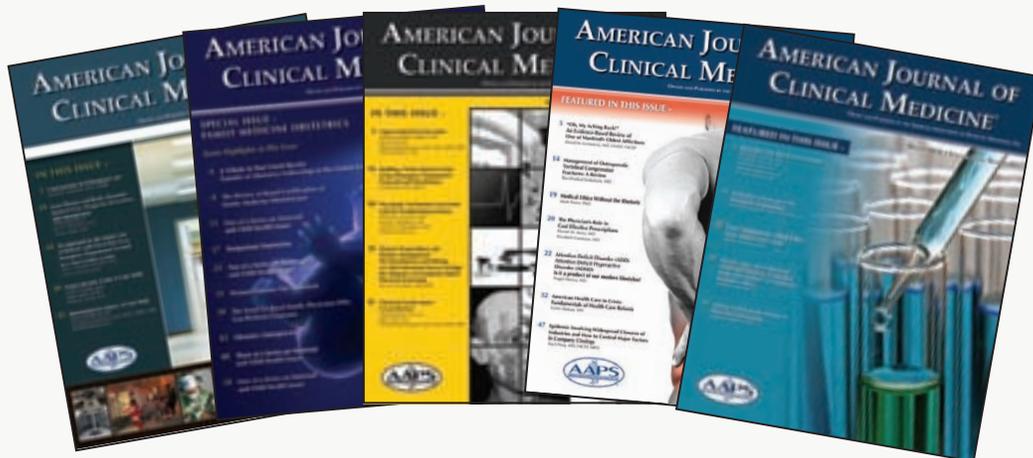
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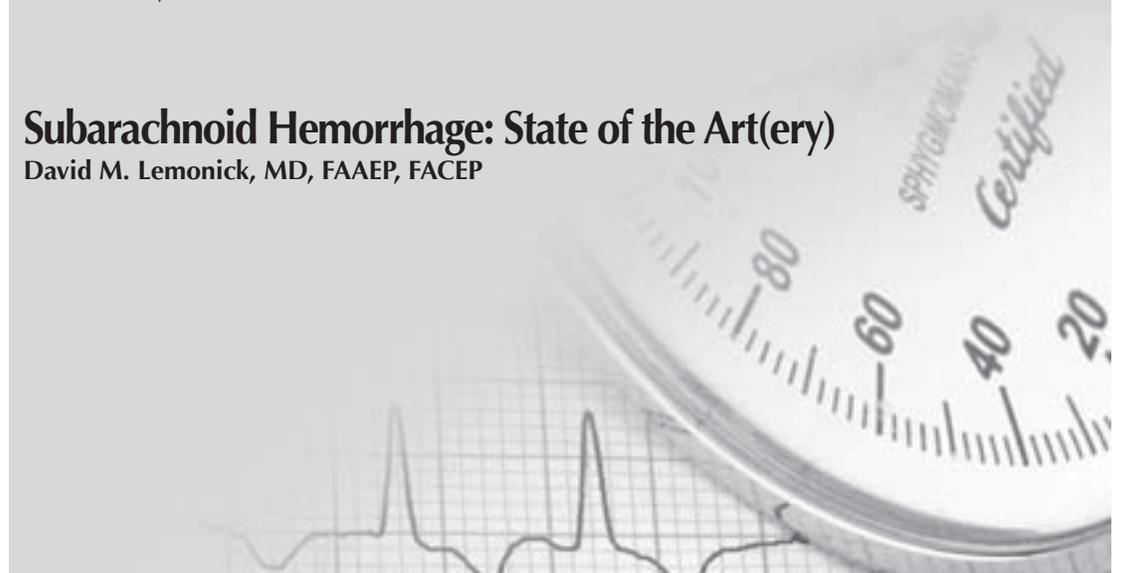
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Articles that appear in the *AJCM* are peer reviewed by members with expertise in their respective specialties. Manuscripts submitted for publication should follow the guidelines in The International Committee of Medical Journal Editors: "Uniform requirements for manuscripts submitted to biomedical journals" (*JAMA*, 1997; 277:927-934). Studies involving human subjects must adhere to the ethical principals of the Declaration of Helsinki, developed by the World Medical Association. By *AJCM* policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of their article that might create any potential conflict of interest. More detailed information is included in the *AJCM* Manuscript Criteria and Information on pages 96 and 97.

**WE WANT YOUR INPUT.** As always, we welcome your comments and opinions. In this issue, please note the Editorial Comment and the Letters to the Editor features on page 50. In addition, Sounding Board offers you the opportunity to express your views on a subject of particular interest to you. Recent topics have included such diverse subjects as autopsies, competency-based education in rural medicine, and medical effects of the Chernobyl nuclear disaster. Thank you also for your support of the Medical Ethics feature with your responses to the cases that have been presented.

As mentioned in the last issue, the *AJCM* has begun a regular series of clinically-focused cases using radiographic, ultrasound, and ECG images as a means of simulating clinical cases commonly used for competency assessment. These cases do not represent material taken from board examinations, which are confidential. We encourage you to submit these types of items as they will be of general interest to physicians among the AAPS specialties. These activities are excellent ways for you – our readers – to participate actively in the AAPS.

Wm. MacMillan Rodney, M.D., FAAFP, FACEP  
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## Editorial Comment

The review of Dengue Fever in this issue of the American Journal of Clinical Medicine® is very timely. Citizens from all over the world travel to the tropical islands just south of the United State, in the Caribbean Sea. To this number, add the number of North American citizens (United States and Canada) who have volunteered to help in the tragedy which recently occurred in Haiti. Then, considering the fact that Dengue Fever is a re-emerging infection, it is important for physicians to be familiar with this article's information when evaluating the febrile patient, especially in patients who may have recently traveled to southern states or the Caribbean.

Thomas G. Pelz, D.O.  
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## *In Memory of*

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**Victor Miranda, M.D.**

## Letters to the Editor

*The following letter was written in response to Sounding Board, Volume 7, Number One, Winter 2010, "Why Are Very Few Autopsies Performed Today?" by Daniel M. Avery, MD*

### Dear Sir:

It is extremely unfortunate that the number of autopsies being performed in hospitals throughout the U.S. has declined dramatically over the past three decades. Much of what we have come to learn about the pathology and physiology of the human body has been derived from post-mortem dissections and analyses since the 18th century.

Despite the sophistication of various new diagnostic technologies, several research studies have demonstrated that many disease processes cannot be accurately and universally detected. Hence, in the absence of a complete autopsy, we can reasonably conjecture that complete diagnoses are not being correctly and scientifically established during patients' lifetimes. The future implications of such benign ignorance are easily foreseeable, not to mention the various legal ramifications of mistaken or missed diagnoses in the case of a particular patient at the time of his or her death.

A very regrettable, albeit not publicly expressed, reason for this decline is the great fear that many physicians and hospitals have regarding potential medical malpractice lawsuits. After all, if an autopsy is not done, then the cause of death and all other relevant information pertaining to the patient's treatment and ultimate demise will be whatever the attending physician sets forth on the death certificate. This is a selfish, ostrich-like approach that cannot be defended morally or ethically. Such an attitude is potentially harmful to both the practice of medicine and the pursuit of justice in future years.

Very truly yours,

Cyril H. Wecht, M.D., J.D.

Corrections to Ethical, Legal and Professional Challenges Posed by "Controlled Medication Seekers" to Healthcare Providers – Part 1, by Ken Solis, M.D., MA:

(1) Acknowledgements: Arthur Thexton, J.D., for his legal advice on how Wisconsin State Privacy Law would conflict with national HIPAA laws and the expected consequences of that conflict; (2) Tom May, Ph.D., for critically reviewing the article and for his cogent insights and suggestions for improvements.

# Dengue Fever/Dengue Hemorrhagic Fever

Bruce R. Guerdan, MD, MPH

## Abstract

In September 2009 the first documented, locally acquired cases of Dengue Fever in eighty years were diagnosed in individuals living in or visiting Key West, Florida. Dengue Fever is a mosquito-borne viral illness found throughout the tropics. There are four serotypes of the disease, which is accepted as the most common arthropod-borne disease in the world. Dengue Hemorrhagic Fever and Dengue Shock Syndrome are clinical variants of this disease, which have significant morbidity and mortality. *Aedes aegypti* is the mosquito species which carries the disease. *A. aegypti* breeds exclusively in standing fresh water and bites humans exclusively. The disease has a large percentage of clinically asymptomatic cases. When symptomatic, common symptoms are frontal headache, fever, and severe musculoskeletal pain. There are no currently approved vaccines.

## Introduction

The first documented cases of locally acquired Dengue Fever (DF) in the Florida Keys in eighty years (forty years for the state of Florida) were diagnosed in the summer and fall of 2009.<sup>1</sup> This disease is commonly found in the tropics and is considered endemic to such vacation spots as Puerto Rico and the Virgin Islands. Also known as “breakbone fever,” due to the intense pain experienced by some patients, this illness is caused by a member of a family of viruses known as hemorrhagic fevers. Dengue Fever is a mosquito-borne (*Aedes aegypti* and rarely the *Aedes albopictus* species) disease with no apparent animal reservoir. The disease can also be transmitted via both blood transfusion and transplacentally during pregnancy. The initial case in the Florida Keys was found in a tourist who visited Key West and returned home to New York where she was diagnosed. The Centers for Disease Control and Prevention (CDC) deployed a team from its Dengue Branch in Puerto Rico to Key West in September 2009 after two additional cases in “locals”

were diagnosed. An extensive study of the local population was completed with the results showing a large number of IGM and IGG-positive individuals.

## History

Illnesses consistent with DF are well documented throughout history. Dr. Benjamin Rush first described “bilious remitting fever” in the modern medical literature during an outbreak in Philadelphia, PA. in 1780. His account noted the general population had named the illness “breakbone fever.” The word “dengue” may be derived from the Swahili “Ka-Dinga pepo,” which means a cramp-like seizure from an evil spirit.<sup>2</sup> The exact origin of the disease is in question. Both the U.S. and the Japanese militaries were impacted by DF during the Pacific Campaign of World War II. DF was rare in North America and the Caribbean basin after World War II due to aggressive spraying programs. DENV-1 (see description below) was first seen in Cuba in 1977. The first case of Dengue Hemorrhagic Fever seen in the Americas was in Cuba in 1981 when DENV-2 was introduced to the island. Dengue Hemorrhagic Fever (DHF) was first documented in 1953 in Manila, the Philippines.<sup>3</sup>

## Epidemiology

The disease Dengue Fever/Dengue Hemorrhagic Fever is caused by four closely related viruses (Flaviviridae family) DENV-1, DENV-2, DENV-3 and DENV-4. The four variants are indistinguishable clinically. The viruses are composed of a single strand of RNA, which is in the same genus as the Yellow Fever and West Nile viruses. Between 500,000 and one million individuals worldwide contract Dengue Fever/Dengue Hemorrhagic Fever annually, making it the most common arthropod-borne disease in the world.<sup>4</sup> The disease is very common to urban environments in the tropics, and it is estimated that tens of millions of U.S. travelers visit endemic

areas annually.<sup>5</sup> In the United States twenty-six states require the reporting of dengue infections. It is felt that competent mosquito vectors are located in twenty-eight of the fifty states.<sup>6</sup> There are two passive surveillance systems run by the CDC. One is compiled from state health departments, while the other, ArboNet, also accepts reports from private laboratories. As well, there is a disease specific surveillance system run by the World Health Organization (WHO), known as Denguenet, which tracks cases worldwide. Isolated outbreaks of DF have been seen along the Texas/Mexico border intermittently between 1986 and 2005, and a large outbreak, 122 cases, occurred in Hawaii in 2001-2002. The Hawaii outbreak included at least one autochthonous case.<sup>7</sup>

### Pathophysiology/Clinical Disease

The incubation period of DF in the mosquito is 8-12 days followed by an additional 3-14 days in the human host. This is followed by clinical disease lasting 3-7 days. The virus is easily passed from mature female mosquitoes to their offspring, and an infected mosquito is infectious throughout its lifespan. The levels of clinical dengue infection range from asymptomatic infection to critically ill patients with hemorrhage and shock. Case definitions are:

#### A. Asymptomatic or mild infection

Very common and by definition few or no symptoms besides fever

#### B. Dengue Fever (DF)

Fever and two or more of the below:

- a) Retro-orbital/ocular pain
- b) Headache
- c) Rash
- d) Myalgias
- e) Arthralgias
- f) Leukopenia
- g) Hemorrhagic symptoms not meeting the definition of DHF

#### C. Dengue Hemorrhagic Fever (DHF)

Meets criteria for Dengue Fever plus:

- a) Thrombocytopenia (less than 100,000 cells per mm<sup>2</sup>)
- b) Evidence of plasma leakage manifested by hemo-concentration (increase in hematocrit >20% of normal for age or a decrease < 20% after rehydration) or pleural effusion, ascites, or hypo-proteinemia.

#### D. Dengue Shock Syndrome (DSS)

Meets all of the criteria for DHF plus:

signs of shock; i.e., rapid pulse, narrow pulse pressure, and, most importantly, poor end organ perfusion.<sup>8</sup>

It is well established that individuals will develop antibody and lifelong immunity to the specific variant that they contracted as well as a transient immunity to all four of the variants. Only

when a second, third, or fourth infection (i.e., with a different variant) is contracted does the possibility of developing a clinical case of hemorrhagic disease occur. Transplacental transfer of maternal antibody can put an infant at risk for DHF.<sup>9</sup>

### Differential Diagnosis

As fever and headache are the primary symptoms seen in DF, similar illnesses will drive the differential diagnosis process. Meningitis, encephalitis, and sinusitis are some of the etiologies of fever and headache that must also be considered. The lack of respiratory symptoms, such as sore throat and cough, make illnesses such as influenza less likely. A history of travel to a tropical environ should be elicited. Currently, the lack of such travel should remove DF from the differential.

### Diagnostic Studies

DF is confirmed in reference laboratories using polymerase chain reaction (PCR) technology, which makes confirmation of the diagnosis during a case of clinical disease very unlikely. There is also PCR cross reactivity between the Dengue virus and several other similar organisms, such as West Nile Fever.<sup>10</sup> The cases found in Key West were all DENV-1. Again, this information, if and when available, will almost certainly be reported long after the resolution of clinical disease. Routine diagnostic studies, such as a complete blood count (CBC), coagulation studies, and serum chemistries, will be of little value in the diagnosis of the disease but important in the management of the more complex cases. As long as there is only one variant of the disease found locally and the patient has no prior history of DF (i.e., prior travel), the hemorrhagic forms should not be an issue. In locations, such as Asia, where all four variants are found, DHF or DSS are of real concern.

### Therapeutics

As with many viral illnesses, supportive measures are the cornerstone of therapy. In the more routine cases, analgesia may be the only therapeutic requirement. The uncommon cases of hemorrhagic disease may require more aggressive interventions, such as volume support, aggressive hemodynamic monitoring, and potentially transfusion of blood products including platelets.

### Public Health Interventions

The primary public health intervention regarding mosquito-borne disease is the reduction in mosquito breeding habitat. Monroe County, Florida, the home of Key West, has an extensive mosquito control apparatus run by a Mosquito Control Board. Aggressive spraying, both airborne and on land, with trucks has been accelerated. The geographic location of the cases in Key West is in an area known as Old Town. This area is covered by large trees and foliage typical of many tropical cities and is likely impervious to spraying. The reduction of standing

water, probably best through a public education program, will likely have the best outcome. As the *A aegypti* mosquito breeds solely in standing fresh water, an intervention that focuses on reduction of these breeding habitats has been shown to be the most successful. Programs, such as those seen in Cuba in the early 1980s and Singapore in the 1990s, which focused on paramilitary style neighborhood programs have been the most successful.<sup>11</sup> There are no currently approved vaccines for DF, although several are being investigated.

## Re-emerging Disease

The return of DF to the Florida Keys is another example of re-emerging diseases. DF has been described as one of the most common re-emerging infections.<sup>12</sup> It is currently unknown specifically how the disease re-established itself, but in a time of worldwide air travel, legal and illegal immigration, and international commerce, it should be no surprise that this has occurred. Key West is a mere ninety miles from Cuba, and it is not uncommon for "rafters" to land unannounced on its shores. It is possible the disease has actually been in the mosquito population in Key West and the Lower Keys for an extended timeframe. As stated above, much of the disease is asymptomatic or very mild and would therefore elude detection. The diagnosis of the tourist who returned home to New York may have opened an investigation into a disease not as new (to Florida) as might be originally thought. Primary care providers throughout the United States, Canada, and Europe should routinely include a travel history in all evaluations of a febrile patient.

## Acknowledgements

Special thanks to Mark Whiteside, MD, MPH, Medical Director, Monroe County (FL) Department of Health, for his assistance.

*Bruce R. Guerdan, MD, MPH, is board certified in Emergency Medicine, Family Medicine, and Disaster Medicine. He is an attending physician in the Emergency Department at Lower Keys Medical Center in Key West, FL, and serves in the Florida Air National Guard as the Florida State Air Surgeon.*

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# Successful Treatment of Bilateral Refractory Chylous Effusion of Non-Hodgkin Lymphoma Patient with Octreotide

Jennifer I-Hsuan Shen, MS  
Ajithkumar Puthillath, MD  
Lynn Steinbrenner, MD  
Michele Cook, MD  
Deborah Farolino, MD, FACP

## Abstract

Chylothorax is leakage of the thoracic duct content into the pleural cavity due to congenital defects, trauma, tumor infiltration, postoperative cardiac or esophageal procedures. Traditional approaches to chylothorax include thoracentesis and pleurodesis, which are invasive and often result in significant patient morbidity and mortality. In a patient who is a nonsurgical candidate, octreotide presents a better alternative. Octreotide, an analogue of somatostatin, was first used more than a decade ago to treat chylous effusions. Octreotide's treatment for lymphoma-induced chylothorax has rarely been reported. Through our literature search, we found that our case is among the few bilateral lymphoma-induced chylous effusion cases treated by octreotide in the world. Our patient was treated with octreotide and combined with medium chain triglyceride (MCT) with strict low fat/low calorie diet. After ten days of treatment, there was a significant reduction of chylous effusion. Patient was subsequently discharged and recovered significantly without recurrence for the past eight months.

**Keywords:** Lymphoma, chylothorax, octreotide, thoracentesis, Medium Chain Triglycerides (MCT).

## Case

The patient is a 64 year-old female with Stage II Non-Hodgkin Lymphoma involving mediastinum, thoracic T5 to T12, and adjacent GI wall. The patient was initially treated with 3000 cGy

radiation to thoracic spine followed by R-CHOP. Ten days after her first course of R-CHOP, she presented to the emergency room with severe dyspnea, pleuritic chest pain, and dry cough. Chest x-rays (Figure 1) showed extensive bilateral pleural effusion. Initial left thoracentesis revealed the presence of a chylous effusion, and subsequent analysis of the right effusion revealed the same. The triglyceride levels were 3397 mg/dl and 2218 mg/dl on the left and right side of the chest respectively.

**Figure 1:** Patient's chest x-ray upon admission



Following three days of continuous infusion octreotide at 24-50 mcg/hour and a low-fat diet supplemented with medium chain triglycerides (MCTs), patient had recurrence of bilateral chylothorax. The second set of bilateral thoracentesis showed triglyceride levels were 438 mg/dl and 441 mg/dl on the left and right side of her chest respectively. However, it represents an 80% and 87% decrease on the left and right sides of her chest accordingly since first set of bilateral thoracentesis. Re-accumulation of the chyle stopped after second set of bilateral thoracentesis, and she required no further pleural interventions. Chest X rays at two months showed complete resolution (Figure 2).

**Figure 2:** Chest x-ray two months after discharge



During her octreotide treatment, the patient experienced low-grade fever, minor hyperglycemia, and diarrhea for the first few days of octreotide initiation, which resolved after two days when octreotide was stopped accompanied by lab tests to investigate the origin of fever. No infectious etiology was delineated. Mild temperature elevated again after patient was started back on octreotide, and the temperature resided after a few days of treatment.

Dietary modification for our patient initially consisted of Enlive® and a clear liquid diet. She was transitioned to a low-fat (40 to 70 mg of fat per day) diet and medium chain triglycerides (MCTs) on day six, and she reported an improved mood and appetite. Total parenteral nutrition was not considered due to possible complications and patient's immune-suppressive state. On day 14 of inpatient stay, patient was discharged home on subcutaneous octreotide injection (.05 mg/ml). After one month of outpatient treatment, the MCT diet and octreotide were successfully discontinued, and there has been no recurrence of chylothorax since then.

## Discussion

Chyle is a white milky fluid that is rich in triglyceride, calories, vitamins, and immunoglobins.<sup>1,2</sup> Chyle is produced at a rate of from two to four liters per day and can increase to five liters per

day on a high-fat diet. Chyle drains into the venous system via the thoracic duct at the junction of the internal jugular and subclavian veins. Chylous fluid consists of greater than 30 gm/L of protein, 4-40 g/L of lipid (mostly triglycerides), and cells (primarily lymphocytes).<sup>2</sup>

Chylothorax is leakage of the thoracic duct contents into the pleural cavity due to congenital defects, trauma, tumor infiltration, and postoperative cardiac or esophageal procedures. Traditional approaches to chylothorax include thoracentesis and pleurodesis, which are invasive and often result in significant patient morbidity and mortality. Repetitive or continuous drainage of chylothoraces can cause loss of lymphocytes, immunoglobulins, and fats, which can further compromise the patient's immune status and lead to cachexia over a short period of time.

Approximately 60% of the cases are due to lymphoma. The diagnostic criteria vary, but the fluid is often milky appearing and the triglyceride content of the pleural fluid should be greater than 150.<sup>1</sup>

The mainstay in the management of chylothorax is to decrease the rate of flow of chyle. This can be done with surgery or medical management. Surgical intervention is indicated when chylous effusion accumulates at a rate that is more than 1500 ml per day for five to seven days.<sup>3</sup> Ligation at the site of lymphatic leakage is an option, but can involve an extensive thoracotomy and requires surgical expertise, and was not a good option for our patient due to her immunocompromised state and extensive mediastinal involvement. Other surgical techniques include pleurocentesis, pleuroperitoneal or pleurovenous shunt,<sup>4</sup> thoracoscopy suture, or thoracoscopy with fibrin glue application.<sup>5</sup> Less invasive method using pleurodesis with talc administration still requires usage of chest tube or a thoracoscope. There were articles reporting 58% of chylothorax patients resolving symptoms with radiation treatment;<sup>6</sup> however, our patient's chylothorax appeared after a course of radiation. All these methods, although used often in chylothorax cases, can cause significant morbidity and even mortality.

Dietary management is the key and should include restriction of long chain fatty acids and triglycerides, which are absorbed into the lymphatic system. Medium chain triglycerides (MCTs) are often recommended, because they are absorbed directly across the GI mucosa into the portal circulation.<sup>7</sup> Often parenteral nutrition is required, but should be reserved when there are no other options. With reduction in chyle flow, there is a reasonable chance that a spontaneous closure of the leak may occur, thus avoiding the morbidity and mortality associated with tube thoracotomy.<sup>8,9</sup>

Octreotide, an analogue of somatostatin, was first used more than a decade ago to treat chylous effusions. It can be administered intravenously, subcutaneously, or intramuscularly for both pediatric and adult chylothorax cases.<sup>8,9</sup> In a patient who is a nonsurgical candidate, octreotide presents a useful adjunct.<sup>1,10,11,12,13</sup> However, even though octreotide has been used clinically for over a decade, its usage for lymphoma-induced chylothorax has rarely been reported.

Through our literature search, we found that our case is among the few bilateral lymphoma-induced chylous effusion cases treated by octreotide in the world.<sup>14</sup> Our patient was treated with octreotide and combined with medium chain triglyceride (6 to 12 carbon chain triglycerides) with strict low fat/low calorie diet. After six days of treatment, there was a significant reduction of chylous effusion. Adverse effects were limited to minor hyperglycemia and flu-like symptoms when first initiated, but resolved within a week. After one month follow up, octreotide treatment stopped. The patient recovered significantly and resumed activities of daily living and has not had recurrence of chylothorax for eleven months since discharge.

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# Blood Component Therapy

Daniel M. Avery, Jr., MD

Kathy T. Avery, MT (AMT), RN

Blood products are living tissue and are used in patient care.<sup>1</sup> Blood components include whole blood itself, packed red blood cells, fresh frozen plasma, cryoprecipitate, platelets, granulocyte components, and Rhogam. Because whole blood and blood components come from human blood, there is a risk of infectious disease transmission despite meticulous screening. Blood components may contain small amounts of immunizing substances and other components. Because of risks, the need for transfusion should be specific.<sup>1</sup>

## Testing of Donated Blood

Donated blood is tested prior to distribution for transfusion.<sup>1</sup> Blood is typed including ABO and Rh type. Testing is also performed for Human Immunodeficiency Virus, Hepatitis C Virus, Human T-cell Lymphotropic Virus, Hepatitis B core Antigen, Hepatitis B Surface Antigen, HCV RNA, HIV-1 RNA, and syphilis.<sup>1</sup>

## Transfusion of Blood Products

Transfusion of whole blood and all blood components must be transfused through a filter designed to remove clots and aggregates in 0.9% Sodium Chloride without any other medications or solutions.<sup>1</sup> Lactated Ringer's or other solutions containing calcium should never be used with blood transfusions. Blood may be warmed prior to administration for exchange or massive transfusions or for patients with cold-reactive antibodies. The initial administration of blood is very slow should a life-threatening reaction occur.<sup>1</sup> Transfusion should be completed within four hours and prior to expiration of the blood product.<sup>1</sup>

## Immunologic Complications of Transfusion

Immunologic complications of transfusion may be immediate. In hemolytic transfusion reaction, transfused red blood

cells are destroyed by hemolysis due to incompatibility of antigen on transfused cells with antibody in the recipient's circulation. Immune-mediated platelet destruction occurs when alloantibodies destroy transfused platelets. Febrile nonhemolytic reactions occur with about 1% of transfusions.<sup>1</sup> This reaction is manifested by a temperature elevation of  $>1^{\circ}\text{C}$  or  $2^{\circ}\text{F}$  after transfusion without other cause of a fever. Such patients are often treated by leukocyte-reduced packed red blood cell transfusions.<sup>1</sup>

Allergic reactions usually occur with urticaria, wheezing, and angioedema.<sup>1</sup> Anaphylactoid reactions are dangerous complications of transfusion and consist of autonomic dysregulation, severe dyspnea, pulmonary and/or laryngeal edema, and bronchospasm.<sup>1</sup> Transfusion-related acute lung injury occurs with pulmonary leakage of fluid into the alveolar and interstitial spaces.<sup>1</sup>

Immunologic reactions may also be delayed. Delayed hemolytic reaction may occur. Alloimmunization to red blood cell antigens may occur days or weeks after transfusion. Post-transfusion purpura may cause sudden thrombocytopenia 7-10 days after transfusion. Graft-vs-host disease (GVHD) occurs when T lymphocytes react against tissue antigens in the recipient.<sup>1</sup>

## Non-Immunologic Complications of Transfusion

Non-immunologic complications include transmission of infectious disease, usually viruses such as CMV, bacterial contamination, and circulatory overload, leading to pulmonary edema, hypothermia and metabolic complications.<sup>1</sup>

## Whole Blood and Red Blood Cell-Containing Components

Red blood cells contain hemoglobin, which carries oxygen to tissues and increases the mass of circulating red blood cells.<sup>1</sup>

Red blood cell components are indicated for the treatment of symptomatic deficit of oxygen-carrying capacity and exchange transfusion.<sup>1</sup> Red cell transfusions should not be used to treat anemias that can be corrected with specific medications, such as iron, folate, and B12. Whole blood is rarely kept in hospital blood banks, because the collection process is more rigorous, the shelf life shorter, and the risk of reaction greater because of all of the components.

Whole blood contains red blood cells and plasma components of circulating blood. A single whole blood donation contains 450-500 mls of blood with a minimum hematocrit of 38%.<sup>1</sup> Shelf life of whole blood is 21-35 days, depending on the anticoagulant/preservative.<sup>1</sup> When the plasma is removed, red blood cells remain and have a hematocrit of 65-80% and a volume of 225-350 mls.<sup>1</sup> Additive solutions mixed with the red cells result in a hematocrit of 55-65% and a volume of 300-400 mls.<sup>1</sup>

Each unit of whole blood or packed red blood cells contains sufficient hemoglobin to raise the hemoglobin concentration in an average size adult by 1 g/dL and the hematocrit by three percentage points. The increase is less in an obese person.

Blood is typed and cross-matched prior to administration to make sure that it will be compatible. The only exception to this is when any delay in transfusion would be life threatening. O negative blood is the universal donor and may be given when there is no time for a type and cross-match. In life-threatening emergencies any blood type may have been given in hopes of saving the patient from exsanguination and dealing with transfusion reactions and complications if the patient survives. Rhogam may need to be given if a Rh negative patient receives Rh positive blood.

In life-threatening hemorrhage a patient may know his blood type, so that type-specific blood may be administered. An obstetric patient that has had prenatal care will have had a type, Rh, and antibody screen performed as part of initial prenatal laboratory studies. Type-specific blood could also be administered to this patient.

## Plasma Components

Fresh Frozen Plasma (FFP) is obtained from whole blood or apheresis and frozen at -18 degrees or colder. FFP contains functional amounts of Coagulation Factors V and VIII.<sup>1</sup> FFP is indicated in massive transfusion with coagulation deficiencies, active bleeding with patients on coumadin or who need to undergo an invasive procedure before Vitamin K could reverse the anticoagulant effect, transfusion or plasma exchange in patients with TTP, management of selected coagulation factor deficiencies when coagulation concentrates are unavailable, or management of rare specific plasma protein deficiencies.<sup>1</sup>

## Transfusion of Fresh Frozen Plasma

Fresh frozen plasma must be ABO compatible with the recipient's red blood cells, but compatibility testing is not necessary.<sup>1</sup>

Fresh Frozen Plasma contains all of the coagulation factors.<sup>1</sup> In massive blood loss and blood component replacement, one unit of FFP is transfused for each two to three units of packed red blood cells.

## Cryoprecipitate Components

Cryoprecipitate AHF is prepared by thawing FFP and recovering the precipitate. It contains coagulation Factor VIII, Factor XIII, Fibrinogen, vWF, and Fibronectin. Each unit of Cryoprecipitate AHF contains >80 IU Factor VIII and > 150 mg of fibrinogen.<sup>1</sup> Cryoprecipitate is indicated for therapy in von Willebrand disease, hemophilia A (Factor VIII deficiency), and bleeding associated with fibrinogen deficiency.<sup>1</sup>

## Administration of Cryoprecipitate

When cryoprecipitate is administered, ABO compatibility is preferred without consideration of Rh status. Compatibility testing is unnecessary. Thawed cryoprecipitate is given as soon as possible after thawing.<sup>1</sup> The cryoprecipitate is mixed well with 10-15 mls of 0.9% Sodium Chloride.

## Platelet Components

Platelets are essential for normal hemostasis and work through a series of reactions inducing platelet adherence to vessel walls and platelet activation leading to platelet aggregation and formation of a primary hemostatic plug.<sup>1</sup> Platelet transfusions are indicated for the treatment of bleeding associated with decreased numbers of platelets, usually less than 50,000.<sup>1</sup> Platelet transfusion is not indicated when the count is >100,000. Platelets are not effective in disorders which destroy circulating platelets, such as ITP.<sup>1</sup>

One unit of platelets is a concentrate of platelets separated from a single unit of whole blood in 40-70 mls of plasma containing  $5.5 \times 10^{10}$  to the tenth power platelets.<sup>1</sup>

## Transfusion of Platelets

Compatibility testing is not necessary when transfusing platelets.<sup>1</sup> One unit of platelets should increase the platelet count by 5-10,000 in a normal size adult and by 20,000 in an 18 kg child.<sup>1</sup> The administration dose is 4-8 units.<sup>1</sup> Platelets can be infused quickly but not in less than four hours.<sup>1</sup>

## Granulocyte Components

Granulocyte concentrates are collected by hemapheresis.<sup>1</sup> Granulocyte transfusion therapy is controversial.<sup>1</sup> The number of granulocytes in the concentrate is  $>1.0 \times 10^{10}$  to the tenth power. Infused granulocytes kill bacteria and fungi. A granulocyte infusion does not increase the patient's granulocyte count but is used to treat neutropenia (<500) in patients with infections who have not responded to antibiotics and in whom eventual bone

marrow recovery is expected.<sup>1</sup> Red blood cells leukocyte reduced or leukocyte poor red blood cells are used to decrease febrile nonhemolytic transfusion reactions.<sup>1</sup> Leukocyte reduced blood components may cause severe hypotension in patients, especially those on ACE inhibitors.<sup>1</sup>

## Transfusion of Granulocytes

Granulocytes should be transfused as soon as possible. The red blood cells in the infusion should be ABO compatible. Therapy should be continued daily until infection and fever are resolved, and the absolute granulocyte count returns to at least 500.<sup>1</sup> Because most patients receiving this transfusion are immunocompromised, granulocytes should be irradiated to prevent graft-versus-host disease.<sup>1</sup>

## Rhogam

Rhogam or Rh Immune Globulin is used to prevent the development of antibodies to red blood cell antigens following exposure to such antigens from another individual, such as a transfusion or pregnancy.<sup>2</sup> A person that lacks a specific red blood cell antigen can produce an antibody when exposed to it.<sup>3</sup> These antibodies can cross the placenta and hemolyze fetal red blood cells, leading to anemia.<sup>2</sup> Antibodies can also form with Kell, Duffy, and Kidd antigens. Anti-Lewis and Anti-I usually do not cause problems.<sup>2</sup> Anti-Kell is the most important non-Rh cause of hemolytic anemia.

Rh Immune Globulin is given to prevent active antibody response by passive immunization.<sup>2</sup> Rhogam only works for the D antigen. The standard dose is 300 micrograms, which will neutralize 15 mls of fetal red blood cells. The amount of Rhogam needed is determined by the Kleihauer-Betke Test.<sup>2</sup> Rhogam is usually given within 72 hours of delivery but can be given up to four weeks.

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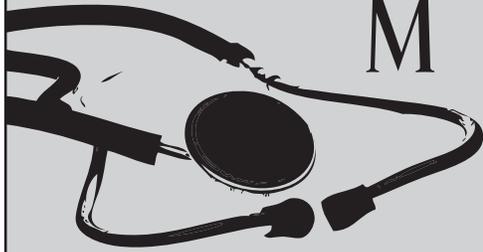
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# MEDICAL ETHICS WITHOUT THE RHETORIC

Cases presented here involve real physicians and patients. Unlike the cases in medical ethics textbooks, these cases seldom involve cloning, bizarre treatments, or stem cell research. We emphasize cases common to the practice of medicine.

Most cases are circumstantially unique and require the viewpoints of the practitioners and patients involved. For this reason, I solicit your input on the cases discussed here at [council@aol.com](mailto:council@aol.com). Reader perspectives along with my own viewpoint are published in the issue following each case presentation. We are also interested in cases that readers submit. The following case is particularly relevant in these days when healthcare reform – and who is going to pay for it – is on everyone's mind.



**Mark Pastin, PhD**

Mark Pastin, PhD, is president and CEO of the Council of Ethical Organizations, Alexandria, VA. The Council, a non-profit, non-partisan organization, is dedicated to promoting ethical and legal conduct in business, government, and the professions.

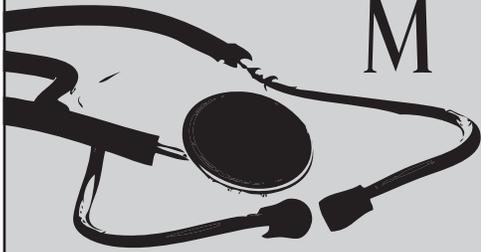
## CASE SIX EASE MY CONSCIENCE

A terminal patient is in great pain but, with the concurrence of the patient's family, refuses, for religious reasons, to allow the plug (on further therapeutic treatment) to be pulled. However, the patient requests that everything be done to reduce the pain to the maximum possible extent. The patient's physicians explain that the pain can be reduced and almost eliminated but at the expense of the patient's consciousness and, imminently, life. The patient and the patient's family find this consequence acceptable. The physicians, however, wonder if they are participating in an assisted suicide. Should the patient's wish be granted?

This is an actual case. Of course, there are any number of complicating circumstances and additional details; but please address the case on the basis of the information provided.

There will be an analysis of this case and a new case in the next issue.

**Your input is requested. Email your responses to: [council@aol.com](mailto:council@aol.com).**



# MEDICAL ETHICS WITHOUT THE RHETORIC

## CASE FIVE ANALYSIS

*In our case from the last issue, a patient is diagnosed with terminal cancer after learning that she is pregnant. The woman and her husband request that her body functions be maintained after she is legally dead until the baby is safely delivered. Her physician advises that this is a reasonable although not certainly successful course of action. The issue? According to the hospital where she is receiving treatment, the cost of maintaining her bodily functions would exceed \$500,000. A dead patient has no health insurance, and the couple does not have the money. Our question: What should be done by the various parties?*

**This case provoked many reader responses. One of the most thoughtful is the following (edited):**

"The patient and her family request that her body be maintained "alive," even after she is brain dead, until the baby can be delivered. This is a perfect case for this day and time. We have the technology to keep the OB patient alive until her baby can be safely delivered, but at what expense. Until now, we as a culture have not given serious thought to what health care is going to cost. Now we are. For decades, health maintenance organizations have sought to reduce health care costs, but it required diminished access to medical procedures, which led to numerous headline legal actions that resulted in patients receiving costly treatments, experimental treatments, and dying in the end.

We now are faced with living with the collective burden of realizing that if we spend the money

to keep her alive, how many others are going to be denied life-saving care? We are faced with deciding the good of the many as opposed to the good of the one. It may be the flip side of the coin where a healthy young adult becomes an organ donor due to a tragic accident and allows several others to have the gift of life through their death. Perhaps, in this case too, it would be the more humane thing to do to allow the demise of herself and her baby, so that others might live. We have lived under a false impression that there are no limits to what we can do and achieve. There have always been those limits. We just chose to ignore them. In the past, the cost of what we were doing ethically and financially was put on the back burner. Now, they are front and center, and we are having to make tough decisions on both fronts."

While I respect the reader's position, I disagree. The first question is how the hospital arrived at this cost estimate. Upon scrutiny, this estimate, like so many of the numbers used by rationing of care advocates, was based on the hospital's billing rates to an uninsured patient – its "rack rate" – a rate seldom if ever paid by anyone. The actual cost was much lower. I cannot find an ethical basis for not allowing this baby to be born. Even \$500,000 is barely the amount of some "patient satisfaction" surveys, which we seem to find socially acceptable. The reader is certainly right on one point – we tend to avoid these issues. In the end, the patient's insurance company agreed to provide a "contribution" in the amount of \$50,000 without admitting that there was coverage. The hospital agreed to sharpen its pencils on the pricing, and several providers contributed their services. So, while no solution was reached, the baby was delivered alive and healthy. In ethics, sometimes "no solution" is a better solution than a precedent-setting decision with unforeseeable consequences.

# Subarachnoid Hemorrhage: State of the Art(ery)

David M. Lemonick, MD, FAAEP, FACEP

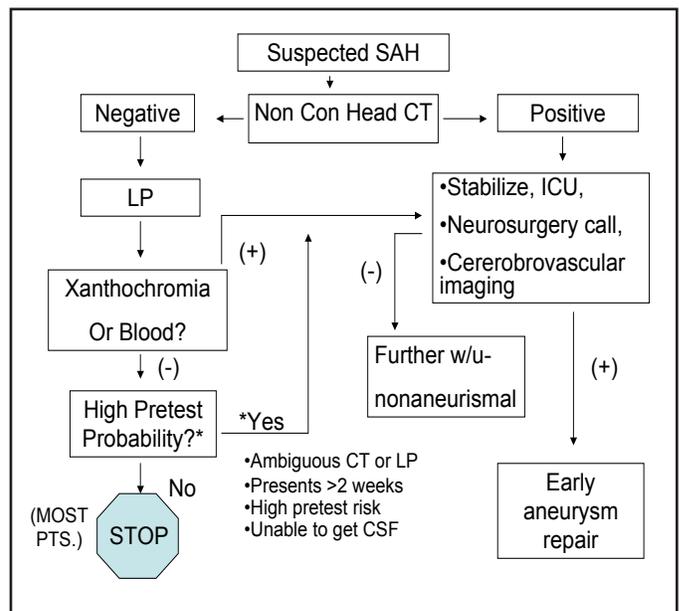
## Abstract

Headache is a common chief complaint in primary and ambulatory care settings. The etiology of headache is usually benign, its workup is often minimal and straightforward, and the clinical approach focuses primarily on symptom control. A small proportion of headaches is caused by subarachnoid hemorrhage (SAH), most of which are due to ruptured cerebral aneurysms. Such hemorrhage carries a high mortality, with significant rates of devastating disability among survivors. Misdiagnosis of SAH is frequent and results in medicolegal risk to the unwary physician. An organized, algorithmic approach emphasizes high risk historical and physical examination features that suggest SAH. Expedient neuroimaging and lumbar puncture are employed to confirm suspected SAH. Diagnosis of SAH requires immediate stabilization, followed by neurosurgical consultation for definitive management. Transfer to high-volume neurosurgical and endovascular centers is associated with improved outcomes for SAH.

## Introduction

Headache is a common chief complaint in the emergency department (ED), constituting approximately 2% of all visits. Of these patients, about 1% will have subarachnoid hemorrhage (SAH).<sup>1,2</sup> In the subset of patients who present with a severe, sudden onset, or “thunderclap,” headache and a normal neurologic examination, 10% to 16% will have SAH.<sup>3-8</sup> Thus, emergency physicians see only one subarachnoid hemorrhage out of every 100 headache patients. Further, it is estimated that 5-15% of these are initially misdiagnosed.<sup>9-13</sup> Delayed diagnosis of SAH confers worse outcomes, highlighting the importance of early recognition and treatment.<sup>14</sup> The mortality of SAH is approximately 40%, with another 30% surviving with significant neurological disability.<sup>15</sup> Misdiagnosis of SAH is an important cause of medico-legal actions against physicians.<sup>5,7,9-13,15-18</sup>

Figure 1: Algorithm for workup of suspected SAH

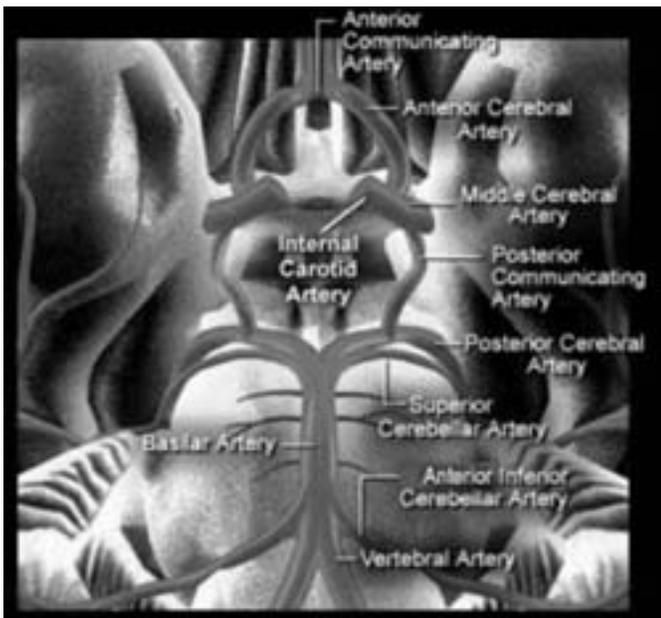


At the same time, an extensive workup of every headache patient in the ED is neither practical nor necessary. Because the diagnosis of SAH will not be made if it is not considered, deciding whom to evaluate for SAH and how to conduct this evaluation can be difficult. CT and LP are the mainstays of the emergency evaluation for SAH. Once the decision has been made to perform these tests, interpreting their results may also be challenging.<sup>18</sup> This article will review the current literature on the diagnosis and management of SAH. Emphasis will be placed on an algorithmic approach that is aimed at a rapid risk assessment utilizing history and physical examination and on the selective use and correct interpretation of CT and LP (Figure 1). Using this approach, patient outcomes may be optimized, while medicolegal risk may be reduced.

## Etiology and Pathophysiology

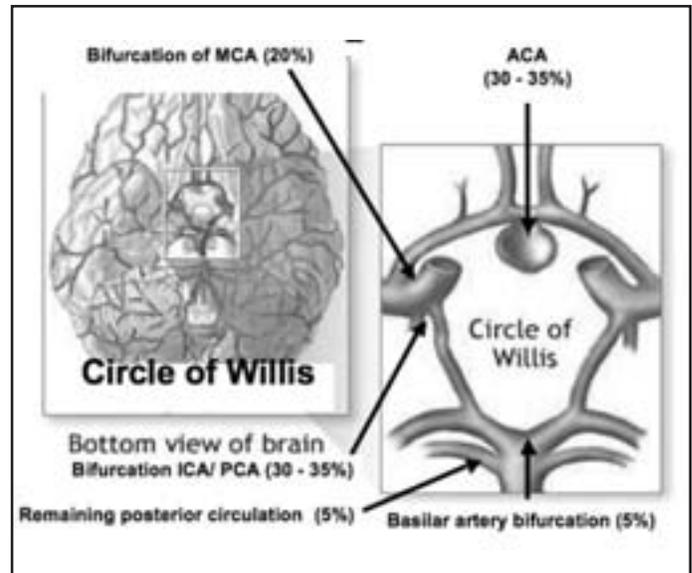
Subarachnoid hemorrhage occurs with an annual incidence of approximately eight people per 100,000 population per year.<sup>19, 20</sup> While most spontaneous cases of SAH are due to ruptured cerebral aneurysms, trauma remains the most common cause of SAH overall.<sup>21,22</sup> Eighty-five percent of spontaneous SAH result from rupture of saccular aneurysms arising in the vessels at the base of the brain. These vessels constitute the “Circle of Willis” (COW) (Figure 2A). Symmetrical to the sagittal plane, the circle of Willis is composed of anterior and posterior portions. The anterior portion is composed of the paired *anterior cerebral arteries*, joined by a single *anterior communicating artery*, and the paired *internal carotid arteries*. Eighty-five percent of ruptured aneurysms arise from the anterior portion of the COW. Fifteen percent of ruptured aneurysms arise from the posterior portion of the COW, which is composed of the paired *posterior communicating arteries* and *paired posterior cerebral arteries* that originate at the bifurcation terminus of the basilar artery (Figure 2B). In contrast to a previous consensus that saccular intracranial aneurysms are always congenital, it is currently believed that aneurysms develop gradually over the lifetime of the patient. Such aneurysms occur in approximately 2% of asymptomatic adults.<sup>22,23</sup>

**Figure 2A:** Anatomy of the Circle of Willis



Several factors have been associated with a higher risk for aneurysmal SAH (Table 1). These risks include a family history of intracranial aneurysms, connective tissue diseases, hypertension, smoking, atherosclerosis, oral contraceptives, older age, African-American and Japanese ethnicity, female sex, polycystic kidney disease, and heavy alcohol drinking.<sup>24,25</sup> There is a three- to seven-fold increased risk of SAH in first-degree relatives of patients with SAH. African-Americans have a risk of 2.1:1, compared to Caucasians, while females are found to have a risk of 1.6:1, compared to men.<sup>13</sup>

**Figure 2B:** Location of aneurysms responsible for SAH



The risk of rupture increases as the size of the aneurysm increases. Aneurysms larger than 10 mm in diameter are five times more likely to rupture than are smaller ones. The annual risk of rupture for aneurysms <10 mm is 0.7%.<sup>26</sup>

Two scales that are widely used to grade the severity of SAH are the Hunt and Hess and the World Federation of Neurological Surgeons scales (Table 2).<sup>27,28</sup> In both scales, there is a useful correlation between higher scores and worse patient outcomes. These scales should be used in conjunction with other clinical information to guide management. This information includes the size and location of the aneurysm and the overall condition of the patient.

**Table 1:** Risk Factors for Aneurysmal Subarachnoid Hemorrhage

- Family or personal history of SAH
- Smoking
- Alcohol abuse
- Cocaine, methamphetamine, MDMA use
- African American or Japanese ethnicity
- Hormone replacement therapy
- Diabetes mellitus
- Polycystic kidney disease
- Heritable connective tissue disorders
  - Ehlers-Danlos syndrome (Type IV)
  - Pseudoxanthoma elasticum
  - Fibromuscular dysplasia
- Sickle cell anemia
- Alpha1-antitrypsin deficiency

**Table 2A:** Hunt and Hess Severity Scale

Grade	Criteria
1	Asymptomatic, mild headache
2	Moderate to severe headache, nuchal rigidity, no focal deficit other than cranial nerve palsy
3	Mild mental status change (drowsy or confused), mild focal neurologic deficit
4	Stupor or moderate to severe hemiparesis
5	Comatose or decerebrate rigidity

## History

Sudden onset and severe, or “thunderclap,” headache has a broad differential diagnosis (Table 3). A rapid and organized clinical approach to these differential diagnoses is based upon a focused history and a directed physical examination. These are supplemented by selective laboratory testing when indicated. While approximately 10 to 15% of thunderclap headaches have been found to be associated with SAH, most SAH patients manifest the symptom of a sudden, severe headache.<sup>1,29,30</sup>

Approximately two-thirds of SAH present with isolated headache in the absence of neurological deficits. Three-quarters of SAH are associated with a “thunderclap” onset.<sup>31</sup> Specific historical features that may be useful in distinguishing SAH from other causes of headache are onset, severity, quality, and

**Table 3:** Differential Diagnosis of Thunderclap Headache

• Subarachnoid hemorrhage
• Sentinel headache
• Cerebral venous thrombosis
• Cervical artery dissection
• Spontaneous intracranial hypotension
• Pituitary apoplexy
• Retroclival hematoma
• Ischemic stroke
• Acute hypertensive crisis
• Reversible cerebral vasoconstriction syndrome
• Colloid cyst of the third ventricle
• Acute complicated sinusitis
• Primary thunderclap headache
• Infections (e.g., acute complicated sinusitis)
• Acute myocardial infarction
• Anaplastic oligodendroglioma
• Aqueductal stenosis
• Pheochromocytoma
• Vogt-Koyanagi-Harada syndrome

**Table 2B:** World Federation of Neurological Surgeons

Grade	Glasgow Coma Scale	Focal neurological deficit
1	15	Absent
2	13-14	Absent
3	13-14	Present
4	7-12	Present or absent
5	3-6	Present or absent

associated symptoms.<sup>32</sup> An abrupt onset of headache and a severity that is described as the “worst-ever headache” both suggest SAH. The headache is lateralized in 30% of patient, often ipsilateral to the side of the aneurysm. In one ED study, 12% of headache patients who responded, “Yes,” to the question, “Is this the worst headache of your life?” or rated their headache pain a 10 on a 1-to-10 scale, had SAH.<sup>6,13</sup>

If a headache presentation is described as unique or different in quality from other headaches, closer attention must also be paid. An adequate history will also determine if there are focal associated symptoms, such as neck stiffness syncope, double vision, or seizure. While seen in 75% of SAH, vomiting also occurs in up to half of benign causes of thunderclap headache. Thus, the presence of vomiting is poorly discriminatory. Seizures accompany SAH in 6% to 9%.<sup>30</sup> Fifty percent of patients presenting with SAH are found to have seizures, transient loss of consciousness, or altered level of consciousness.<sup>30</sup> Photophobia and visual changes may also be reported. Symptoms of meningeal irritation are present in more than three quarters of SAH, but may take several hours to develop. These include neck stiffness, low back pain, and bilateral neck pain. Historical features of SAH are summarized in Table 4.

In one prospective study of 102 emergency department patients with acute severe headaches suggestive of SAH only two characteristics, seizures and diplopia, were present only in patients with SAH.<sup>30</sup> These occurred so infrequently in SAH, however, that they were of little or no use for risk stratification.<sup>30</sup>

## Physical Examination

The physical examination begins with an assessment of the vital signs and the airway, breathing, and circulatory status of the patient. Once stabilized, the patient is assessed by a focused neurological examination. This examination should search for the presence of mental status change, meningismus, cranial nerve palsies, nystagmus, leg weakness, abulia, anisocoria, ataxia, dizziness, hemiparesis, aphasia, neglect, Papilledema, and retinal hemorrhage. The proportion of patients with SAH who present with particular significant physical findings is summarized in Table 5.

Global or focal neurological abnormalities are found in more than 25% of patients, while no localizing signs are present in 40%. Neck stiffness is found in 60% of patients with SAH,

**Table 4:** Historical Features of SAH

Typical presentation
Sudden onset of severe headache, (frequently described as the "worst ever")
Nausea
Vomiting
Neck pain
Photophobia
Loss of consciousness
Atypical Features
Confusional state
Seizure
Associated head trauma

compared with 10% of benign thunderclap headaches in one small study.<sup>31</sup> One-quarter have motor deficits, abnormal speech, or inappropriate responses to commands.<sup>33,34</sup>

Certain physical findings in SAH may suggest the location of the aneurysm. Bilateral leg weakness and abulia are associated with anterior communicating artery lesions. Hemiparesis with aphasia or neglect are seen in middle cerebral artery aneurysms in 15% of cases. Nystagmus, ataxia, dizziness, and third nerve palsy are seen in posterior communicating aneurysms. Isolated third cranial nerve palsy is seen in some aneurysms near the

junction of the internal carotid and posterior communicating arteries and is the most common cranial nerve palsy seen in SAH. Aneurismal neuropathy typically causes the pupil to be dilated because of compression of parasympathetic nerve fibers that run along the outside of the nerve. In contrast, third nerve dysfunction is most often due to infarction of the nerve from diabetes rather than aneurismal compression. In diabetic neuropathy there is flow from collateral vessels that results in sparing of the parasympathetic nerve fibers and, therefore, the pupil is reactive. Abducens nerve palsy and monocular vision loss may also be seen. Of note, the physical examination is entirely normal in many cases of SAH and thus is a nondiscriminatory finding.

### Cerebrovascular Imaging

Noncontrast head CT is the initial laboratory examination to perform in patients with suspected SAH. CT has 90-98% sensitivity if performed within the first 24 hours of the bleed.<sup>18</sup> The sensitivity of CT for detecting SAH decreases with the time from the bleeding due to the reabsorption of blood within, and recirculation of, the CSF. In one review, sensitivity of CT on the day of aneurysm rupture was 92% and progressively decreased to 86%, 76%, and 58% on days 1, 2, and 5 post-rupture.<sup>33-37</sup>

Other limitations to CT include its use in patients with normal neurologic examinations and smaller volumes of hemorrhage who are less likely to have CT abnormalities. Anemia may also reduce the sensitivity of CT, and patients with hematocrit less than 30% may have bleeding that is not detectable because the blood is isodense with brain tissue.<sup>38-43</sup> Technical limitations of the scan, such as motion, bone artifacts, and inexperienced interpretations, may also decrease its sensitivity.

**Table 5:** Physical Examination in SAH

Finding	Likely Location of Aneurysm
Mental status change • Seen in about one-fourth of patients <sup>7,64,67</sup>	Any
Meningismus • Seen in 60% of patients with subarachnoid hemorrhage <sup>7</sup> • Takes 3-12 hours to develop and may not be appreciated in comatose patients <sup>68</sup>	Any
Third nerve palsy <sup>39,69</sup> • 90% of patients with third nerve palsy due to aneurysm (versus other causes of third nerve palsy) have anisocoria >2mm <sup>70</sup>	Posterior communicating artery
Sixth nerve palsy <sup>70,71</sup> • Presents 3-14 days after onset of subarachnoid hemorrhage • Associated with higher clot burden • Gradually resolves	Any (due to increased intracranial pressure)
Bilateral leg weakness, abulia <sup>15,35</sup>	Anterior communicating artery
Nystagmus, ataxia, dizziness <sup>15,35</sup>	Posterior circulation
Hemiparesis with aphasia or neglect <sup>15,35</sup>	Middle cerebral artery
Subhyaloid (retinal) hemorrhage (Terson syndrome) <sup>66</sup> • Seen in about 10% of patients with subarachnoid hemorrhage • Associated with worse clinical grades on presentation and poorer prognosis	Any

The location of blood on a CT may suggest the etiology of the SAH. Blood from a ruptured aneurysm is usually located around the basal cisterns, while the blood is typically higher in the cerebral convexities in traumatic SAH.<sup>32</sup>

There is some debate in the current literature about whether a CT angiogram (CTA) may be used instead of a CT-then-LP workup sequence. This CTA-only approach to sudden severe headache is partly a response to the evidence that fewer than half of these patients, when the initial CT is negative, then undergo LP in actual practice.<sup>40,41,44</sup>

In one well-designed prospective study of CTA in 116 patients, six cases of aneurysms were detected that had not been seen on noncontrast CT.<sup>45</sup> All of these patients had a positive LP result; but there were also three cases of negative CT and negative spinal fluid that had a positive CTA. These cases were thought to represent unruptured aneurysms. Since approximately 2% of the general population harbors intracranial aneurysms, a CTA-first approach might subject headache patients to unnecessary risks of angiography, interventional radiology, and surgery. Some patients have iodine dye allergies or renal insufficiency, further limiting the utility of CTA as a primary diagnostic tool in suspected SAH.<sup>32</sup>

The role of magnetic resonance imaging (MRI) in the evaluation of SAH has also been studied. MRI is superior to CT in detecting subacute and chronic SAH, especially with fluid-attenuated inversion recovery (FLAIR) and T2-weighted imaging.<sup>45-50</sup> FLAIR sequence MRI negates the signal from CSF, permitting the detection of tiny amounts of blood in the subarachnoid space. A small 2004 study identified 12 cases of acute SAH in which CT was negative for SAH and LP was positive for SAH. FLAIR MRI findings were positive in only two of these cases and were false negative in ten.<sup>57</sup> Because of this and because MRI is of limited availability in most EDs, MRI is not currently recommended to replace LP in the evaluation of SAH.<sup>51-58</sup>

Considerable literature exists regarding the optimal imaging modality for the planning of definitive therapy of SAH.<sup>57,58,62-66</sup> The choice of the modality for cerebrovascular imaging to be used to identify the aneurysm will depend in large part upon the preference of the neurosurgical consultant.<sup>55-61,67-69</sup> Cerebral digital subtraction angiography (DSA) has for many years been considered the imaging technique of choice for preoperative imaging of SAH. DSA identifies the bleeding site, size, and location of the aneurysm, and it maps the relevant operative anatomy and elucidates vascular anatomy.<sup>69</sup> Angiography may be negative in 10% to 20% of patients with SAH because of perimesencephalic hemorrhage, vasospasm, thrombosed aneurysm, or other rare causes.<sup>47</sup>

Cerebral computed tomographic angiography (CTA) is rapidly replacing DSA as the primary neurosurgical planning study. Its sensitivity and specificity are comparable to DSA, and it is widely available, fast, and noninvasive.<sup>48</sup>

MRA is another imaging study that, while widely available for the diagnosis and neurosurgical planning for SAH, is less well studied.<sup>47,50,57,59,70</sup> The choice of imaging modality for neurosurgical planning is best made in consultation with the neurosurgeon or neuroradiological interventionalist. In many cases definitive treatment of SAH is now being accomplished by endovascular techniques at the same time as the initial diagnostic angiogram. Because of the sequential dye loads entailed during these two phases of diagnosis and treatment, thoughtful coordination of these steps by the neurosurgeon and neuroradiologist are especially important.

## Role of Lumbar Puncture

Because of the limitations of CT as outlined above, all patients with suspected SAH who have a negative CT should undergo lumbar puncture.<sup>39</sup> This is especially important in awake, neurologically intact patients who present with sudden, severe headaches. Such patients are particularly likely to have a negative CT despite the presence of SAH. In one study, only 25-50% of such patients underwent LP after a normal CT.<sup>40</sup> In another study, 10% of neurologically normal ED patients with acute headache were diagnosed on the basis of positive CSF results after a normal cranial CT.<sup>41</sup> Other studies have determined that 2-7% of CT negative SAH patients were ultimately picked up by positive LP results.<sup>13,36,42,43</sup>

The cerebrospinal fluid (CSF) obtained by LP should be assessed for the presence of red blood cells (RBCs) and for xanthochromia. An absolute number of RBCs that establishes the diagnosis of SAH has never been established.<sup>32</sup> A traumatic tap is estimated to occur in 10-15% of LPs.<sup>44</sup> Typically, progressive clearing of blood in each succeeding tube of CSF of the four collected represents evidence that the tap was traumatic. Ideally, if the final tube has zero RBCs, it may be concluded with confidence that the tap was traumatic. When a less than complete clearing of RBCs is demonstrated, the higher the RBC count in the final tube, the more likely it is that a true positive (i.e., SAH) is present. In this situation, additional diagnostic testing may be indicated and includes computed tomographic angiography (CTA), magnetic resonance angiography (MRA), or conventional cerebral angiography.<sup>45-50</sup>

Xanthochromia refers to a yellow discoloration that may be detected in CSF that suggests the presence of SAH. This discoloration results from the enzymatic breakdown of red blood cells in the CSF, and it is rarely seen within the first four hours of SAH. Xanthochromia usually requires at least six hours, and sometimes up to 12 hours, to develop after SAH. Xanthochromia is detected either by spectrophotometry or by simple visual comparison of the CSF sample to an equal volume of water against a white background. Although spectrophotometry, more commonly employed in Europe, is the most sensitive method of detection of xanthochromia, many hospital laboratories in the United States rely upon simple visual inspection of the CSF sample. The absence of xanthochromia by either visual or spectrophotometric analysis has a high negative predictive value for SAH, with one estimate of 99%.<sup>51</sup> Several

conditions may create false positive xanthochromia, including jaundice (total bilirubin > 0-15 mg/dL), increased cerebrospinal fluid protein (>150 mg/dL), rifampin use, and excess dietary carotenoids.<sup>32</sup> Xanthochromia may persist for up to two weeks following SAH.<sup>52</sup>

Measurement of the opening pressure (OP) is also useful in the evaluation for SAH. OP is elevated to >20 cm H<sub>2</sub>O in 60% of patients with SAH.<sup>52</sup> CSF pressures may also be elevated in cerebral venous thrombosis and in idiopathic intracranial hypertension, and low pressures may be seen in spontaneous intracranial hypotension.<sup>53,54</sup>

## Initial Management

Once the diagnosis of SAH is confirmed, the priorities are: 1) airway management, 2) analgesics, 3) arrhythmia monitoring, 4) blood pressure control, 5) seizure prophylaxis, 6) vasospasm prophylaxis, 7) hydration, and 8) hydrocephalus management.

Simultaneously, an immediate call should be made to a neurosurgeon. In this way, further imaging and management of the patient may be coordinated while definitive care is arranged. Next, some form of cerebrovascular imaging is obtained in consultation with neurosurgery (e.g., magnetic resonance angiography [MRA], CT angiogram [CTA], or traditional cerebral angiography- see "Cerebrovascular Imaging," above).

## Airway Management

Airway management is chosen based upon usual clinical criteria. Endotracheal intubation is almost never needed in patients with mild SAH (grades 1-3), while for more severely affected patients (grades 4 and 5 or Glasgow Coma Scale of ≤8), most will require intubation for airway protection, oxygenation and/or ventilation. Rapid sequence intubation should be used. While the efficacy of lidocaine and/or fentanyl to blunt the rise in intracranial pressure in this setting is unclear, it is the author's practice to use them. Short-acting agents with attention to adequate sedation should be used for rapid-sequence protocols, so that the neurological examination can be followed by the consulting neurosurgeon. In most circumstances, a quick baseline neurological examination prior to drug administration should be performed and documented. Thiopental and etomidate are optimal induction agents in SAH; the former has a cytoprotective effect, but its use is limited to hypertensive patients because of its tendency to drop systolic BP. Hyper- and hypoventilation are to be avoided, with a target pCO<sub>2</sub> of 30-35 mmHg.

A rapid baseline neurological examination should be performed and documented prior to drug administration, and subsequent excessive sedation is minimized so as to allow serial neurological examinations. Excessive sedation has also been reported to increase ICP directly.<sup>55</sup>

The patient is placed at bed rest with the head of the bed elevated 30° and is given nothing by mouth except for medications.

## Analgesia

Analgesia is provided for severe headache and for painful procedures, using small doses of a short-acting, reversible agent. Sedation is provided to intubated patients, preferably with short-acting, titratable or reversible medications that allow repeat neurological examinations.

## Arrhythmia Monitoring

Subarachnoid hemorrhage is associated with symptomatic bradycardia, ventricular tachycardia, and ventricular fibrillation. A variety of twelve-lead electrocardiogram changes have also been described, including ST-segment elevation or depression, QT interval prolongation, and T-wave abnormalities, often mimicking myocardial ischemia or infarction.<sup>55, 58</sup>

In their 2009 guidelines on management of SAH, the American Heart Association has recommended that all patients with SAH be treated in an intensive care unit setting with cardiac and blood pressure monitoring.<sup>57</sup>

Blood pressure is managed, aiming at a target blood pressure goal chosen in consultation with the neurosurgical critical care team. The available research on the control of blood pressure in SAH is inconclusive and contradictory, and individual practices vary.<sup>21,34,39,55, 62,63,64</sup> Control of blood pressure reduces the rate of rebleeding, but this reduction is achieved at the expense of increasing the rate of cerebral infarction; the rate of rebleeding was 15% in patients treated with antihypertensives, compared with 33% in the nontreated group. But the rate of cerebral infarction doubled in the treatment group compared with the nontreatment group (40% vs. 22%).<sup>60,61</sup> Most centers begin to address blood pressure by treating pain, anxiety, and nausea. Stool softeners are also commonly administered.<sup>58</sup>

There is no consensus on the degree to which blood pressure should be reduced. Suarez et al. have recommended that systolic blood pressure (SBP) be maintained at 90-140 mm Hg before aneurysm treatment, after which SBP of ≤ 200 mm Hg is permitted.<sup>21</sup> If blood pressure reduction is chosen, this is often accomplished with short-acting intravenous agents, such as labetalol, nicardipine, and esmolol.<sup>32,39,62-64</sup> Nitroprusside and nitroglycerine are generally avoided because of their potential to increase intracranial pressure (ICP).<sup>58</sup> Vasopressor agents and optimal preload; i.e., central venous pressure (CVP) 5-8 mm Hg, may be required to maintain SBP over 120 mm Hg in order to avoid further CNS damage in the ischemic penumbra due to vasospasm that is seen in SAH (see section below on vasospasm management). In the event of vasospasm, a higher CVP of 8-12 mm Hg, or a pulmonary capillary wedge pressure (PCWP) of 12-12 mm Hg, is recommended.<sup>21</sup>

## Seizure Prophylaxis

Antiepileptic drugs (AEDs) are widely administered to prevent seizures in patients with SAH, but their use continues to be a subject of debate.<sup>58</sup> A loading dose of phenytoin, or its equivalent, is often given early in the course of ICU stabilization.

Barbiturates or benzodiazepines are avoided as first-line AEDs, because they tend to over-sedate the patient, masking potentially neurologic examination findings.<sup>55</sup> While the prevention of seizure activity and resulting secondary brain injury is reasonable, one large series has found that AED exposure may be correlated with worse cognitive and neurologic outcome.<sup>62</sup> In the setting of perimesencephalic blood without cortical layering; i.e., blood only at the base of the brain, conferring a good prognosis, withholding of AEDs seems to be reasonable. After definitive treatment of the aneurysm in patients without acute seizures and with a lower grade SAH, discontinuation of AED is often recommended.<sup>58, 59</sup>

## Vasospasm Prophylaxis

Vasospasm occurs in up to 30% of patients with SAH, and it is the leading cause of death and disability after aneurysm rupture.<sup>33, 59</sup> It is manifested by a decline in neurological status, usually occurring after 72 hours from the ictus, and peaking at the eighth day. Vasospasm may develop up to two weeks after SAH, and it carries significant risk of morbidity from cerebral infarction.<sup>21, 32, 34, 39, 43, 62-67</sup> Some authors have recommended that transcranial Doppler (TCD) ultrasonography measurement be taken at baseline and during treatment to monitor for vasospasm.<sup>58</sup>

Nimodipine is a calcium channel blocker that is given orally or by nasogastric route, at a dose of 60 mg every four hours. Nimodipine treatment reduces poor outcomes, though the mechanism remains unclear.<sup>61, 65</sup> It has been noted that, contrary to popular belief, nimodipine does not prevent or treat the vasospasm itself. While nimodipine is initiated as soon as possible after SAH, aggressive therapy for vasospasm itself is only appropriate after occlusive therapy of the aneurysm.<sup>58</sup> Intraluminal papavarine and angioplasty have both been employed to treat vasospasm. Other means of treating vasospasm include hemodilution, induced hypertension, and hypervolemia (also known as “triple-H therapy”).<sup>58</sup>

## Hydration

The hydration status of the patient has been referred to above in the section on Blood Pressure Management. Treatment of hypotension with a goal of optimizing cerebral perfusion pressure requires appropriate invasive monitoring; e.g., swan-ganz, ventriculostomy, central venous pressure monitor, arterial pressure monitor. A CVP of 5-8 mm Hg may be required to maintain SBP over 120 mm Hg. In order to avoid further CNS damage in SAH. In the event of vasospasm, a higher CVP of 8-12 mm Hg or a pulmonary capillary wedge pressure (PCWP) of 12-12 mm Hg is recommended.<sup>21</sup>

**Table 6:** SAH Practice Guidelines

<p><b>A. 2008 American College Of Emergency Physicians Clinical Policy On Acute Headache (Evidence-Based Recommendations)<sup>19</sup></b></p> <ol style="list-style-type: none"> <li>1. Emergent head computed tomography <b>IS</b> the initial diagnostic test recommended in the diagnosis of any new, sudden-onset, severe headache or suspected case of subarachnoid hemorrhage* (Class I, Level B recommendation).<sup>20-26</sup></li> <li>2. Lumbar puncture <b>IS</b> recommended for patients with suspected subarachnoid hemorrhage after negative noncontrast head computed tomography* (Class I, Level B recommendation).<sup>20, 22</sup></li> <li>3. Angiography is <b>NOT</b> recommended in patients with sudden-onset, severe headache who have negative findings on head computed tomography, normal opening pressure, and negative cerebrospinal fluid findings (Class II, Level B recommendation).</li> <li>4. Patients with a negative workup including negative computed tomography and lumbar puncture <b>CAN</b> be safely discharged from the emergency department, with outpatient follow-up recommended (Class II, Level B recommendation).</li> <li>5. Response to analgesia should <b>NOT</b> be used as the sole indicator to the etiology of an acute headache (Class III, Level C recommendation).</li> </ol> <p style="text-align: right;"><i>*Also supported by 2009 American Heart Association guidelines.</i></p> <p><b>B. 2009 American Heart Association Guidelines For Management Of Subarachnoid Hemorrhage (Evidence-Based Recommendations)<sup>20</sup></b></p> <p>Recommendations for diagnostic studies</p> <ol style="list-style-type: none"> <li>1. Once subarachnoid hemorrhage is diagnosed, urgent cerebral angiography <b>IS</b> needed to detect the underlying cerebral aneurysm (Class I, Level B recommendation).<sup>20, 21</sup></li> <li>2. When conventional angiography cannot be performed in a timely fashion, magnetic resonance angiography and computed tomographic angiography <b>MAY</b> be considered (Class III, Level B recommendation).<sup>20</sup></li> </ol> <p>Recommendations for management</p> <ol style="list-style-type: none"> <li>1. Patients with subarachnoid hemorrhage should be treated in an intensive care unit setting with cardiac and blood pressure monitoring (Class I, Level B recommendation).<sup>20, 21, 25</sup></li> <li>2. Control of elevated blood pressure <b>IS</b> recommended to balance the risk of stroke, rebleeding, and maintenance of cerebral perfusion pressure (Class II, Level B recommendation).<sup>20, 25</sup></li> <li>3. Oral nimodipine <b>IS</b> strongly recommended to reduce poor outcome from vasospasm (Class I, Level A recommendation).<sup>20, 21</sup></li> <li>4. Prophylactic anticonvulsant therapy <b>MAY</b> be considered in the immediate posthemorrhage period (Class III, Level B recommendation).<sup>20, 21, 25</sup></li> <li>5. Early surgery <b>IS</b> recommended for most patients (Class II, Level B recommendation).<sup>20, 21</sup></li> </ol> <p>Recommendations for transfer</p> <ol style="list-style-type: none"> <li>1. Early referral to high-volume centers with cerebrovascular surgeons and endovascular services <b>IS</b> recommended (Class II, Level B recommendation).<sup>20, 21, 27</sup></li> </ol>
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In contrast, overhydration of the patient is avoided as this increases the risk of hydrocephalus, which is discussed below.

## Hydrocephalus

Hydrocephalus may develop within the first 24 hours of the SAH, either as a result of clotted blood products obstructing the flow of CSF or by decreased absorption of CSF at the arachnoid granulations.<sup>64</sup> In one study, hydrocephalus was documented in 15% of patients by CT scan, of whom almost one-half were asymptomatic.<sup>64</sup> Factors correlated with the development of hydrocephalus include intraventricular hemorrhage, treatment with antifibrinolytic agents, posterior circulation SAH, low GCS on presentation, history of hypertension in patients with hyponatremia. One-half of patients with acute hydrocephalus and decreased level of consciousness will spontaneously improve, while the other half experience rebleeding, infarction, and increased morbidity and mortality.

Ventricular drain placement is considered for patients with hydrocephalus and decreasing level of consciousness and for those whose hydrocephalus does not improve within 24 hours.<sup>65</sup>

## Reversal of Anticoagulation

The American Heart Association/American Stroke Association guidelines recommend discontinuation of all antiplatelet and anticoagulant medications after SAH. In addition, immediate reversal of any anticoagulant effect is advised for SAH until definitive surgery or coiling of the aneurysm.<sup>32,66,67</sup> Intravenous vitamin K and fresh frozen plasma (FFP) or prothrombin complex concentrates (PCC) may be required. PCC has been associated with a faster reversal in international normalized ratio (INR) when compared to FFP.<sup>32,34,68</sup> The use of recombinant human factor VIIa has also been described.<sup>57,69</sup> Management of a supratherapeutic International Normalized Ratio (INR) has been published.<sup>57,69</sup> Specifically, warfarin is withheld and 10 mg vitamin K1 is administered by slow IV infusion. Anaphylactoid reactions to intravenous vitamin K have been documented in a small number of cases despite the widespread use of this drug. This suggests that the reaction is rare. Additionally, anaphylactic reactions and case fatality reports may occur even when intravenous vitamin K is given at low doses by slow dilute infusion.<sup>69-73</sup>

Intravenous vitamin K<sub>1</sub> should be diluted and given at a slow rate (less than 1 mg/min) because of the danger of systemic reactions, including hypotension.<sup>71</sup> Avoidance of hypotension is of paramount importance in SAH in order to maintain CPP.

## Definitive Management

Definitive management of SAH from aneurysm rupture involves either surgical clipping or endovascular ablation. In contrast to earlier protocols, definitive repair now usually takes place within 72 hours of the bleed.<sup>13,27,32,34,38,39,43,55,58,60,61,63,72,73,83</sup>

Improved outcomes have been achieved for eligible patients who underwent endovascular coiling, in comparison to surgical

clipping.<sup>74</sup> Each patient is best evaluated individually for clipping versus coiling, based on anatomical characteristics of the aneurysm, the experience of the treating physicians as well as on the patient's clinical status.<sup>74,75</sup>

Whether they are treated by surgical clipping or by endovascular coiling, patients with SAH have better outcomes when treated in high-volume centers.<sup>32,76-80</sup>

Whenever feasible, SAH patients should be transferred to such specialized neuroscience centers, and such transfer has been found in one prospective study to be both safe and expeditious. In that study, the average time to effect transfer (measured from time of SAH diagnosis to arrival at the receiving facility) was five hours. Only 10% of these patients had a significant decrease in Glasgow coma scale (GCS). Prearranged transfer agreements between community hospitals and referral centers are likely to facilitate and expedite such transfers.<sup>77</sup>

## Complications

Complications occurring after SAH include rebleeding, vasospasm, hydrocephalus, hypothalamic dysfunction, hyponatremia, aspiration pneumonia, and left ventricular (LV) systolic dysfunction. The subjects of vasospasm and hydrocephalus and their treatment have been addressed previously (see Initial Stabilization and Management).

Rebleeding occurs in approximately 15%-20% of patients within the first few hours of SAH.<sup>32,55,58,61,68-82</sup> Rebleeding carries a mortality risk of 40% without intervention.<sup>21</sup>

If the diagnosis of SAH is missed initially, those patients who present in good clinical condition are five times more likely to rebleed and nine times more likely to have poor outcomes than those who are diagnosed without delay.<sup>15,18,55,58</sup> Rebleeding appears to occur with greater frequency in patients with higher Hunt and Hess scores. Control of blood pressure and antifibrinolytic therapies were the principle means of minimizing rebleeding rates in an earlier era in which definitive repair was delayed for weeks after the SAH. Current concepts empha-

**Table 7:** Complications of SAH

Complications
Hydrocephalus
Rebleeding of subarachnoid hemorrhage
Vasospasm
Neurologic deficits
Hypothalamic dysfunction which may lead to <ul style="list-style-type: none"> <li>- Myocardial ischemia or</li> <li>- Labile detrimental BP.</li> </ul>
Hyponatremia
Aspiration pneumonia and other complications of critical care.
Left ventricular systolic dysfunction

size early definitive care as the ideal way to prevent rebleeding, which increases when definitive aneurysm repair is delayed.<sup>57,58</sup>

Hypothalamic dysfunction after SAH may result in excessive sympathetic stimulation, which in turn may cause labile and detrimental blood pressure fluxes and myocardial ischemia.

Aspiration pneumonia, deep venous thrombosis (DVT), and the full range of other typical ICU complications are also seen after SAH. Airway protection is achieved by ETT or by keeping the patient NPO until a formal swallowing evaluation is performed. Nasogastric intubation or percutaneous gastrostomy tubes may be required for enteral nutrition of the SAH patient who fails a swallowing study.

DVT prophylaxis with subcutaneous heparin and serial compression devices are considered or given at the time of SAH. One author has recommended that pneumatic compression stockings be applied prior to aneurysm treatment and that subcutaneous unfractionated heparin 5,000 units three times a day be added once the aneurysm is treated.<sup>65</sup> A Joint Commission performance measure is that DVT prophylaxis be initiated by 48 hours after SAH.

Hyponatremia in SAH results either from inappropriate secretion of antidiuretic hormone (SIADH) characterized by normal or increased intravascular volume or, less commonly, from cerebral salt wasting, with low intravascular volume.<sup>83,84</sup> Treatment of hyponatremia due to SIADH typically consists of fluid restriction, but this may not be appropriate for patients with SAH because it may promote cerebral vasospasm. Instead, hyponatremic patients with SAH should be treated with hypertonic (3%) saline in order to support cerebral perfusion and to prevent hyponatremia-induced brain swelling. Fluid administration is indicated for cerebral salt wasting. Patients with SAH should be kept euvolemic, since hypovolemia is associated with cerebral ischemia and a worse outcome.<sup>85-88</sup>

Cardiac abnormalities associated with SAH include cardiac biomarkers elevation, electrocardiographic changes, and left ventricular (LV) systolic dysfunction. The mechanisms of cardiac dysfunction after SAH remain controversial. In one study regional wall motion abnormalities were present in 35% of patients with Hunt & Hess grades 3 – 5.<sup>88</sup> Up to 20% of patients with SAH demonstrate elevated troponin I levels, and these patients are more likely to have EKG abnormalities and clinical evidence of left ventricular dysfunction. EKG abnormalities include ST segment depression, QT interval prolongation, deep T wave inversions, and U waves. Life-threatening dysrhythmias, including torsades de pointes, have been reported as well as atrial fibrillation and flutter.<sup>58</sup>

## Discussion

### Role of the “Warning” Headache

A “sentinel” or “warning” headache is a sudden, severe headache that resolves and that is retrospectively identified to have occurred days to weeks before the SAH. Sentinel bleeds, only

diagnosed in retrospect, are believed to occur in 10% to 40% of SAH patients.<sup>1-3,9-13,15,23, 32,34,60,61,81,82</sup> The reasons that have been suggested to explain the phenomenon of the sentinel headache include recall bias, initial misdiagnosis of SAH, and minor initial leak of an aneurysm. It has been noted that patients with a sentinel bleed have higher rates of rebleeding and a higher mortality than those without one.<sup>61</sup>

### LP-First Strategy

Although the general recommendation is that CT, followed by LP, is the appropriate diagnostic sequence in the majority of patients with suspected SAH, there are proponents of performing the LP first in selected patients.<sup>6-16,29,32,35,41,46,82,83</sup> Advocates of this strategy reason that certain patients who are neurologically normal and without evidence of increased ICP could undergo an LP alone, which, if normal, would allow for safe discharge from the ED. In this way some patients could be spared the time, expense, and radiation exposure of CT. Critics of this approach argue that the removal of CSF from patients with unrecognized intracranial hematomas may precipitate herniation and rebleeding, each of which carries a significant morbidity.<sup>55,56</sup> These critics also cite the fact that CT may identify other causes of sudden, severe headache, such as paranasal sinusitis and venous sinus thrombosis. No prospective studies are yet available on the effectiveness of the LP-first approach. In one recent retrospective study, 5% of patients with spontaneous SAH and normal neurological exams had computed tomographic findings that contraindicated lumbar puncture.<sup>89</sup>

For these reasons CT followed by LP remains the standard current recommendation for the diagnostic testing in suspected SAH.<sup>43,51</sup>

### CT Scanning Alone (Without LP)

CT imaging could be used as a definitive diagnostic study in SAH patients if its diagnostic sensitivity was sufficiently high. Some authors have suggested that fifth generation CT scanners possess this sensitivity. In one recent study from Denmark, 296 patients were found to have a SAH.<sup>90</sup> In 295 of these patients the diagnosis was based on a positive CT scan. In a single patient, on day six after SAH, the diagnosis was based on a positive lumbar puncture. From day one to day five in this study, CT scanning was found to have a sensitivity of 100%. Overall, CT scanning had a sensitivity of 99.7%. CT scanning was demonstrated to be excellent as a lone diagnostic test in the first days after ictus and a negative CT scan was sufficient to exclude SAH. These authors suggested omitting LP in the first three days after ictus, if the results of the CT scan are negative. Similarly, in a paper by Boesinger et al. patients presented with headache and had a CT scan of the head with a fifth generation multi-detector CT scanner followed by a LP to rule out SAH. There were 177 patients who presented to the ED with headache and who went on to have a CT scan and an LP to rule out SAH. No patients who had a negative CT were found to have a SAH. It was concluded that fifth generation CT scanners are probably more sensitive than earlier scanners at detecting SAH.<sup>91</sup>

In contrast, Byyny et al. evaluated the sensitivity of noncontrast cranial CT in detecting all spontaneous subarachnoid hemorrhages and found an insufficient sensitivity as a stand-alone test.<sup>42</sup> This was a retrospective review that identified 149 patients with SAH in whom noncontrast cranial CT scan diagnosed SAH in 139 patients. The remaining ten patients had SAH diagnosed by LP. Thus, CT scan sensitivity was only 93%. For the 67 patients presenting with headache and normal mental status that had a SAH and vascular lesions (either aneurysm or arteriovenous malformation), the sensitivity of cranial CT scan was 91%. The authors concluded that noncontrast CT imaging exhibited inadequate sensitivity to serve as a sole diagnostic modality in detecting SAH. The ACEP clinical policy on the management of patients presenting to the emergency department with headache states that, "In patients presenting to the ED with sudden-onset, severe headache and a negative noncontrast head CT scan result, lumbar puncture should be performed to rule out subarachnoid hemorrhage."<sup>42,43</sup>

Early deterioration in the SAH patient's neurological status may occur due to cerebral infarction, rebleeding, hydrocephalus, or subdural extension. Repeating the CT scan in cases of acute decline in neurological status is recommended because some of these etiologies are potentially reversible.<sup>32, 43, 62, 64, 85</sup>

## Prognosis

Ten to 15% of patients with SAH die before reaching medical care. In-hospital mortality for SAH is approximately 30%. Thirty-day mortality of SAH approaches 50%.<sup>15,18,21,23,32,34,43,62,78</sup>

The occurrence of rebleeding after SAH is the single most prognostic factor, conferring an 80% rate of mortality or serious disability. Other significant prognostic factors are age, comorbidity, perioperative complications, vasospasm, level of consciousness and clinical grade of bleeding at time of presentation, and amount of blood on initial head CT. Other conditions associated with poor prognosis are hyperglycemia, fever (both infectious and non-infectious), cerebral infarction and symptomatic vasospasm, and renal insufficiency.<sup>58</sup> Long-term disability occurs in up to half of all SAH patients and includes cognitive and other neurological deficits and epilepsy.<sup>32</sup>

## Summary

Of the approximately 120 million patients who present to America's emergency departments annually, over two million will have a chief complaint of headache. Approximately 2% of these patients, or 27,000, have SAH. Of these, almost one-half, or 14,000, will die within the first month after hemorrhage. Of the 14,000 patients who survive more than a month, over half will have a serious and permanent neurological disability, especially when the diagnosis is delayed.

When faced with a patient with a sudden-onset, severe headache, the physician must do the following rapidly: 1) conduct an expeditious history and physical examination, 2) perform an emergent non-contrast CT scan, and 3) if it is negative, perform

an LP. At the same time, the clinician must be aware of the limitations of these studies, and must be expert in their interpretation. Because of the wide spectrum of complaints in, and the atypical presentations of, patients with SAH, missed diagnoses occur with alarming frequency.<sup>5,7,9-13,15,16,18,21,23,29-32,34,37,43,55-57,8</sup>

Emergency physicians must stabilize the SAH patient, beginning with basic cardiorespiratory monitoring and resuscitation, and then by reversal of anticoagulation and by neurologic management, including nimodipine, to improve outcomes related to vasospasm, and optimization of cerebral perfusion. If SAH is diagnosed, prompt and appropriate cerebrovascular imaging will be needed. It should be ordered in consultation with the receiving neurosurgeon or interventional neuroradiologist.<sup>91</sup>

Finally, because patients with SAH fare better when they are transferred to high volume neurocritical care centers, such transfer should be arranged if possible. Once there, neurosurgical clipping or endovascular coiling of the aneurysms may be accomplished.

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# A Case of Surfers' Myelopathy

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## Introduction

Surfer's Myelopathy is a rare, non-traumatic spinal cord injury that occurs with first-time surfers. This syndrome has been described in the emergency medicine literature only twice; first by Thompson et al. in 2004. This is a case of Surfer's Myelopathy with a similar presentation as first reported by Thompson et al.

## Case Report

A 29-year-old man was brought to the emergency department via EMS because of lower extremity weakness. He was surfing for the first time, with an instructor, when he felt a "spasm" in his back. He continued to surf, and, shortly after he finished, he noticed weakening of his legs. By the time he reached the ED by ambulance, he had total motor deficit (0/5) of the lower extremities. There was no alteration of consciousness. The patient developed tactile hyperesthesia to the level of the waist, which he described as "like frostbite."

The patient has a past medical history of bilateral thrombectomies for lower extremity DVTs in the distant past. He was taking aspirin at home daily and has a vague history of an allergic reaction to penicillin. The patient was a fitness trainer from Canada, visiting Hawaii. He denies tobacco, alcohol, or drug use, and he was not currently on any medications.

On physical examination in the ED the patient was alert and oriented with mild pain in his extremities. His blood pressure was 127/79 mmHg, pulse 97 beats/minute, respiratory rate 16 breaths/minute, and temperature 98.7°F. His ears, nose, throat, neck, cardiovascular, respiratory, rectal, and skin examinations were all normal. He had urinary retention. His back was grossly normal with decreased range of motion and pain with range of motion. His neurologic examination showed a motor deficit with decreased strength bilaterally. The patient had 0/5 strength in his quadriceps and hamstrings bilaterally, 0/5 strength in his

foot flexor and extensor, and slight movement of his right hip. Sensation was intact but decreased to pinprick on his feet. He also had hyperesthesia to touch. His reflexes were 1+ bilaterally, but deep tendon reflexes were absent at the patellar and Achilles' tendon. His mood and affect were normal. His extremities were without clubbing, cyanosis, or edema. Pulses and capillary refill were full throughout.

In the ED the patient was given Solu-Medrol empirically per spinal cord injury protocol. He was also given Dilaudid with resolution of his back pain. A Foley catheter was inserted to relieve urinary retention.

The patient's laboratory values were within normal range. The patient had an MRI done of his lumbar spine. There was a 1.3 cm high signal posterior to the L3 vertebral body, which was a benign hemangioma. There was also a minor disc bulge at L4-5 with mild spinal stenosis and minor disc bulge at L3-4 with no stenosis. There was no enhancement of the conus and no enhancing masses.

The clinical impression at this time was spinal cord insult without radiologic evidence of injury, consistent with Surfer's Myelopathy. The patient was admitted to the intensive care unit (ICU).

While in the ICU the patient had a neurologic consult where he revealed a past history of blurring vision and ptosis of the eyes. A brain MRI was done, which was normal. An MRI of the cervical spine was unremarkable. A follow-up MRI of the spine showed increased linear signal in the thoracic cord from T9 through the conus. There was no significant central stenosis, cord enlargement, disc protrusion, or abnormal marrow signals.

The patient was kept on Solu-Medrol for the next 23 hours and had motor strength checks every two hours. After 12 hours in the ICU the patient had plantar and dorsiflexion capabilities in both his feet. After 24 hours the patient was able to flex

and extend his knees and eventually progressed to abduction and adduction at the hip. By the time of discharge, the patient was able to plant his feet on the ground, though his legs were unable to bear weight. His strength had progressed to 4/5 bilaterally in all muscle groups. The patient had some lingering urinary retention, but he was taken off the Foley and put on a straight catheter. He also had back muscle spasms, which were treated with baclofen and Tylenol. The patient was discharged to a physical therapy and rehabilitation clinic at his home. His prognosis for full recovery was felt to be good, given his health, age, and progress.

## Discussion

The patient was diagnosed with Surfer's Myelopathy, a relatively new condition first reported by Thompson et al. in 2004.<sup>1</sup> Thompson's series of cases were collected by retrospective review of hospital records between 1998 and 2003. All of the cases were first-time surfers who experienced back pain and then progressive paralysis. Various diagnostic tests were done on the patients, though no tests were definitive in confirming the diagnosis or etiology of the condition.

Another case of Surfer's Myelopathy was reported by Aviles-Hernandez et al. in 2006.<sup>2</sup> This case is thought to be Surfer's Myelopathy because of the progression of paralysis, lack of diagnostic imaging abnormalities, and, most importantly, the patient was injured while learning to surf. Interestingly, the patient had experience with another water sport, body surfing, and also was physically fit. Although he did have experience body surfing, bodysurfers will often wear fins and kick their legs to catch the wave without the use of a surf board. When surfing with a board the arms are the main means of locomotion, while lying prone on the surf board, and Thompson et al. postulated that this position and associated hyperextension was most likely the cause of Surfer's Myelopathy.

Surfer's Myelopathy is thought to be a non-traumatic ischemic event to the spinal cord caused by the hyperextended prone position required to paddle on a surfboard. The area of ischemia would most likely occur in the watershed region of the lower thoracic spine, before the largest of the radicular arteries, the artery of Adamkiewicz, joins the anterior spinal artery.<sup>6</sup> This type of ischemic injury is mostly seen with disease and subsequent repair of the aorta, which compromises the blood flow to the radicular arteries.<sup>5</sup> Signal changes in this area are reported in the cases presented from Thompson et al. and Aviles-Hernandez et al., increasing the probability of ischemic injury in this area.<sup>1,2</sup>

As to why only certain first-time surfers are afflicted with Surfer's Myelopathy, it would require more research into the body mechanics of first-time surfers.

There is still relatively little known about Surfer's Myelopathy due to lack of recognition and study into the condition. Since it only affects inexperienced surfers, the incidence is small compared with more traumatic injuries associated with surfing. To

date, there have been only ten cases reported in the literature, and all cases occurring in Hawaii. Since Hawaii is not the only area that is popular for surfing, it is important that physicians and surfers in other popular surfing destinations have an awareness of this condition. It is also important to increase awareness in surfing instructors who may be able to stop additional injury in those first-time surfers who begin to experience back pain.

The Surfer's Myelopathy Foundation works to increase awareness among surfers and to support the victims of Surfer's Myelopathy. They are currently working on a "Surf Safe" program that is a training program for surfing instructors to improve awareness of this condition. They are also developing a database for cases of Surfer's Myelopathy, which will help in tracking the incidence and progression of the condition and may support further research efforts in prevention.

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# A Family Physician's Role in the Prevention, Diagnosis, and Management of Breast Cancer

Daniel M. Avery, Jr., MD

## Abstract

Family physicians have an integral role in the prevention, diagnosis, and management of breast cancer. Breast cancer is the greatest medical threat to a woman's health. The risk of breast cancer in a woman's lifetime is 1 in 8. Physicians must be familiar with risk factors for breast cancer, be confident in examining the breasts, evaluating breast masses, and referring for treatment. This paper summarizes a family physician's role in patients with breast cancer.

## Introduction

Breast cancer continues to be the greatest threat to a woman's health besides trauma. Breast cancer is the most common cancer in women. The risk of a woman developing breast cancer in her lifetime is 1 in 8, and it appears to be increasing. There are about 217,000 new cases of breast cancer diagnosed in the United States yearly.<sup>1</sup> About 40,000 women die from breast cancer yearly in this country.<sup>1</sup> Increasing age increases the risk of breast cancer. About 85% of women with breast cancer have a negative family history. Survival depends more on size of the tumor rather than cell type. The majority of breast cancers are infiltrating duct carcinoma.

Breast cancer appears to be two different diseases: those with disease older than 40 and those with disease younger than 40. Younger women with breast cancer generally have less differentiated, more aggressive, harder to treat malignancies that are more apt to recur and metastasize. Younger women have a more virulent disease.

## Role of Family Physicians

Family physicians have an integral role in the prevention, diagnosis, and management of breast cancer. Primary care physi-

cians are the gatekeepers to health care in the United States. Women should have the best access to their family physician than any other physician. Patients see their primary physician more than any specialists. Family physicians usually have a long-term, trusted and valued relationship with patients. Patients rely on their family physician for the advice given them by other specialists and subspecialists.<sup>2</sup>

Family physicians must be competent in examining the breasts to be able to recognize abnormalities. Concomitantly, they must be able to teach breast self-examination to patients. Physicians must also be familiar with risk factors for breast cancer, how to evaluate breast masses, imaging techniques, and when to refer to breast surgeons for further evaluation.

## Risk Factors for Breast Cancer

Table 1 lists the Risk Factors for Breast Cancer below. Primary Care Physicians should be familiar with these risk factors.<sup>2,3</sup>

## Breast Examination

The physician must be competent at examining breasts of women, men, adolescents, children, and newborns. The primary method of screening for breast cancer is the physical examination, both by a physician and breast self-examination by the patient. Obviously, a physician must be competent in examining the breasts in order to teach breast self-examination to a patient. The breasts should be very easy to examine.

The real advantage to starting breast self-examination early is teaching self-examination when the patient is young with normal smooth breast tissue unadulterated by fibrocystic changes that ensue with time. Patients learn what is always there, and, subsequently, what is new, hard, tender, and has not been there. Some 90% of breast masses are found by women themselves.

**Table 1:** Things that cannot be asked in a medical school application interview

- Premenopausal first degree relative with bilateral breast cancer
- Inherited genetic mutation for breast cancer
- Age>65
- Two or more first degree relatives with breast cancer
- Personal history of breast cancer
- Nodular densities in >3/4 breast volume on mammogram
- Ovaries not surgically removed at <35
- Personal history of cancer of major salivary glands
- Atypical hyperplasia on breast biopsy
- One first-degree relative with breast cancer
- Recent hormone replacement therapy
- Recent oral contraceptive use
- Never breastfed a child
- History of cancer of the endometrium, ovary, or colon
- No full-term pregnancies
- First full-term pregnancy after age 35
- Menopause after age 55
- Menarche <age 12
- Term pregnancy before age 35
- Oophorectomy before age 35
- Menopause before age 45
- Menarche >age 17<sup>2</sup>

Recently, the American Cancer Society has stated that they no longer recommend breast self-examination, because it does not make any difference in long term survival.<sup>1</sup> Fortunately, in the last three decades, women have taken a role in examining their breasts.

### Knowledge of Screening Tests

Knowledge of what screening tests are available and what is recommended at what age is important. While mammography is the gold standard for visualizing the breast, it may not be useful for an 18-year-old with a tender two centimeter new mass. Mammograms, compression views, ultrasound, digital mammography, and MRI are all used to visualize breast tissue. MRI screening is now recommended for women at high risk for breast cancer. Interpretation of tests is also essential. For example, a negative breast ultrasound only means that no cystic masses were seen.

BRCA1 and BRCA2 screening is available. Women who carry these genes have a 50-80% lifetime risk of developing breast cancer. BRCA1 and BRCA 2 account for 50% of inherited breast cancers.<sup>1</sup> Women with these genes are high risk and need MRI screening.<sup>1</sup>

### Evaluation of Breast Masses

Evaluation of a breast mass begins with attention to family history and/or personal history of breast cancer and breast disease. Physician examination includes the mass, any areas in question by the patient, and palpation of the axillary and supraclavicular areas for lymph nodes. A breast mass is a mass until proven otherwise. All masses and mammographic abnormalities must be referred for further assessment. Even if a physician cannot feel a mass, a mass felt by a patient must be assumed and pursued to whatever extent is possible. The physician must examine the breasts and decide what screening tests are appropriate for the patient.

A palpable mass must be explained by consultation, aspiration, biopsy, or excision. The gold standard of diagnosis remains an excisional biopsy. Referral to a breast or general surgeon who is experienced in breast evaluation, breast biopsy, and both conservative and radical surgery including lymphadenectomy is important. The surgeon must also be able to appropriately stage the patient if a malignancy is found and refer the patient for adjuvant therapy if indicated. It is important to explain to the patient what to expect from the referral. It is equally important not to try to speculate what the surgeon may or may not do. Any diagnostic studies and x-rays should be sent to the surgeon. Lastly, the patient should be strongly encouraged to keep the appointment. Table 2 lists some of the pitfalls in evaluation of breast masses.<sup>2</sup>

### Breast Imaging

Originally, mammography was developed to detect breast cancers that were too small to be felt. In fact, mammograms were supposed to be able to detect cancers that otherwise would take five years to become palpable. Mammography will not detect all breast cancers. Mammograms have reduced the

**Table 2:** Pitfalls in the Evaluation of Breast Masses<sup>2</sup>

- Mammogram is negative, so don't worry.
- I cannot afford a mammogram.
- I do not want a mammogram—it hurts.
- Physician cannot feel mass, so don't worry.
- Mammogram is negative; no need for exam.
- Come back in six months; don't worry.
- It is probably OK.
- You are too young to have cancer.

mortality from breast cancer by 35%. If all women over 40 had a yearly mammogram, the mortality could be reduced by 50%.<sup>3</sup> The current recommendations for mammography are listed in Table 3 below.<sup>1</sup>

**Table 3:** Current Guidelines for Mammography<sup>1</sup>

- Screening Mammogram at age 35
- Screening Mammogram at age 30 if Family History
- Annual Mammogram beginning at age 40

In Alabama the radiologist who reads the mammogram is required to send a letter to the patient with an explanation of the results, what, if anything, needs to be done, and when the mammogram needs to be repeated. If the results are inconclusive, what diagnostic tests need to be done next. A report is also sent to the requesting physician. The Guidelines are for *normal breasts*. The radiologist may request a compression or “magnified” view or an ultrasound. The radiologist may recommend a tissue biopsy.

Special considerations include: if the breast are too small for screening, if the breasts or portions thereof have been removed, if the breasts are too large, or if there are breast implants present. It also makes a difference if the implants are above or below the pectoralis muscles. Other considerations include supernumerary breasts and nipples, male breasts, and gynecomastia. Still other challenges include tram flap reconstruction and transgender reassignment surgery. Male breasts with implants and high-dose estrogen hormone replacement therapy raise other questions.<sup>4</sup> The explanation of the Birads Mammographic Diagnoses is listed in Table 4.<sup>3</sup>

**Table 4:** Birads Mammography Diagnoses

Category 0	Additional imaging needed
Category 1	Negative
Category 2	Benign finding
Category 3	Probably benign; short-term follow-up
Category 4	Suspicious; needs biopsy
Category 5	Malignant; needs action

Ultrasound is useful for determining if a mass is cystic or solid. A negative ultrasound does not mean that there is no mass present. Ultrasounds sometimes are useful for masses in women under 30. There is a growing interest in MRI of the breast as the standard of care for women at high risk for malignancy of the breast.<sup>1</sup>

## Chemoprophylaxis

Tamoxifen reduces the risk of breast cancer by 50% if the malignancy is estrogen receptor positive.<sup>1</sup> It also increases the risk of endometrial hyperplasia. Any abnormal uterine bleeding or increase in size of the uterus while on Tamoxifen therapy must be evaluated by an endometrial sampling. Evista reduces the

risk of breast cancer by 70% with no increase in endometrial hyperplasia.<sup>1</sup> Cox 2 Inhibitors are currently being evaluated for receptor negative cancers.<sup>1</sup>

## Treatment of Breast Cancer

The primary treatment of breast cancer is surgical. Chemotherapy, radiation, and hormonal therapy are also used to treat breast cancer. **PRIMARY BREAST CANCER IS OFTEN CURABLE. RECURRENT AND METASTATIC BREAST CANCER IS TREATABLE BUT NOT CURABLE!** All the effort is placed in the initial treatment. Doing everything recommended is essential. The larger the breast cancer, the greater the risk of metastasis and also the lesser the risk of cure.

**Table 5:** Surgical Treatment of Breast Cancer

- Lumpectomy
- Quadrant Resection
- Simple Mastectomy
- Modified Radical Mastectomy
- Radical Mastectomy
- Axillary Lymph Node Dissection
- Sentinel Lymph Node Biopsy

**Table 6:** Staging of Breast Cancer

Stage I	Tumor<2 cm; negative nodes; no metastasis
Stage II	Tumor<5 cm; nodes, if palpable, not fixed; no distant metastasis
Stage III	Tumor>5 cm or invasion of chest or skin; supra-clavicular nodes
Stage IV	Distant metastasis

**Table 7:** Common Pitfalls in Breast Cancer Treatment

- Not enough treatment
- Stopping treatment
- False security with negative nodes
- Too conservative surgery
- Lack of aggressive treatment under age 40
- Not treating long enough
- Stopping surveillance
- Individual patient limitations in treatment
- Cosmetic considerations
- Need for hormone replacement therapy
- Unproven therapy
- Failure to take Tamoxifen
- Health problems limiting therapy<sup>2</sup>

**Table 8:** Common Questions about Breast Cancer Treatment

- Prophylactic bilateral total mastectomies
- Prophylactic subcutaneous mastectomies
- Do I really need a lymph node dissection?
- I am concerned about my arm swelling.
- Will my husband still want sex with me?
- Can I take estrogen?
- My breast cancer was 20 years ago; can I take estrogen?
- My hot flashes are awful; I will sign a release not to sue you, if you will give me some premarin.
- I hate sex and it hurts.
- What can I do about hot flashes?
- What about Evista?
- Can I use vaginal estrogen?
- Is testosterone safe?
- Will progesterone help my hot flashes?
- Can I take the estrogen patches since they are low dose?
- What about Ozone therapy?
- I am BRCA1 and BRCA2 positive and so are my daughters; what should they do?
- Tamoxifen makes me feel bad; can I stop it?
- Is death from breast cancer painful?
- When do I stop treatment?
- When will I die from this?
- What about going to Mexico for experimental treatment?
- Do I need to go to hospice?
- What if I get addicted to narcotics?
- How much is enough adjunctive treatment?
- Should I get my final affairs in order?
- I have unresolved conflicts with my children; should I address those with them?
- Do I need a will?
- Should I have had more extensive surgery?
- I am unsure about my religious beliefs.
- Will death hurt?
- What if I wake up dead tomorrow?
- Is all this suffering in vain?
- Do you believe that I will go to Heaven?

Surgical treatments for breast cancer are listed in Table 5. Surgical treatment ranges from breast-conserving lumpectomy and quadrant resection to variations of mastectomy and lymph node dissection.<sup>2</sup> Table 6 lists the staging of breast cancer.<sup>3</sup> Table 7 lists the common pitfalls in the treatment of breast cancer.<sup>2</sup> As a general rule, patients with a malignancy have better long-term results with treatment at a large cancer treatment center than with an individual physician.

Patients have a longstanding relationship with their family physician. Despite what other specialists and subspecialists tell patients, most patients want confirmation from their own physician. Table 8 lists common primary care questions about breast cancer treatment. Often, patients will ask questions of their primary physician that they will not ask other physicians.

Patients ask these questions of their primary physician and not their treating oncologist. Many of these are straightforward and many are not. Many of these will be answered over time. Very personal questions are truly better left with one's family physician.

Family physicians have an important role in the prevention, diagnosis, and management of breast cancer.

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# Clinical and Diagnostic Findings in Patients with Lumbar Radiculopathy and Polyneuropathy

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## Abstract

### Background

When lumbar radiculopathy and polyneuropathy occur together a complex situation that is capable of causing disability occurs. Physicians need to be able to recognize when these conditions present together and know how to diagnose and treat them.

### Methods

The clinical signs and symptoms, electrodiagnostic findings, and lumbar spine imaging in 70 patients with lumbar radiculopathy and polyneuropathy were analyzed.

### Results

Precisely 27% of patients with lumbar radiculopathy were diagnosed with polyneuropathy of the lower extremities. Patient reports of bilateral neuropathic symptoms with findings of bilateral distal muscle weakness, distal decreased sensation to sharp pin, and ankle reflex diminishment were the most consistent indicators of a polyneuropathy in addition to the lumbar radiculopathy.

### Conclusion

If a patient with low back pain presents with bilateral neuropathic symptoms and signs in the lower extremities, imaging studies and electrodiagnostic studies are recommended to diagnose and treat the radiculopathy and polyneuropathy.

## Introduction

The dominant medical factors associated with the development of disability in patients with low back pain is the presence of severe leg pain and a history of prior episodes of low back pain.<sup>1,2</sup> In patients presenting with leg pain greater than low back pain,

lumbar radiculopathy and stenosis are described as the most common etiologies.<sup>3,4</sup> Lumbar radiculopathy refers to a pathologic process involving the lumbar nerve roots causing radicular symptoms into a lower extremity. The nerve root pathology arises primarily from direct neural compression irrespective of whether the etiology is an acute herniated or displaced disc, bony spurs, foraminal stenosis, central stenosis, or hypermobility of a vertebral segment.<sup>5</sup> The prevalence of lumbar radiculopathy varies from about 2.2% to 8% and the incidence ranges from 0.7% to 9.6%.<sup>6</sup>

Despite the large number of nerve roots subject to potential compromise in the lumbosacral region, approximately 76.1% of lumbar radiculopathies involve the L5 and S1 nerve roots.<sup>5</sup> L5 and S1 radiculopathy results in sensory loss over the dorsum and lateral foot and weakness of ankle and toe extensors and flexors. Although most radiculopathies result in unilateral symptoms, lumbar central canal stenosis can result in single, bilateral, and multilevel lesions which cause bilateral symptoms. Neurogenic claudication with bilateral leg pain, numbness, tingling, weakness, and muscle cramping radiating into the feet upon activity can be symptoms of lumbar stenosis.<sup>7,8</sup> However, patients with radiculopathy and stenosis usually present with low back pain and unilateral more than bilateral leg pains, numbness, and weakness. Physical exam most commonly reveals reduced lumbar range of motion, lumbar paraspinal muscle spasm, and lower extremity muscle weakness, sensory loss, and reflex changes associated with a L4, L5, or S1 radicular pattern.<sup>3</sup>

Diagnosis of lumbar radiculopathy is particularly challenging due to the anatomy involved. In the lumbar spine, the dorsal and ventral lumbar roots exit the spinal cord at the T11-L1 bony level and travel in the lumbar canal as a group of nerve roots in the dural sac known as the 'horse's tail' or cauda equina. Multiple nerve rootlets that are descending in the cauda equina can be affected by a single central disk herniation or single level lumbar central stenosis. For example, a central L3-4 disc herniations

or central canal stenosis can impact the L5 and S1 nerve roots bilaterally.<sup>9</sup> This anatomy poses challenges to the diagnosis of lumbar radiculopathy and locating the compression site.

The most useful test for confirming the presence of a radiculopathy is needle EMG (electromyogram). An EMG study is considered diagnostic for radiculopathy if muscles innervated by adjacent nerve roots are normal but EMG abnormalities are found in two or more muscles innervated by the same nerve root and different peripheral nerves.<sup>3</sup> The needle EMG examination can identify only the root or roots that are physiologically involved, not the precise anatomic site of pathology in the lumbar spinal canal. This is an important limitation which requires correlation with imaging findings to determine the anatomic location of the offending site.<sup>11</sup> The most accurate imaging study to assess neural structures within the lumbar spine is MRI scanning.<sup>16</sup> The needle EMG is helpful however, due to the high false positive rate of lumbar spine MRIs with around 30% of normal subjects having a disk protrusion.<sup>13,14</sup>

Polyneuropathy is a common neurologic disorder affecting the peripheral nerves with a frequency among the general population above 5%.<sup>10</sup> Pathophysiological changes can include: axonal degeneration, axonal atrophy, demyelization, and metabolic changes that alter nerve conduction.<sup>15,16</sup> Presenting symptoms of polyneuropathy are described as pain, dysesthesias, and weakness in the feet and legs.<sup>17,18</sup> Signs and findings associated with polyneuropathy are usually present with bilateral relatively symmetrical distal sensory loss, weakness, and hypoactive or absent reflexes.<sup>13</sup> The sensory loss is described to demonstrate a distal-to-proximal sensory loss gradient of small or large sensory fibers. Signs of sensory loss occur in an acral, nondermatomal, nonsingle-nerve distribution. This varies from the radicular sensory loss due to it presenting in a sock distribution, rather than on the lateral or dorsal foot. Ankle jerks that are relatively depressed or absent are valuable signs of polyneuropathy.<sup>19</sup> Motor signs may include atrophy and weakness of intrinsic foot muscles, including ankle and toe extension and flexion.

The American Association of Neuromuscular and Electrodiagnostic Medicine in conjunction with the American Academy of Neurology and the American Academy of Physical Medicine and Rehabilitation had recently recommended protocols and criteria for diagnosis of distal symmetric polyneuropathy. They determined that the most accurate diagnosis of polyneuropathy comprised of a combination of clinical signs, symptoms, and electrodiagnostic findings. These associations state electrodiagnostic findings should be included as part of the case definition because of their higher level of specificity.<sup>19</sup>

In the researcher's experience referring physicians rarely recognize and acknowledge the potential presence of a peripheral polyneuropathy in addition to a known lumbar radiculopathy. Polyneuropathy confounds the diagnoses of radiculopathy and spinal stenosis in patients known to have diabetes.<sup>20</sup> It is also our experience that a diabetic patient presenting with distal leg greater than low back symptoms is assumed to be suffering from diabetic neuropathy and the additional radiculopathy is

not acknowledged. The presenting symptoms and signs found upon examination of the distal lower extremities are similar between polyneuropathy and lumbar radiculopathy. It is important that the practicing physician be able to recognize symptoms and signs that may be indicative of an overlying polyneuropathy with lumbar radiculopathy. In order to properly diagnose the co-existence of both disorders, imaging studies, and electrodiagnostic tests are needed.

The purpose of this study is to emphasize the importance of using clinical symptoms and signs along with electrodiagnostic and imaging studies to properly diagnose a polyneuropathy with radiculopathy. The frequency of polyneuropathy being diagnosed in patients with lumbar radiculopathy who presented with low back and leg symptoms is studied. The researchers review the common clinical and diagnostic findings in these patients to provide practitioners with the identifiable combination of clinical symptoms and signs that are most indicative of an additional polyneuropathy. The complexity of diagnosing this dual central and peripheral nerve lesion is acknowledged.

## Methods

Patients studied were all referred to a physician specialized in Electrodiagnostic Medicine and Physical Medicine and Rehabilitation for treatment of low back and lower extremity symptoms. The patients seen with lumbar radiculopathy were counted. These patients presented with low back pain and radicular lower extremity symptoms of weakness, numbness, and pain. All these patients had abnormal lumbar MRI findings to confirm the diagnosis of lumbar radiculopathy. Of these patients, the ones clinically suspected and then diagnosed with polyneuropathy were used as the patient sample for this study. This sample of convenience was gathered from January 2009 to October 2009 and consisted of 70 patients. All patients signed informed consent and procedures were in accordance with the Helsinki Declaration.

These 70 patients were all diagnosed with polyneuropathy based on the combination of clinical signs, neuropathic symptoms, and electrodiagnostic findings as established by the American Association of Neuromuscular Electrodiagnostic Medicine. Abnormal electrodiagnostic findings for the diagnosis included abnormal sural sensory and/or peroneal motor in one limb with additional abnormal sensory and/or motor nerve conduction findings in the contra lateral limb in a pattern consistent with neuropathy.<sup>19</sup> Additional nerve conduction testing was completed as indicated by the pattern and severity of the abnormal findings to determine the sensory, motor, axonal, and demyelinating features of the polyneuropathy.

Reported patient symptoms at time of the initial consultation were used in this study. Neuropathic symptoms of numbness, altered sensation, and pain in the feet made by the patient were noted. The physician examined patients for clinical signs of neuropathy. Ankle reflexes were tested and graded utilizing the standard scale of normal equals 2 and absent equals 0. Pin wheel examination was used to document the pattern of sharp pin

**Table 1:** Presenting clinical symptoms and signs in patients diagnosed with polyneuropathy and lumbar radiculopathy (n=70)

Clinical symptoms or signs	Percent of patients Bilateral	Percent of patients unilateral	Percent without symptom or sign
Reported numbness, altered sensation, or pain in the feet	93%	7%	0%
Decreased or absent ankle reflexes	80%	5%	15%
Decreased sensation to sharp pin in feet	96%	4%	0%
Distal toe & ankle extension/flexion weakness	94%	6%	0%

**Table 2:** Summary of abnormal lumbar MRI findings in patients diagnosed with polyneuropathy and lumbar radiculopathy (n=70)

MRI finding	Lumbar central stenosis	Lumbar foraminal stenosis	Lumbar disc displacements
Percent of patients with finding	57%	90%	88%

sensory loss of the legs and feet. Distal muscle strength of ankle and toe extensors and flexors was graded utilizing the standard 0 to 5 grading system. Feet and lower extremities were closely inspected for evidence of muscle atrophy. Once the necessary data was collected, Excel was used to organize the data into tables.

## Results

Seventy out of 255, or 27% of patients referred to the physician with lumbar radiculopathy symptoms in a ten month period were also diagnosed with polyneuropathy of the lower extremities. These 70 patients consisted of 31 males and 39 females with a mean age of 65±10.8. In these patients, polyneuropathy was suspected in addition to the lumbar radiculopathy due to a combination of bilateral distal lower extremity symptoms and bilateral distal lower extremity clinical signs.

The common clinical symptoms and signs recorded upon evaluation of these patients are displayed in Table 1. Bilateral symptoms were reported in 93% of patients while 7% reported only unilateral symptoms. The majority of signs or symptoms found were bilateral in above 90% of patients except for ankle jerks at 80%. All patients had unilateral or bilateral distal toe or ankle weakness, decreased sensation to sharp pin, and complaints of pain, numbness, or altered sensation in the feet. Exactly 15% of patients had normal ankle reflexes, but the remaining 85% had diminished ankle reflexes.

Table 2 summarizes the abnormal MRI lumbar findings in the studied patients. Lumbar foraminal stenosis and lumbar disc displacements were present in around 90% of the patients. Significant degree of central lumbar stenosis was reported in slightly over half of the patients. Central stenosis was less common than foraminal stenosis or disk displacements.

Table 3 presents the common abnormal EMG/NCS (electromyogram/nerve conduction study) findings from electrodiagnostic testing of the patients in the study. Abnormal EMG/NCS findings tended to be bilateral. Sensory sural and EMG abnormalities were the most common followed by peroneal motor abnormalities, and F wave abnormalities were the least common.

## Discussion

The findings of this study verify that the combination of bilateral distal neuropathic symptoms and multiple bilateral clinical signs are the best clinical indicators of additional polyneuropathy in a patient with lumbar radiculopathy. All patients complained of distal neuropathic symptoms of numbness, altered sensation, or pain in their feet. The clinician found decreased sensation to sharp pin and weakness of the distal leg in all patients. Overall, the data states that bilateral distal foot signs and symptoms are the best indicators of an additional polyneuropathy. Precisely 93%-96% of the patients reported bilateral foot numbness or altered sensation, had bilateral decreased distal sensation to sharp pin, and had distal muscle toe extension or flexion weakness. Since radicular leg and low back symptoms were often the primary chief complaint of these patients, the bilateral rather than unilateral clinical findings and signs are crucial in the recognition of the additional polyneuropathy.

Reviewing the symptoms and clinical findings associated with neuropathy and radiculopathy is helpful in the diagnosis of both conditions. Frequently described sensory symptoms with neuropathy include bilateral numbness, burning, prickling paresthesias, dysesthesias, and allodynia involving all toes, both feet, and distal legs in a sock distribution.<sup>19</sup> The sensory symptoms and findings associated with lumbar radiculopathy are usually unilateral and in a single nerve root pattern starting above or

**Table 3:** Summary of abnormal EMG/NCS findings in patients with diagnosed polyneuropathy and lumbar radiculopathy (n=70)

NCS/EMG Finding	Bilateral findings	Unilateral findings	Percent of patients without finding
Abnormal sural sensory	86%	11%	3%
Abnormal peroneal motor	76%	13%	11%
Abnormal F waves	66%	4%	30%
EMG abnormalities	84%	16%	0%

just below the knee and radiating down into the foot. The pattern of sensory loss and paresthesias is over the medial aspect of the calf, ankle, foot, and 1st toe with L4 radiculopathy; lateral knee to anterior ankle, dorsum of the foot, and lateral (2nd-5th) toes with L5 radiculopathy; and lateral mid-distal leg to lateral foot and 5th toe with S1 radiculopathy.<sup>3</sup>

If there is significant weakness secondary to the neuropathy, it initially manifests as distal and bilateral weakness during extension and flexion of the 1st and lateral toes. Weakness will progress up the feet and result in weakness during ankle dorsiflexion and plantarflexion. However, the weakness from a lumbar origin is in a radicular pattern and most commonly unilateral. Lumbar weakness will often involve proximal hip and thigh muscles. This is because hip flexors and adductors along with knee extensors are L2, L3, and L4 innervated while hip extensors and abductors and knee flexors are L5, S1, and S2 innervated. It should also be noted that weakness from L4 and L5 radiculopathy results in weakness of ankle dorsiflexion and 1st toe extension. S1 radiculopathy, however, will most commonly result in weakness of ankle plantarflexion and 1st toe flexion. L5 and S1 lumbar radiculopathy can both result in weakness of lateral toe extension and flexion.<sup>3</sup>

With lumbar radiculopathy, the radicular sensory and motor findings are most commonly unilateral and associated with specific muscle stretch reflex abnormalities of the involved side. S1 radiculopathy results in a reduced or absent ankle jerk reflex. L5 radiculopathy results in alteration of the internal hamstring muscle stretch reflex. An abnormal knee jerk reflex is associated with L3 and L4 radiculopathy. Neuropathy, however, initially results in bilateral reduced and then absent ankle jerk reflexes. The neuropathy then progresses to bilateral alterations of knee jerk reflex and internal hamstring muscle stretch reflexes.<sup>21</sup>

The authors have found using a physical examination template to document the clinical neurologic examination findings to be very helpful in evaluation of patients. By using a template, all abnormal findings from the physical examination are systematically documented for analysis. Plotting the exact distribution of reported abnormal sensations to sharp pin and touch on an anterior and posterior drawing of the body allows clear representation of where the abnormal sensory patterns are present. Grading and recording muscle strength during: hip abduction, adduction, flexion, and extension; knee extension and flexion; ankle dorsiflexion and plantarflexion; and 1<sup>st</sup> and lateral toe extension and flexion are recommended. Ankle, knee, and hamstring reflexes should be graded and recorded. Posture, need to support the trunk throughout the evaluation, gait alterations, and ability to squat, stand, and walk on toes and heels are important to note. An ideal template would also include evaluation for distal muscle atrophy and edema, skin discolorations, pulses, straight leg raising test, and foot deformities such as hammer toes, pes planus, and pes cavus. All abnormal findings from this assessment can then be analyzed. Focus should be on whether the abnormal findings are in a radicular or neuropathic pattern to correctly diagnose lumbar radiculopathy, polyneuropathy, or polyneuropathy with lumbar radiculopathy.

Out of a ten-month sample, 27% of the patients referred to the physician with lumbar radiculopathy were diagnosed with polyneuropathy. The possibility of an additional polyneuropathy should be considered in patients with lumbar radiculopathy when bilateral distal neuropathic symptoms are described and physical examination reveals bilateral distal loss of sharp pin sensation, bilateral distal leg muscles weakness, or abnormal ankle reflexes. If these clinical signs are found in a patient with low back and neck pain, the practitioner should proceed with electrodiagnostic and imaging studies to properly diagnose and treat the polyneuropathy and radiculopathy.

Lumbar MRI imaging revealed varying severity and locations of lumbar pathology to associate with the radiculopathy. Foraminal stenosis and disc displacements were present in nearly all the patients. Central stenosis being present in only 57% of patients strengthens the concept of bilateral signs and symptoms being viable indicators of additional polyneuropathy. Some of the bilateral symptoms could be due to central stenosis. However, the central stenosis could only account for around half of the bilateral symptoms, assuming all incidents of central stenosis caused bilateral symptoms. Overall, literature agrees that the most accurate imaging study to assess neural structures within the lumbar spine is MRI scanning.<sup>16</sup> Correlation of the abnormal lumbar MRI, clinical symptoms and signs, and findings from electrodiagnostic studies is ideal in the diagnosis of lumbar radiculopathy and polyneuropathy.

The data indicated that EMG abnormalities, sural sensory abnormalities, and peroneal motor abnormalities are the best indicators of a polyneuropathy. These findings are consistent with previous literature.<sup>19</sup> Electrodiagnostic studies with EMG/NCS testing are essential for the definite diagnosis of both the polyneuropathy and radiculopathy. Two recent multicenter studies have shown that EMG testing of paraspinal muscles with six to eight other properly chosen muscles in each lower extremity increases the detection of radiculopathy to 96% to 100%. EMG findings of a radiculopathy are described to have a higher level of specificity and are considered diagnostic for radiculopathy.<sup>3,11,19</sup>

If the EMG/NCS initial testing yields abnormal findings, additional testing is needed for proper classification of the polyneuropathy into sensory, motor, axonal, or demyelinating. The proper selection of muscles and nerves for the EMG/NCS testing and interpretation of the recordings for proper diagnosis and classification of radiculopathy or polyneuropathy require the expertise of a well trained physician. It should be noted that non-physicians perform 17% of the EMG/NCS studies in the US.<sup>22</sup> A past study consisting of 6381 diabetic patients undergoing electrodiagnostic testing demonstrated that neuropathy identification rates were six times higher for physiatrists, osteopathic physicians, and neurologists versus podiatrists and physical therapists performing testing despite controlling for case mix differences. These findings state the need for well trained physicians to accurately diagnose patients with these complex cases of polyneuropathy and radiculopathy.<sup>3,23</sup>

When these conditions present together, both the radiculopathy and the polyneuropathy need to be treated in order to assure optimal recovery. Each of these conditions alone can result in disabling lower extremity symptoms. The diagnosis and treatment of an additional polyneuropathy compounding the lower extremity radicular symptoms is crucial. The researchers suspected that an overlying polyneuropathy further exacerbated the central radiculopathy in the group studied. If bilateral symptoms and signs are present, the patient should receive electrodiagnostic and imaging tests to confirm the diagnosis of radiculopathy and polyneuropathy.

Once polyneuropathy is diagnosed, work-up for treatable causes of the polyneuropathy with screening laboratory testing is currently recommended, even in patients with a known cause, such as diabetes. In one trial, 55% of patients with a diagnosis of diabetic polyneuropathy ultimately were found to have additional etiologic or contributory factors.<sup>24</sup> Common causes of polyneuropathy include: autoimmune disorders, chemotherapy, infections, inflammatory nerve disorders, malignancies, medication induced, nutritional deficiencies, renal disorders, and toxic exposures.<sup>25</sup> Treatment of both the radiculopathy and polyneuropathy are needed for optimal patient improvements. Recognizing the initial clinical signs and symptoms of a polyneuropathy and radiculopathy is the first step in assuring the patient receives optimal care and recovery.

## Conclusion

Diagnosis of both conditions is helpful for the patient and physician in understanding, monitoring, and treating the polyneuropathy and radiculopathy. Practitioners need to be aware that the presenting symptoms may be coming from either or both the polyneuropathy or radiculopathy. Proper diagnosis of both conditions requires the recognition of peripheral and central signs and symptoms associated with the dual diagnosis of lumbar radiculopathy and polyneuropathy. The study showed the reliable indicators of additional polyneuropathy are bilateral signs and symptoms including: reported bilateral numbness, altered sensation, or pain in the feet, bilateral distal loss of sharp pin sensation, bilateral distal leg muscles weakness, or abnormal ankle reflexes. Once the initial clinical signs and symptoms are recognized, the patient should receive imaging and electrodiagnostic studies to confirm the diagnosis and describe the nature of the polyneuropathy and radiculopathy. Diagnosis and then treatment of the etiologies of both disorders will yield optimal patient improvements and reduction of future disability.

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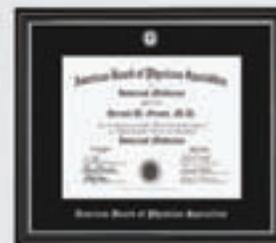
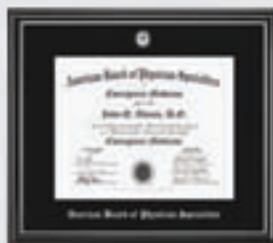
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# Ethical, Legal, and Professional Challenges Posed By “Controlled Medication Seekers” to Healthcare Providers, Part 2

Ken Solis, MD, MA

## Abstract

Abuse and diversion of controlled prescription medications is a large and growing problem in the U.S. The first of this two-part paper discussed the pragmatic, ethical, and legal issues that challenge healthcare professionals who must care for someone suspected or confirmed to be using deception to obtain controlled medications because of addiction to the medication, for resale, recreational use, or other reasons not sanctioned by the medical profession. The second part will focus on the pragmatic, legal, and especially ethical aspects of identifying and caring of patients who are suspected of controlled medication seeking behavior in the acute care setting. It is the hope of the author that this paper will be a catalyst for deeper and wider discussions and research on this difficult, multifaceted, and widespread healthcare issue.

## Should the Diagnosis of “Controlled Medication Seeking Behavior” Ever be Made?

The pragmatic, ethical, and legal challenges that patients with known controlled medication seeking behavior pose to providers are perhaps dwarfed only by the challenge of making the determination that an individual *is* conclusively exhibiting seeking behavior on *this* presentation. For example, in cases of “pseudoaddiction” patients have been misdiagnosed as having “drug seeking behavior” because their undertreated pain led them to seek additional pain relief by going to various other providers.<sup>1</sup> Also, undoubtedly on some occasions patients will exhibit some of the warning flags of seeking behavior listed below because they are cognitively impaired, a friend *did* steal

their medications, they actually have a genuine condition on *this* presentation, and so forth. Thus, the provider often faces substantial challenges in ascertaining whether the patient before them is really seeking a controlled medication for a covert or illicit reason. As a caveat, most authors in the drug abuse literature believe that it is usually better to treat a suspected but unverified seeker’s complaint with a conservative amount of the controlled medication in question, if it is the only viable option, than it is to erroneously misdiagnose “controlled medication seeking behavior” and subsequently mislabel, dismiss, or under-treat a patient with genuine healthcare needs.<sup>2,3,4</sup> In fact, The American Society for Pain Management Nurses believes that the diagnosis of “drug seeking” should be avoided all together because the diagnosis can create prejudice, bias, and barriers to care.<sup>5</sup> These concerns are all legitimate and bear serious consideration. However, if the evidence is compelling that a patient is exhibiting controlled medication seeking behavior, it would be dishonest and inconsistent to avoid making this particular diagnosis to guide treatment while still being willing to make other stigmatizing diagnoses such as “cocaine abuse” or “Munchausen syndrome.” Furthermore, the potential harms from prejudice, bias, and barriers to care posed by this diagnosis must be weighed against the many potential harms to the individual and society in not recognizing, documenting, and properly addressing it as I discussed in part 1 of this article series. The U.S. Drug Enforcement Agency, state licensing agencies, and at least one state court have also concurred that substantial harms can occur and a provider held culpable for prescribing controlled medications to someone who is known to be abusing them.<sup>6,7,8</sup> Similarly, patients and families have blamed emergency physicians for causing their addiction because they gave them narcotics every time they presented.<sup>9</sup>

## Evaluating the Likelihood of Controlled Medication Seeking Behavior

Some providers protest that the evaluation typically needed to determine the likelihood of controlled medication seeking behavior amounts to police work and is, therefore, outside their scope of practice.<sup>10</sup> However, the type and intent of evaluation of a patient with suspected controlled medication seeking behavior does not differ substantially from doing a careful evaluation of other unusual, suspicious, or potentially problematic patient presentations. If certain historical or exam findings raise suspicions of the patient being disingenuous, the provider should do an appropriate evaluation to try to confirm or assuage those suspicions. Additionally, if we are to accept the common assertion that psychological and physiological dependencies to various substances *are* diseases, that they are frequent motivations behind controlled medication seeking behavior,<sup>11,12</sup> then doing an evaluation to make such a diagnosis is within the profession's province. Making an accurate diagnosis should also lead the provider to offer the correct treatment with greater confidence. For example, if the concerns about controlled medication seeking are assuaged, the provider can now treat the condition with less concern about medication abuse potential, or, conversely, if seeking behavior is confirmed, the provider can decide to avoid contributing to an underlying drug dependency and direct them to drug rehabilitation.

## Some Warning Flags of Possible Controlled Medication Seeking Behavior

Studies which attempt to determine sensitive and specific signs or tools for detecting patients who are abusing controlled medications have been applied to chronic pain patients already on narcotics for treatment.<sup>13,14,15,16,17,18,19,20,21</sup> Chou et al. systematically reviewed and critiqued this literature and determined that the nine relevant studies all had methodological shortcomings, and most of the results may not be applicable to primary care or other settings<sup>22</sup> (nor, of course, to non-narcotic medications).

The warning flags listed below are *some* of the most commonly cited warning flags for seeking behavior derived from the two highest quality studies,<sup>16,19</sup> as determined by Chou et al., and the opinions or observations by various experts in the field and drug enforcement agencies.<sup>23,24,25,26</sup> Perhaps unsurprisingly, there appears to be general agreement in how controlled medication seekers tend to present or can be detected in both the more formal chronic pain research and the less formally determined assertions made by experts in other healthcare settings and drug enforcement agencies. Based on face value as well as extrapolating from the chronic pain literature, I have divided the different kinds of "controlled medication seeking" warning flags into those that are likely to have a high probability of indicating controlled medication seeking behavior ("red flags") from those that have a lesser probability of indicating seeking behavior ("yellow flags"). These warning flags might be detected from historical information gleaned from nurses, other personnel such as paramedics, past medical records, or

the provider's own interview with the patient. The detection of a warning flag(s) should often prompt the provider to look for other flags, particularly if it is a flag that is a strong indicator of seeking behavior in that patient's particular context.

### Red flags of controlled medication behavior (strong evidence):

- Patient believes that he/she is addicted to medications or has undergone narcotic detoxification in the past.
- Patient frequents different providers, institutions, or pharmacies in a short time period to obtain controlled medications (especially if patient denies the practice).
- Steals or diverts prescriptions from family members, e.g., brings a dependent family member in for an alleged condition, but then takes the medication for themselves.
- Obtains controlled medications from non-medical sources, such as the "street."
- Steals medical goods, such as prescription pads or syringes.
- Forges or alters a prescription for a controlled medication.
- Frequently loses the controlled medication by misplacing it, having it stolen, etc.
- Notification by another provider, institution, or a family member that the patient is addicted to controlled medications.
- Has drug-related deterioration in work performance, family relationships, or other social dynamics.
- Concurrently abuses illicit drugs, e.g., positive urine drug screen for illicit drugs.
- Asserts that they take a controlled medication regularly and recently for their condition, but the urine drug screen is negative; are they diverting the medication for resale? (Caution: check with your laboratory to determine their screen's sensitivity in detecting the medication in question.)
- Patient gives false identification information.
- Patient injects an oral formulation of the controlled medication.

### Yellow flags for possible drug seeking behavior (less strong evidence):

- Patient frequently visits or contacts your facility with requests for refills of the controlled medication or has multiple unsanctioned escalations in the dose. (Caution: evaluate to determine if symptoms are genuine and they simply are not being adequately managed.)
- The patient is away from home or has passed by closer healthcare facilities and presents with a subjective condition that typically requires controlled medications.
- Gives an improbable story for running out of a medication, e.g., "I accidentally flushed them down the toilet," "my dog ate them . . ."

- States that they are allergic or intolerant of every other class of relevant medication besides the directly or indirectly requested controlled medication or its class.
- Has an unusual amount of knowledge about the controlled medication in question or requests a particular controlled medication by name. (Caution: the patient might also simply be familiar with their condition and knows what works from past experience.)
- Has little interest in the diagnosis or alternative treatments.
- Fails to keep appointments with other providers who are necessary for referral or continuity of care.
- Uses the controlled medication for purposes other than for which it was prescribed, e.g., using a narcotic to decrease anxiety.
- Other healthcare professionals have terminated care of the patient due to concern of controlled medication abuse.
- History of abuse of other substances, such as alcohol or recreational drugs.

Again, although multiple red and yellow flags might strongly suggest that a patient has at least a history of controlled medication seeking behavior, they do not guarantee that a patient does not have a genuine condition on *this* presentation; thus, a careful evaluation and treatment decision must still be made each time – seekers are also mortal and, thus, will eventually become genuinely ill or injured like everyone else.

### Additional Historical Elements

Chronic pain patients, at least, often significantly underreport their medication use.<sup>27,28</sup> Nevertheless, providers should obtain a detailed history of recent controlled medication use, including the last time that they have had them administered or prescribed, the amount, by whom, and the approximate frequency of use. If the provider is concerned about a particular class of medications, they should recount the various names of medications within that class to the patient rather than just naming one or using medical jargon. Some individuals will later state that they did not understand what the provider meant if they ask, “Have you had any narcotics or benzodiazepines lately?” Also, consider asking questions that might be relevant from the list of warning flags listed above, e.g., “do you have a past history of addiction or dependency to any substance, such as alcohol, cocaine, or other recreational drug?”

If the patient reports being intolerant or allergic to multiple medications, the provider should determine the exact nature of those past reactions. Some drug reactions can be addressed, and the patient might be willing to try a non-controlled medication again if proper precautions can be taken, e.g., giving diphenhydramine *before* prochlorperazine for a migraine headache to decrease the risk of akathisia. Similarly, the provider will need to determine if the patient is taking other medications or substances that can adversely interact with many controlled medications and preclude, or at least mitigate, their use. For

example, an alcoholic who is taking a benzodiazepine for anxiety should not be prescribed narcotics due to the increased risk of causing apnea when combined with the other sedating substances that they already use.

### Exam Elements

If an objective exam or diagnostic test finding corroborates a patient’s complaint (e.g., the presence of a tooth abscess on exam), the provider should generally offer the most effective medication, even if the patient has a history of controlled prescription medication seeking behavior. Of course, measures to try and limit the risk of reinforcing a tendency towards addictive behavior should be taken, such as counseling, prompt definitive treatment of the problem, limited prescription of the medication in question, and avoiding medications with high abuse potential, such as meperidine.<sup>26</sup> Conversely, if an exam finding or witnessed patient behavior is fully out of character with a presenting complaint that has no objective corroboration, the observation should be documented, discussed with the patient, and considered in the overall evaluation.

### Medical Records

The provider should check for a patient’s local medical records, even if the patient claims to have never been at the clinic or institution previously. The patient might have forgotten or wish to conceal previous visits to your institution that could demonstrate a pattern consistent with controlled medication seeking behavior. Many emergency departments also have a separate in-department file of patients that habitually frequent them.<sup>29</sup> The usual purpose of these files, which are separate from routine medical records, is to help a provider determine if a patient has habitually used the emergency department to obtain controlled medications for non-therapeutic reasons.<sup>30</sup> In many, if not most, institutions, these files are not created and maintained via a formal, consistent process<sup>31</sup> and, therefore, could present ethical, legal, and practical problems for the provider and the healthcare institution alike as opined by Doctor Geiderman of the Cedar-Sinai Center for Health Care Ethics, Burns and Allen Research Institute. Nevertheless, he also believes that “habitual patient files” can be pragmatically, ethically, and legally defensible if at least the following precautions are taken: 1) obtain legal counsel to determine if and how such a file can be created and managed within federal and state laws; 2) avoid pejorative terms, such as “kook book” or “frequent flyers” for naming the file and avoid pejorative terms in regards to the patient; 3) there should be a formal and rigorous process for creating, maintaining, and accessing the file; 4) the information should not be shared with other parties without the patient’s consent; and 5) the file should be used as a tool only and not as the final arbiter for making a decision about a patient’s diagnosis or treatment. In addition, these files can also be used for the purpose of describing typical presentations, treatment plans, or other important information for patients who present with some frequency to your service with known recurrent medical conditions, such as sickle cell disease, seizures, and the like.<sup>32</sup>

Another source of information of possible past controlled medication seeking behavior is the patient's medical records from other providers or healthcare institutions. Healthcare information can legally be shared with other healthcare professionals *for treatment purposes* according to the Health Insurance Portability and Accountability Act (HIPAA) and does not necessitate a patient's consent.<sup>33</sup> However, state patient privacy laws<sup>34</sup> or institutional regulations might require a patient's written consent before you can obtain medical records from another party. Hence, in many cases the patient will have to be willing to sign a "release of medical information" form before you can receive confidential information from another institution or provider, even though it is not required by federal law.

### Other Sources of Information

Pharmacy records are another frequently helpful resource for determining a patient's history of controlled medication use. Some retail chains, such as Walgreen's® and CVS®, have proprietary national databases that include the prescriptions filled for an individual anywhere within the chain and is accessible by contacting any of their stores.<sup>35,36</sup> Also, with internet search engines, such as Google Map®, it is easy to quickly find the phone number of various pharmacies close to a patient's address by simply typing in their address followed by the word "pharmacy" in the search box.

Additionally, currently 34 states have legislatively authorized databases called "prescription drug monitoring programs" (PDMPs) that record various prescription information for different scheduled medications (the exact information recorded varies by state). Ten more states have either enacted legislation or are considering legislation to initiate PDMPs.<sup>37</sup> Many state PDMPs allow providers to access these databases to help achieve their major stated goals that include "to help identify and deter or prevent drug abuse and diversion; and to facilitate and encourage the identification, intervention with and treatment of persons addicted to prescription drugs."<sup>38</sup> Admittedly, PDMPs are not without their detractors who express concern that the databases compromise patient privacy and might at times deter providers from prescribing controlled medications, even when it is indicated.<sup>39</sup> As with any medical record, it is preferable professionally and ethically to ask a patient for their permission to call pharmacies or state databanks for prescription information, even if not required by federal law.

### What to Do?! A Generalized Approach to Patients Suspected of Controlled Medication Seeking Behavior

Determining how best to *specifically* manage a particular individual that a provider suspects or confirms of controlled medication seeking behavior is beyond the scope of this paper. Available resources, local medical norms, various laws or institutional policies, and especially the patient's particular situation will often constrain or even dictate a provider's options. The

following general approach attempts to maximize beneficence or "doing good" for the patient while minimizing the potential for maleficence or "doing harm" and attempts to preserve or restore a functional patient-provider relationship. This approach also hopes to improve the future likelihood of attaining these ideals as well. The autonomy of the patient is also respected within the framework of the law, unless it is determined that they are very likely or conclusively illicitly seeking controlled prescription medications. Finally, even though confirmed controlled medication seekers are typically acting criminally due to their violation of laws surrounding the procurement of controlled medications, the proposed approach avoids compelling the provider to contact legal authorities unless the patient is egregiously breaking the law, is a potential harm to others or imminently to themselves. In the end, the potential harms to the individual and society that could be mitigated by reporting controlled medication abuse to legal authorities must be weighed against the potential harms of compromising the patient-provider relationship from the loss of patient confidentiality and fidelity. The proposed approach first relies on the provider's determination of the likelihood that the patient is inappropriately seeking controlled medications.

### Controlled Medication Seeking Unlikely or Inconclusive

If the evaluation reveals that controlled medication seeking is unlikely or inconclusive, the provider should treat the patient's condition as appropriate. In general, narcotics and other controlled substances should be used only if other more specific or less potentially harmful approaches have been tried and failed, e.g., bupivacaine dental block for a toothache, acetaminophen for a backache. The provider should also consider having the patient "help you help them" so that the warning flags that popped up during their evaluation pose less of a problem for them with future encounters with the healthcare system. For example, the provider should encourage them to work with their primary provider or specialist as much as possible, minimize the use of emergency or urgent care centers where the efficacy and side effects, including the development of dependency of controlled medications, are difficult to monitor; they should anticipate if they will run short of the medication before the pharmacies are closed, etc. In many cases, the provider will find it helpful to contact the primary provider or specialist during office hours to develop a complimentary and perhaps formal written treatment plan for the patient. If the patient does not have a primary provider due to their financial situation or other circumstances, the provider or another staff person (e.g., social worker) might be able to help the patient find one at an affordable or charitable clinic.

The provider can also consider more formally informing the patient regarding the limits of what they can and cannot do for them according to what they believe is both effective and safe. As an example, some primary providers and pain specialists develop and utilize a "pain management contract" which de-

lineates the boundaries of who, what, how much, and how controlled medications will be prescribed; states that compliance is expected with treatment plans, tests, referrals, and the law; and spells out the consequences of departure from the contract.<sup>40</sup>

### Controlled Medication Seeking Behavior Very Likely or Conclusive

If the provider has determined that a patient is very likely or conclusively exhibiting controlled medication seeking behavior, it is preferable to address the problem appropriately rather than “giving them a few pills to get them out the door.” This tactic only ensures that they will likely return at another time, contributes to potential harms, and does not address the underlying problem. In a similar vein, a provider would not provide an alcoholic with a few beers just to get them out the door. Obviously, it is preferable for the patient to admit to a drug-related problem and get appropriate help rather than to just go away. However, until they are willing to admit to the problem and accept help, there usually is little that a provider can do other than to avoid continuing to contribute to the controlled medication seeking and attendant behaviors. Instead, offer your determination to the individual, preferably with a chaperone at your side because you might need their support and to be a witness during this sometimes difficult “confrontation.”

A particularly challenging situation is the patient who exhibits various conclusive warning flags of controlled medication seeking behavior, but also is believed to have a genuine chronic or recurrent condition that warrants the use of the medication that is being abused. The many psychological and physiological changes that occur with conditions such as chronic pain, anxiety, and the use of narcotics or other medications that affect the nervous system is far beyond the scope of this paper – and is a rapidly evolving area of healthcare science. Nevertheless, in this situation, a thorough assessment and frank discussion is needed to determine why the patient is exhibiting these behaviors. If the symptom is simply not being adequately treated, then perhaps a different treatment regimen or referral to an appropriate specialist should be considered. If the patient has become addicted to the psychological effects of the drug, then a referral to a multidisciplinary team with an addiction specialist, specialist of the underlying medical condition, and perhaps other disciplines might be in order. In either case, the benefits of symptom control must be carefully weighed and guarded against the potential harms from the potential abuse of the controlled medication. As noted above, a “pain management contract” can also be a helpful tool for steering the patient towards a regimen which strives to meet their needs for symptom control and dissuades abuse of the medication(s) in question.

When the provider gives the evidence to the patient that supports the conclusion that they cannot in good conscience provide them with a controlled medication and offers to help them seek more appropriate alternative care, I have received several responses that seem to be most common:

1. *“Okay, I have a problem with drugs and I would like to get help.”* While this response is not as common as we would like, when it does occur, the provider now has, and hopefully will continue to have, a patient that has become a truthful, genuine, and licit patient with whom the provider can work to get the proper treatment. If the patient agrees to rehabilitation, but also requests controlled medications to prevent withdrawal, it is important to note the constraints dictated by federal DEA regulation [21 CFR 1306.07(b)], which only allows a provider who does not have a special license to treat for drug dependency or addiction to administer (and not prescribe) enough medication to prevent withdrawal on three separate days and in lieu of arranging rehabilitation.

2. *“That’s not me you’re talking about, it’s someone else.”* This response typically occurs when the provider presents the individual with evidence that a call to a pharmacy or state prescription drug monitoring program reveals a pattern of controlled medication use that is substantially inconsistent with the history that they offered earlier. In this case, the individual is alleging either a mistaken identity or identity theft. Therefore, the provider should double-check the spelling of the patient’s full name and birth date with their informational resource. If mistaken identity is ruled out, the individual has the option of contacting the police, so that the alleged “identity theft” can be investigated. Given the frequency that this objection has been given to me compared to the actual likelihood that identity theft is being practiced at the very pharmacies or other healthcare institutions in their locale, I’ve been reluctant to accept their allegation and prescribe the controlled medication in question. Furthermore, I have never learned of a patient who actually involved the police to address the alleged identity theft.

3. *“I don’t want any controlled prescription drug; but what are you going to do for my symptoms!”* In this scenario, the patient is indicating that the provider has misunderstood their true intention, and the provider may very well have to be sure that they *have* seriously evaluated their stated problem rather than just focusing on possible controlled medication seeking behavior. Nevertheless, in many cases the patient will already have typically stated or implied that every non-controlled medication is ineffective or causes side effects, and various other warning flags of seeking behavior have also been discovered. Therefore, the provider is often left with the option of trying a different non-controlled medication that they might not have tried in the past or simply stating that they do not have anything to offer the individual with which they are comfortable administering or prescribing. If the evidence of controlled medication seeking behavior is not incontestable and the provider believes that it is possible that the patient is being honest, then the provider will have to use his/her judgment to decide if and how liberally they will treat them until the issue can be fully resolved.

4. *“I’m not a drug abuser, you #@%\*&!” (or worse).* This is a situation in which a chaperone is particularly helpful to act as visible support for the provider and as a witness. If the patient could react violently, the provider should also take precautionary measures, such as having extra personnel close by, keeping

themselves between the patient and the door, etc. The provider should also try to distance themselves emotionally from the confrontation as much as possible and try to de-escalate the situation by indicating that they are here to help them, but only in a way that they believe is medically safe and appropriate. If necessary, the provider should indicate that this behavior is not conducive for a working relationship and will not persuade them to change their mind. If past encounters with the patient indicate that he or she is potentially violent or particularly malicious, the provider should consider having security or law enforcement close at hand for everyone's safety when they confront them. Note that HIPAA does allow for contacting law enforcement when an individual commits a crime on the premises of a healthcare provider<sup>41</sup> and Wisconsin Statute 940.20 (7) makes it a felony to assault emergency healthcare workers.

### Contacting Law Enforcement

Besides concerns for personal safety or the disruption of the healthcare setting noted above, there are other circumstances where the provider might consider contacting law enforcement: (1) the drug seeking behavior is egregious (e.g., theft or tampering of prescriptions or other medical goods, witnessed to be reselling the drug); (2) the patient poses an imminent threat to public safety, such as driving away in a vehicle after getting a medication despite your warning that they must have somebody else drive them home; and (3) if the patient is an imminent threat to themselves. The final criterion is perhaps the most unlikely and difficult for a provider to make. Contacting legal authorities due to the concern of self-harm is theoretically defensible because controlled medications are involved in about 30% of drug-related deaths.<sup>42</sup> However, this situation should only occur if a provider discovers that the patient has a serious problem with controlled medications *after* they have been discharged, because a provider should not logically have provided the medication otherwise. The rarity of being compelled to contact legal authorities for fear of patient self-harm is compounded by the fact that, while controlled medications are involved in a substantial percentage of drug-related deaths, the odds of any single drug use event causing substantial harm is undoubtedly substantially smaller. Therefore, the decision that a recent encounter warrants contacting law enforcement because of the potential for patient self-harm requires considered judgment.

### Conclusion

Individuals who feign or exaggerate medical conditions to obtain controlled medications from the healthcare system are a large and growing problem. The duty that providers have to treat patients' pain, anxiety, or other conditions that are often treated with controlled medications adds to the various ethical, legal, and professional challenges posed by individuals suspected of seeking behavior. Before diagnosing an individual with "controlled medication seeking behavior," the provider must gather enough reliable supporting information to achieve the high evidentiary standards needed to make this diagnosis,

which carries the attendant stigmatizing label of "drug seeker" and could bias future medical evaluations of the patient. Additionally, even in patients with a definite history of controlled medication seeking behavior, the provider must do a careful and thorough evaluation and not presume that their presenting condition is non-genuine yet again. If the provider determines that he has enough evidence to be certain that a patient is exhibiting controlled medication seeking behavior, then a professional approach, honesty, moral courage, and perhaps support staff are required to guide the patient in the right direction.

Exactly how providers should best relate to and manage controlled medication seekers while still meeting their own ethical, legal, and professional duties requires more reflection, perspectives, and empirical research. For example, the University of Wisconsin Hospital emergency department reported remarkable success in decreasing the *apparent* use of narcotics of patients who had greater than ten annual visits to their ER for nonmalignant pain and no co-morbidities, by sending them and their primary provider a letter that stated that they would no longer receive narcotics for their subjective pain and why. However, given the study design limitations, it is not clear if the patients truly dramatically decreased their use of narcotics or simply obtained them from other sources. More discussions are also needed to decide:

- How reliable are the warning flags of controlled medication seeking behavior listed by the DEA and other authorities?
- At what threshold and what kind of controlled medication seeking behavior should a physician report a verified seeker to legal authorities?
- How effective and accurate are existing state prescription drug monitoring programs in assisting providers to better detect controlled medication seekers, and are patient privacy rights still adequately protected?
- Which strategies are most effective at persuading controlled medication seekers to get the true treatment that they need – assuming that they are dependent or addicted to the medication and not simply obtaining them for resale or for a recreational "high."

In conclusion, the healthcare professions need to come to a consensus on how providers with aspirations of maintaining various duties to the patient and the community at large can most effectively detect, relate to, diagnose, and treat patients who are harming themselves and the very system that is supposed to help them with their genuine healthcare needs.

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# MEDICAL-LEGAL

## Legal Guidelines in Interviewing Medical School Applicants

Daniel M. Avery, Jr., MD  
 Daniel M. Avery, III, MD  
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Interviewing applicants for medical school is a very important function of the medical school education system. However, there are many questions that are not appropriate to ask applicants. While some would be useful to know, there are many one cannot ask, unless the applicant first brings them up.

In 1973, very few women applied to medical school and even professional schools as a whole. The following is a true story about a friend who applied to medical school.

### Case Study

A young woman in graduate school applied to medical school. During the interview, the woman was asked if she was married, and she responded, "Yes." She was then asked her age. But then the interviewer asked, "What if you were on call and you and your husband were having sex and the hospital called for you to come to the emergency room to attend one of your patients in cardiac arrest? What would you do?" The young woman responded, "I would go to the hospital like any physician." The young woman kept her cool. When the interview was over, the interviewer asked her how she felt about the interview. She replied, "Good, my uncle is the Dean of the Dental School and President of the University."

### Discussion

In 2010 one cannot ask these questions when interviewing prospective medical students. Table 1 lists the questions that cannot be asked in a medical school interview today. Some seem benign, such as, "How old are you?" which is usually found on

most applications. Some medical schools have established age limits on medical school applicants, based on the premise that if someone who is older completes medical school and residency, he or she will have less time to practice medicine and serve society. In "the old days," age did not matter. One of the authors had many in his medical school class in their 30s, ten in their 40s, and several in their 50s, one of whom was the class president. One is uncertain if age really matters.

At one time it was traditional – and *very honest* – to ask how an applicant planned to pay for medical school. This led to a discussion about loans and scholarships as well as the need for physicians in rural areas and the willingness for communities to offer stipends for a graduate to return to their area to practice medicine. Debts, credit, and difficulties thereof are not important.

Marital status used to be an important question because married students were thought to be more stable and mature. Their marital responsibilities mandated that they do well in school and not fail. Single students were thought to party more, although that is of doubtful importance.

One cannot ask about children. However, if brought up by the applicant, it could be discussed by the interviewer. "Are you pregnant or plan to get pregnant or have you ever had an abortion?" is not appropriate now or in the past. Along with the above, an individual's method of contraception should not be asked and certainly not whether they are sexually active. Today, that would be considered sexual abuse or at least sexual misconduct.

Religious beliefs should not be an issue in applying to medical school. What difference should it make? One's national-

**Table 1:** Things that cannot be asked in a medical school application interview

- How old are you?
- Where are you from?
- What nationality are you?
- Do you believe in God?
- Where do you go to church?
- What is your religious preference?
- What political party do you belong to?
- Why is your hair so long?
- Always had the ring in your nose?
- Do you have any tattoos?
- Do you have any learning disabilities?
- Do you do drugs?
- How much do you drink?
  
- How much money do you have in the bank?
- Ever declared bankruptcy?
- Ever defaulted on a loan?
- How do you plan to pay for medical school?
  
- Are you married?
- Do you have children?
- Are you pregnant or plan to get pregnant during medical school?
- Do you plan to get married in medical school?
- Are you living with someone?
- Have you ever been married?
- How old were you when you got married?
  
- Are you sexually active?
- Ever had sex with someone of your own sex?
- Are you gay?
- Are you on birth control pills?
- Method of contraception?
- Have you ever had an abortion?
- Do you have AIDS or any other STD?

ity begs the same question. However, there are many primary care residency and fellowship programs that are funded by state funds with a commitment to practice in that state. Where one is from may well be an issue as to where they plan to practice. Students from rural areas are more apt to return to rural areas. So, in this context, along with the tremendous need for care in those areas, it may be prudent to know where a student is from. Rural programs for medical clerkships, such as the TERM Program (Tuscaloosa Experience in Rural Medicine), require applicants to come from rural areas.

Sexual preference and experience also have no place in determining if an applicant would be a good medical student. Partying is not a question to ask. To the contrary, it would be useful to know if there was a drug or alcohol problem. With random and cause-and-effect drug screening for physicians and soon-to-be urine drug screens as a part of the application for hospital privileges, drug screening will eventually make its way to medical school applicants as well.

There are, however, issues of professionalism in how medical students present themselves to patients, and these are issues with the Liaison Committee for Medical Education (LCME) as well. Tattoos, inappropriate rings, and modern hair designs are to be discussed. **Patient perception and appreciation of students and their learning to meet patients are important.** Medical students, shadowing students, and pre-med students are required to meet patients with a professional appearance at the University of Alabama School of Medicine in Tuscaloosa, Alabama. To participate in its OB/GYN clerkship, no long hair or beards and no obvious rings or tattoos are allowed. Students who do not comb or brush their hair are asked to do so. The current trend of males to wet their hair, apply mousse or oil, and leave it standing up or unkempt is not appropriate.

### What Can You Ask?

Traditional important questions to ask applicants for medical school include: why does the individual want to become a physician, what medical experiences have they had that have stimulated their interest in medicine, and how have their academic performances been? Many of the above topics can be discussed if the applicant brings them up, such as marriage, children, and the church they attend. It is also appropriate to ask at what point in their life did they decide they wanted to go to medical school.

After my first orientation to interviewing applicants, I wondered if I should do any more than just introduce myself, to be on the safe side. My experience has found applicants who really did want to attend medical school and others who did not. One young woman really wanted to get married and have children, not go to medical school. There are some who felt that it was the right thing to do, but uncertain if it was right for them. But there are many that have worked very hard to get to this point in the process and will make wonderful physicians.

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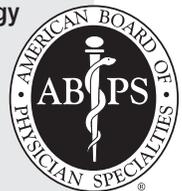
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