Redress Information & Analysis

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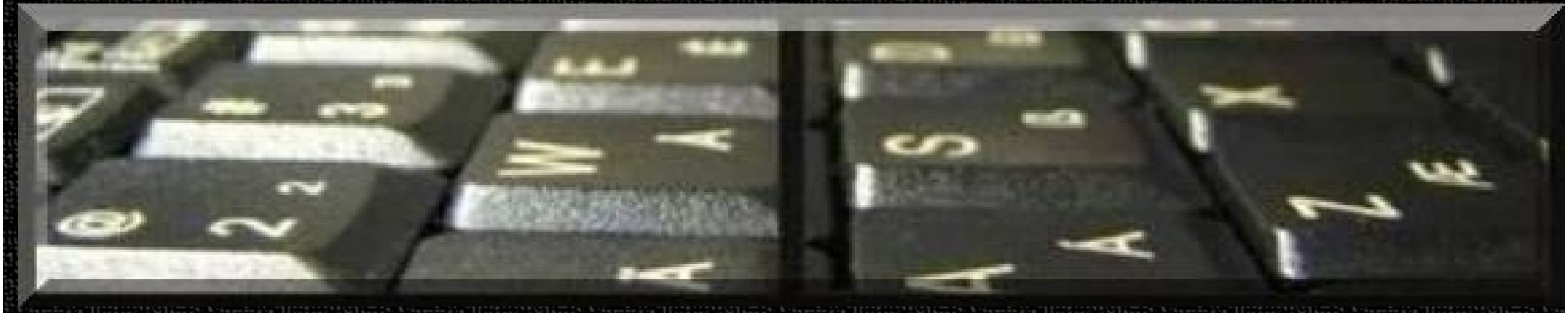

The Arlene Berry Story | Update 2014

INTRODUCTION

Arlene Berry died suddenly and unexpectedly at the early age of 41, less than 24 hours after being admitted to the Kirkland and District Hospital on May 23rd of 2000. There is a public interest in knowing how Arlene Berry came to her death and how her health care providers are implicated.

Arlene Berry was a robust young lady filled with energy and wherever she went it was like spring, like a breath of fresh air. She believed that fishing was a peaceful means of tackling life's stresses while enjoying quality outdoor time with friends and loved ones and especially her children. She was

an avid fishergirl, hiker, camper, and mother of two (a boy and a girl). Both children were only in their early teens at the time of their mother's death. Although her daughter had come of age and was by then living on her own, Arene Berry still cared for her son, the youngest of the two, until her death in May of 2000.

It is interesting to note that Arlene Berry had a history of working in and around logging camps in northeastern Ontario, primarily in reforestation activities, such as "tree planting" jobs between Matachewan and Kirkland Lake. The odd job consisted of cleanup work, such as slashing, gathering, piling and burning dead brushwood. Although she smoked, she was never a heavy smoker. She was an avid angler and loved fishing, hunting, camping, hiking and cooking outdoors. A newfound hobby included gathering driftwood, pine cones, lichen, and spagnum mosses, as well as various ground pines with resultant exposure to fungal antigens, collected usually in mid fall of the year when high humidity and cool temperatures prevailed, and used for creating crafts and curios of all kinds.

The Story Behind The Story

This story behind the story begins with a trip from northeastern Ontario to northwestern Ontario and a several day camp out in Thunder Bay District at the Stillwater Creek Tent and RV Park on Highway 17 just west of Nipigon during the mid summer of 1998. The campground comprises 48+ sites, some in wooded area, some along Stillwater Creek and excepting a hiking trail of medium difficulty, is surrounded with pine and other varieties of densely wooded forest. The creek runs through the property crossing under the highway just in front of the campground.

One day, while fishing along the trout stream on the other side of the highway, opposite the campground, Arlene had a bad fall in rugged terrain, landing on an old fallen tree stump. The end result, a left sided rib fracture(s). A fractured rib could easily puncture the lung resulting in partial or

complete collapse of the lung. Rib injuries include bruises, torn cartilage and bone fractures. Fractured rib ends can lacerate the pleura or lung, leading to the formation of pulmonary hematomas, hemothorax, or pneumothorax.

Within a day or so of her injury, Arlene asserted "it feels like air leaking inside of me, suggestive of pneumothorax, or atelectasis. Since both conditions involve a collapsed lung, symptoms are similar and can range from mild and barely noticeable to severe or chronic. Lung atelectasis occurs as a result of scarring or fibrosis that reduces lung expansion. Common aetiologies include pleural effusion, pneumothorax, granulomatous disease, necrotizing pneumonia and fibrosis.

The injury seemingly resolved just as quickly as it appeared with nothing more than a good night's rest and some ibuprofen, a non-prescription NSAID (non-steroidal anti-inflammatory drug) that is commonly used for skeletal pain and inflammation. Notably, a small pneumothorax may resolve on its own and require no specific treatment beyond rest. At any rate, Arlene did not perceive her injury serious enough to warrant medical attention at that time.

Atelectasis is a collapse of lung tissue affecting part or all of one lung. This condition prevents normal oxygen absoption to healthy tissues. The most common mechanism is due to sharp bony points arising from a rib fracture penetrating pleura and damaging lung tissue. Pneumothorax occurs in about 14% to 35% of rib fractures. Clinical signs and symptoms are nonspecific and include fatigue, weight loss, general malaise, and, less commonly, fever. About one-half of patients remain asymptomatic. Lung atelectasis and localized acute lung injury are factors likely responsible for this unusual histology, and along with the clinical history are important in recognizing the benign nature of this lesion, reportedly "mistaken for adenocarcinoma". Further, Adenocarcinoma, the most common type of lung cancer, is usually located on the outer surface of the lungs (periphery) and can also be mimicked by pulmonary sarcoidosis.

Pulmonary fibrosis (lung scarring) is a condition in which lung tissue becomes damaged and scar tissue forms. In time these mature into scar tissue forming permanent adhesions. Sometimes lung scars are small and do not represent a serious problem. In fact, a person may have small or isolated lung scars without even knowing it. On the other hand, large scars or scarring that is spread over a large area of lung tissue can cause breathing difficulties, shortness of breath, and coughing, which may make it hard for a person to do physical activity. Severe scarring (fibrosis of the lungs can also cause respiratory failure. Patients with badly scarred lungs, heart or muscle involvement complain of tiredness, fatigue or lethargy due to lack of oxygen and increased work of breathing.

The sharp edges of rib fractures have also been known to caused aortic lacerations. Chest trauma may also cause a ruptured aorta and even seemingly minor mechanisms of injury can result in aortic laceration. Lacerations resolve more slowly than contusions, and clearance may take weeks or even months, and they usually end in residual scarring, and residual masses seen on CT are also scar tissue.

Mechanism of delayed aortic injury in left-sided rib fractures have also been reported [Ann Thorac Surg. 2009]. Concomitant injuries tend to mask secondary diaphragmatic injury, which along with a delayed diagnosis often cause later symptoms, e.g., late complications (months to years after injury) such as bowel incarceration or strangulation have also been known to occur.

For the record, pulmonary fibrosis describes a group of diseases which produce interstitial lung damage "mimicking lung malignancy". Notably, if you have a collapsed lung, you are more likely to have another one in the future. A delayed pneumothorax occurs in up to 12 percent of patients with penetrating thoracic injuries that initially appear to be innocuous. Most etiologies are benign in nature, however, there are several syndromes, including predisposing factors. Predictors of recurrence include pulmonary fibrosis, "scarring of the lung". Whatsrmore, pulmonary fibrosis has also been known to “masquerade” as metastatic lung cancer.

The most frequent cases of pulmonary fibrosis are related to sarcoidosis — fibrosis associated with certain occupational diseases. Pulmonary fibrosis occurs in 20%–25% of the patients with sarcoidosis. It commonly affects young adults of both sexes, with a preponderence towards people from certain geographical regions, particularly women. Sarcoidosis is a rare disease that results from inflammation. It commonly affects the lungs and is the most common of the scarring lung disorders. People who have sarcoidosis are predisposed to pulmonary fibrosis (i.e., the hardening and thickening of tissue), although it is not clear if fibrotic processes are active from the onset of sarcoidosis in predisposed individuals, or whether a profibrotic state develops as a response to ongoing inflammation. Within 2 to 5 years, about 25% of those with Sarcoidosis will develop "residual fibrosis" in the lungs or elsewhere, ie., "residual disease on the aorta".

Sarcoidosis is a systemic disorder with a wide variety of clinical and radiologic manifestations, resulting in a variety of complaints. The disease presentation differs in different parts of the world. It appears to be more common in cooler climates, but the reason for this is unclear. Sarcoidosis shows a predilection for adults <40 yrs, peaking between 20–29 yrs. There is a slight female predominance. Sarcoidosis is an enigmatic multisystemic disease caused by an overactive immune system allowing inflammation to spread out of control. Granulomas in the lungs or elsewhere are not considered malignant growths. Furthermore, many pathologists will also mistakenly classify a biopsy of sarcoid tissue as cancer when it is not. Faulty reasoning, low qualification, gross medical negligence or incompetence may predispose.

Ninety percent or more of people with sarcoidosis have lung involvement, whether they have symptoms or not. Common lung symptoms are dry coughing, trouble breathing, wheezing, or pain with breathing, chest pain, tightness, or discomfort and occasionally coughing up blood, which is rare, especially in the early stages of sarcoidosis. Past research suggests that sensitivity to environmental factors may be associated with sarcoidosis risk. It is widely believed that sarcoidosis

may be caused by a faulty immune response to an inhaled substance. This theory is supported by evidence demonstrating that people who work and live in certain places appear to have an increased chance of developing sarcoidosis, such as people who spend a lot of time around dust, chemicals, and building materials (through their antigenic or adjuvant properties) are all at a slightly increased risk of contracting sarcoidosis.

Numerous studies, have observed a predilection for sarcoidosis to become clinically apparent in winter and early spring, peaking in spring months, and variations show higher peaks in winter . If it is assumed that the latency between exposure to the causative agent and development of sarcoidosis related symptoms is in the order of a few weeks to a few months, it seems likely that exposure may first occur in many cases in the late fall to early spring.

Wood smoke, such as using wood stoves or fireplaces for home heating may also be a risk factor for Sarcoidosis. The incidence increases in winter through early spring. More than one million Canadian families heat their homes at least partly with wood. Late fall to early spring is the peak time for wood burning, when home heating becomes a factor. Significant air quality problems occur in winter months due to nearby residential wood burning. We smell the smoke in our houses and it irritates the eyes and throat to go outside.

Past studies have also noted a clustering in parts of the country where there is more lumbering activity. In particular, a study by Dunner and Williams suggested that sarcoidosis cases occurred twice as often where lumbering and wood milling was a principal industry. The past sarcoidosis literature should be considered carefully for the possibility that the associations with forests, lumbering, wood milling and wood burning are surrogates for the sensitising antigens they harbour.

Arlene Berry arrived back in Red Lake, Ontario during the later part of the summer of 1998, where she had been living and working as a housekeeper for almost a year before. At some point she

claimed to have pulled a muscle in her back flipping a mattress while working at the Red Dog Inn and often complained of aching discomfort in her lower back. Her back pain would come and go as it did, which had been confined primarily to the tailbone area. While in Red Lake she had been seeing a Dr. Jinot of the Red Lake Medical Associates for a variety of ailments, obviously not considered by her doctor to be urgent enough to warrent serious medical attention.

By mid to late February of 1998 Arlene Berry had moved back to Kirkland Lake, Ontario, where she had been living previously. Her family MD, Dr. Edward Jordan began treating her assumptively for what he termed to be a "suspected bronchitis", in spite of enlargement of the distal segments of the fingers, what is known as digital clubbing. Finger clubbing is a thickening of the fingertips that gives them an abnormal rounded appearance.

Digital clubbing typically is a sign of underlying disease, usually of pulmonary or cardiovascular origin. Although clubbed fingers are mostly asymptomatic, it often reflects the presence of dreadful internal illness like lung cancer, pulmonary fibrosis, sarcoidosis, or underlying suppurative conditions, e.g. lung abscess.

On August 6th of 1999, Arlene. Berry attended the Kirkland and District Hospital Emergency Department where a chest x-ray was ordered by a Dr. Beeston. As the radiology deparmtent was closed that evening she was advised to return the following day. Her family MD, Dr. Edward Jordan was on duty in the ED when she returned and a chest x-ray was performed. The patient was started on antibiotics for a "suspected bronchitis" and sent home pending the radiology report.

When Dr. Jordon failed to get back to her with the results of her x-ray she sought him out at his office situated at the old hospital on Secont Street and was told he was probably working the ER at the hospital at that time. She returned to the ED in November of 1999, thinking perhaps that the result of her x-ray was negative, since she had not heard back from anyone. According to Jordan "I believe

that I saw Ms. Berry in the Emergency Room of the Kirkland and District Hospital at some point in November of 1999, although I do not have a copy of the record", infers that he conveniently misplaced or destroyed it. The excuse given by Dr. Jordan was that because the patient had not attended his office he "made no connection" with respect to the request for a follow-up of the radiology report.

The film had been transcribed on or about the 9th and returned to Dr.Jordan shortly thereafter. He claims he attempted to contact the patient "unsuccessfull, first by phone, then via mail" asking her to book an appointment. At that point, Arlene became frustrated and angry because, firstly, she had no phone of her own, although the hospital had been giver the telephone number of her foster brother as a contact, and secondly, she received no such written notification by mail, neither had her foster brother been contacted in any way, prompting her to publicly berate her physician, Dr. Jordan, in front of everyone in the ED. According to a hospital insider, she was probably "blacklisted" (alienated, estranged) from that point on, and also "labeled as a problem patient". Gone was the doctor patient relationship, gone was the compassion that had once made the Kirkland & District Hospital a place she could rely on.

Blacklisting is multiple providers denying care to a certain patient or putting a patient in harms way with a connotation of willfulness or willful blindness, or neglect. In northeastern Ontario, this is readily accomplished through a network of providers who share information about difficult, or difficult to diagnose or difficult to treat patients, with nothing more than a phone call or a nuance in a referral, negatively branding the patient. Launched in March 1998, NORTH Network had been the telehealth organization serving communities in Northern and Central Ontario. NORTH's head office was located in Toronto and its clinical headquarters in Timmins. NORTH was a program of Sunnybrook Health Sciences Centre.

A repeat chest film was obrained on or about the time Dr. Jordan claimes he had seen the patient in

November 1999, however, it took another doctor to read her x-ray chart, and to order more appropriate testing before anything was done. According to correspondence received by the CPSO, "the radiologist reported that there was an area of consolidation noted in the left lung base posteriorly with blunting of left costophrenic sulcus, suggestive of a bronchial obstructing lesion, such as a carcinoma left main stem bronchus". For the record, an area of consolidation is a general term referring to the accumulation of "any foreign substance" – consolidation of the lungs most commonly infers that an infection, or pneumonia, is present. A lesion is any damaged or abnormal area of tissue in or on the body. Lesion is a broad medical term that might refer to a wound, sore, ulcer, tumor, cyst or some other type of tissue damage.

The patient was admitted at that time and was referred to the general surgeon, Dr. Rumball. On December 13th of 1999. Dr. Rumball performed a bronchoscopy. A C.T. was obtained in Timmins on December 14th. of 1999, which reported a complete collapse of the left lung, possibly due to an endobronchial lesion. Arrangements were made for the patient to undergo a second bronchoscopy on December 17th, 1999. The procedure was performed by Dr. Claudio de la Rocha at the Timmins and District Hospital. On December 20th, the patient was again seen at the Timmins and District Hospital for a repeat bronchoscopy, mediastinoscopy and left anterior mediastinotomy.

Bronchoscopy is an examination of the air passages leading to the lungs. Most lung cancers are not visualized with the bronchoscope because they are located toward the edge of the lung, rather than in a major bronchus. A false positive test can occur in the presence of inflammation or infection.

Mediastinoscopy is a procedure that enables visualization of the contents of the mediastinum, usually for the purpose of obtaining a biopsy, although there is nothing on record locally to suggest that a biopsy was ever done. The mediastinum is the area in the middle of the chest between the lungs. Mediastinotomy is a procedure in which the doctor inserts a tube into the chest to view the organs in the mediastinum. According to Dr. Jordan, "carcinoma of the left main bronchus was diagnosed. Ms. Berry was staged as T4 NI with residual disease on the aorta".

On January 13th of 2000, Arlene Berry was admitted to the Timmins and District Hospital and a left pneumonectomy was performed under the care of Dr. Claudio Alberto De La Rocha, a Cardiovascular and Thoracic Surgeon.

Following surgery, Arlene Berry was discharged home 5 days later. An early hospital discharge can either suggest a cost driven premature discharge, or portend a low risk with a reasonably favorable prognosis. However, as health insurers look to cut the costs of patient care, one of the most frequent (and dangerous) tactics addopted by doctors and hospitals in Ontario is discharging patients too early from the hospital or other site of care.

On or about March 16th of 2000, Arlene Berry returned to Timmins where she underwent follow-up study and testing at the same hospital, consisting of a CT scan, and a mediastinoscopy as part of her post-operative evaluation. Mediastinoscopy is carried out in hospital, under general anaesthetic. If you are having a mediastinoscopy, a small cut will be made at the bottom of your neck, usually in the normal skin folds. A mediastinoscope is inserted through the incision. What the family had found to be peculiar following the mediastinal procedure, was a dramatic voice change, suggesting a partial vocal fold paralysis, or decreased vocal fremitus fixation, and believed to have been procedure related. Gone was her uniquely distinctive, naturall voice.

Possible complications linked to mediastinoscope include haematoma (a collection of blood, usually clotted), injury to the esophagus or voice box (larynx) with change in voice quality for some time, and infection at the site of the procedure. Although Arlene began to regain her voice in the weeks that followed, her voice remained somewhat whispery (speech volume was low) for the remainder of her days.

According to the Outpatient record at OP-54, the patient’s recent head CT scan showed "NO

METASTASIS", and her mediastinoscopy, a surgical procedure to examine the mediastinum inside of the upper chest between and in front of the lungs, were found to be "NEGATIVE". Metastasis is the process that involves the spread of a tumor or cancer to distant parts of the body from its original site. A normal result for a mediastinoscopy means no abnormal tissue, growths or signs of infection are present at the time. From that record it is clear that NO clinically detectable metastasis were found.

Following that testing, Arlene confided "I don't have AIDS, or brain tumors, or anything like that, but I might have a cyst, or infection", and elaborated briefly from what Dr. De La Rocha had told her about how "some people could be carriers and not even know it". It would have been an incidental finding, which was not present initially at admission, detected March 16th of 2000, with a timeline between January 13th and March 16th postoperative.

A condition is considered to be asymptomatic if it fails to show the noticeable symptoms with which it is usually associated. Iatrogenic (doctor caused) infection is influenced by factors like poor sanitation and hygiene, e.g., external inoculation with contaminated hands, surgical gloves, instruments (such as insertion of a bronchoscope into the lung through the mouth), etc. resulting from medical treatment or surgical procedures, and because No record of it is created in the first place, the surgeon passes the buck together with all the possible blame for whatever happens, and the patient doesn't get diagnosed or treated in a timely manner, or at all.

There are many conditions in which cysts develop, but in most cases, they are not cancerous. Occasionally, cysts can be related to cancer, but cancer is not associated with an assymptomatic carrier state because cancer is NOT contagious. The word cyst is derived from the Greek word meaning "bladder". A cyst is a suitcase for the material inside. Cysts can be congenital but are usually acquired. Some of them relatively harmless, and some of them trojan horses of the potentially lethal variety.

A Google search of the terms: "cyst, infection, asymptomatic carrier" with the quotation marks omitted is highly suggestive of a parasitic infection of protozoan origin and in particular, "amoebiasis" in which "CYSTS" are the infectious form found in asymptomatic carriers. Entamoeba histolytica is the protozoan parasite that causes amoebiasis. Amoebae spread by forming infective cysts. The clinical spectrum of amoebiasis is broad ranging from asymptomatic passage of cysts through fulminant colitis to localized abscesses of the liver, lung, brain, and other tissues. Systemic or extra-intestinal amebiasis may develop in 3 months or later after infection.

An asymptomatic carrier is one who harbors disease organisms in their body without manifest symptoms, hence, "a person could be a carrier and not even know it". Detected March 16th of 2000, with a timeline between January 13th and March 16th, or about 2 months plus postoperative, suggests an "iatrogenic" etiology. Iatrogenic (doctor caused) infection is influenced by factors like poor sanitation and hygiene, e.g., external inoculation with contaminated hands, surgical gloves, instruments, etc., during testing, or surgical procedures. Amoebiasis is a communicable disease that can spread from human to human.

There are two basic types of amebiasis: intestinal and extraintestinal disease, which may exist simultaneously. Amebiasis, however, does have some severe forms. These include amebic dysentery and amebic abscesses. The most common clinical symptoms of amebic infection are amebic colitis, which has various symptoms, and amebic liver abscess. It may result in low gastrointestinal bleeding and bowel obstruction. Carefully look for the presence of blood (hematemesis), preceded by bile (bilious vomiting). Notably, "hematemesis" is documented at A-26 of the record, and is preceded by bilious vomitig as evedenced by upwards of "100cc yellowish fluid" at 1915 hours, evidenced at N-6 of the record, at 0300 hours.

There are usually no symptoms of intestinal infection, but persons with amebic liver abscess do have symptoms, including: abdominal pain (particularly in the right, upper part of the abdomen; pain is

intense, continuous or stabbing), hepatomegaly (is present in some cases), intermittent fever and chills, diarrhea (in only one-third of patients), bloody diarrhea (is present in 7% of cases), general discomfort (uneasiness, or ill feeling/malaise) jaundice (rare), loss of appetite, night sweats (early in the course), jaundice (< 10% of cases, mostly occurs in complicated cases with multiple abscesses or a large abscess compressing the biliary tract), weight loss or weakness, nausea and vomiting are present in 32-85% of cases, pulmonary abnormalities are present in 20-45% of cases, bowel sounds (are present when the abscess ruptures in the peritoneal cavity). In 95% of cases, onset occurs within 5 months of contact. Notably, A-6 of the record documents the presence of "bowel sounds".

Severe forms of amoebiasis include colon ameboma, fulminant necrotizing colitis, and toxic megacolon (a severely enlarged bowel that can rupture). The appearance of symptoms, such as severe dysentery and pain with signs of tenesmus, low grade fever, tachycardia, hypertension, nausea, and anorexia are suggestive of severe forms of intestinal amoebiasis. Low grade fever with chills is suggestive of secondary bacterial infection, occurs in 20% cases. The initial presentation may be intermittent bloody stool with abdominal pain. Severe amoebiasis infections is known as invasive or fulminant amoebiasis. Invasion of the intestinal lining causes amoebic dysentery or amoebic colitis. If the parasite reaches the bloodstream it can spread through the body.

Amebiasis can be asymptomatic, or it can present as colitis or dysentery. Some patients develop amebic colitis, acute dysentery, or chronic diarrhea, and occasionally life-threatening liver or brain abscesses. If left untreated, a cyst can cause liver, lung, and brain abscesses. The diagnosis of amoebic brain abscess is considered when the patient develops symptoms of amoebic dysentery. A brain abscess can grow very quickly, typically becoming fully formed within about two weeks.

Brain abscesses and a mild meningitis associated with a free-living amoeba is reported in The Lancet. The symptoms resemble those of brain tumour. Increased intracranial pressure, stupor, and papilledema are also signs of brain abscess. Brain abscesses can cause brainstem herniation and

can rupture into the ventricular system. Without treatment, the abscess may break open (rupture) and spread into other organs, leading to death. People who are treated have a very high chance of a complete cure or only minor complications. The abscess may rupture into the abdominal cavity, the lining of the lungs, the lungs, or the sac around the heart. Rupture of an amoebic brain abscess can lead to shock and death.

Meningitis can produce mild symptoms, such as headache, low-grade fever and tiredness lasting two to three days in some patients. In other patients, the symptoms can be severe and begin suddenly with fever, headache and stiff neck accompanied by some combination of other symptoms: decreased appetite, nausea, vomiting, sensitivity to bright light, confusion and sleepiness. CBC is usually elevated in meningitis and there is initially a neutrophil predominance; polymorphonuclear leukocytes often predominated early in the course of infection.

Between 10% and 35% of patients, with liver amebiasis, experience gastrointestinal symptoms such as, nausea, constipation, vomiting and diarrhea. Patients with sub-acute liver abscesses experience vague abdominal discomfort, weight loss and anemia. Spread of the trophozoites via the blood stream may lead to abscesses forming in the brain. This is characterized by an abrupt start of symptoms including altered consciousness, nausea, vomiting and headache. Untreated, progression to death may be very rapid.

Cyst formation is triggered by dehydration in asymptomatic carriers. Parasites (and toxins) in the intestines or elsewhere in the body can sometimes enter the blood and be carried to the organs and tissues of the body, including the brain, and here's the kicker: Parasitic cysts of any origin may mimic primary or metastatic brain tumor(s). The pressure exerted by the growing cysts can cause paralysis or brain damage, or even blindness. Sometimes the cysts block the flow of cerebrospinal fluid within the spaces of the brain (ventricles) putting pressure on the brain. This disorder is called hydrocephalus. The increased pressure can cause headaches, nausea, vomiting, and sleepiness.

In Canada and the United States, amoebiasis is most often found in immigrants and in people who have traveled to or who have come into contact with people from developing countries or who live or work in institutions or hospitals that have poor sanitary conditions. Further, many immigrant doctors who come to Canada have very low qualifications, including poor hygiene practices. Many of them have been exposed to malaria, parasites and many unfamiliar infectious diseases, such as amebiasis, which pose health issues for all concerned. In Canada, amoebic infection is mostly encountered in small patches of population that have migrated from endemic areas. Notably, there have been between 2 and 11 cases of amebiasis in Simcoe Muskoka alone every year since 2000.

For the record, Dr. De La Rocha immigrated to Canada from Mexico, where he graduated from the National Autonomous University of Mexico. Mexico is a hotbed for amebiasis, a source of infectious cysts. Multiple abscesses are also frequent in Mexico where parasitic intestinal infections are multiple infections that constitute approximately 40% of analyzed individuals in which it is possible to detect more than one pathogen together with commensal parasites that are an indicator of fecalism.

Amoebic cysts are typically resting, "dormant" and/or resistant stages in the full life cycle. Some cysts can remain viable for up to 4 months or longer. The detected cyst would have been "dormant"" at the time it was discovered, in other words, a very serious infection waiting to happen. This was not cancer and De La Rocha not only identified finding a "cyst" with which he himself was familiar, he knowingly sought to pass it off as terminal cancer, knowing that the cyst, once unleashed, was going to be the death of this patient. He staged her as T4 NI with residual disease on the aorta.

Dr. Claudio Alberto de la Rocha is no stranger to controversy. In April 1993, Dr. de la Rocha was charged with failure to maintain the standard of practice and breaking the law by administering a noxious substance. He killed his patient usng a combination of potassium chloride as well as morphine. He was found guilty. He received a suspended sentence, three years probation and a six-

month suspension of his medical licence for his role in the October 1991 death of a 68-year-old lung cancer patient.

Following her mid March testing, Arlene Berry was then referred to the Northeastern Ontario Regional Cancer Centre situated at the Laurentian Site, Sudbury, Ontario for consideration of radiation therapy under the care of Dr. Hugh Prichard, a radiation oncologist. It would have been about the last week of March of 2000. While receiving treatment in Sudbury she stayed at the Daffodil Terrace Lodge, where, between appointments, she spent her leisure time fishing off the shores of Ramsey Lake. The treatment lasted about 5 weeks. During that time, she remained quite well and in fact remained well untill the last week or two of her life. By the end of April of 2000, Arlene Berry had completed her post-operative course of radiation therapy. In light of this treatment, her condition was seen to be stable. She was dischared home to Kirkland Lake on or about the last day of April, 2000.

Within two weeks following radiation therapy Arlene Berry developed "flu-like" symptoms, suggestive of gastrointestinal illness. It began as an upset stomach with nausea and night sweats, possibly accompanied by a slight fever. She thought she had the flu due to an associated achy feeling. She also felt she might be developing an ulcer, such as a peptic ulcer and so took the odd drink of buttermilk, which wasn't very well tolerated due to nausea. She lost her apetite and developed a serious aversion to food. Then began the abdominal pain and bouts of diahrea with alternating constipation progressing to bloody stools, including at least one abnormally large, hard, painful bowel movement (suggestive of megacolon) that were accompanied by headache, vomiting and general feeling of malaise. Notably, gastroenteritis, gastritis, or peptic ulcers can cause internal bleeding.

For the record, people exposed to amoeba may have more than just flu-like symptoms. Parasitic invasion is often mistaken for vague digestive problems, including flu-like symptoms, colitis, stomach

aches, nausea, unexplained vomiting, etc. The appearance of symptoms, such as severe dysentery and pain with signs of tenesmus, low grade fever, tachycardia, hypertension, nausea, and anorexia are suggestive of severe forms of intestinal amoebiasis. In fact, all of these symptoms are common findings in amoebic infection. The parasite can cause ulcers to form in the intestine resulting in amoebic dysentery.

Further, there are two basic types of amebiasis: intestinal and extraintestinal disease, which may exist simultaneously. Extra-intestinal amoebiasis is the result of dissemination in the bloodstream which produces liver abscesses, and occasionally abscesses of the lung and brain. When detected in time, liver abscess is usually treatable and often can be cured with a course of antibiotics or a combination of antibiotics and a surgical procedure to drain the abscess. Left untreated, however, a liver abscess can burst and spread the infection, leading to sepsis, a life-threatening blood infection.

Amoebiasis is both infectious and transmissible by direct or indirect contact and sooner or later follows the fulminant progress of a potentially lethal opportunistic infection requiring emergent management that, unless diagnosed and treated in a timely manner, can kill within hours or days once symptoms appear. Systemic manifestations such as nausea, headache, low grade fever and anorexia are often present. A fever and chills may occur at first but then disappear as the body fights off the infection. In the early sub acute phase symptoms like anorexia, nausea, and night sweats may dominate. Very quickly, in from one to 14 days (two weeks), the symptoms worsen. A very severe infection like this can be fatal within 24 hours.

Over the last week of her life, Arlene Berry noticed increasing weakness of her legs. She tended to become easily irritated and somewhat confused. She developed muscle weakness, difficulty in walking (unsteady gait/ataxia, i.e. pulling to the right), facial weakness marked by a crooked smile, slurred speech, and drowsiness progressing to extreme fatigue. Futher, weakness of facial muscles produces a characteristic haggard appearance, or a deceptively disinterested facial expression. The

characteristic appearance of a haggard or mournful face and drooping eyelids is caused by facial muscle paralysis. A sagging mouth or a crooked smile is a part of the same problem. When bilateral facial nerve palsies develop in young adults, sarcoidosis is usually the most likely cause. However, facial nerve palsy also has long been considered to have an infectious etiology.

Further, slurred speech is the hallmark of "dysarthria". Speech that is characteristically slurred, slow, and difficult to produce (difficult to understand) are the characteristic features of dysarthria. The person with dysarthria may also have problems controlling the pitch, loudness, rhythm, and voice qualities of their speech. For the record, encephalopathy has been reported in association with cerebellar toxicity characterized by ataxia, dizziness, and dysarthria. All of these signs marked by difficulty ambulating, dizziness and a slurred speech form a part of this record.

An important job of the liver is to change toxic substances that are either made by the body or taken into the body (such as medicines) and make them harmless. However, when the liver is damaged, these "poisons" may build up in the bloodstream. Hepatic encephalopathy may be triggered by: dehydration; electrolyte abnormalities (especially a decrease in potassium) from vomiting; bleeding from the intestines, stomach, or esophagus; infections; low oxygen levels in the body; and use of medications that suppress the central nervous system.

The outpatient record seen at OP-53 documents a 4 day history of bloody bowel movements (bloody stool) when voiding, evidenced by "bloody BM’s x 4 days", can suggest a parasitic etiology. The same record documents that she was "pale-looking and lethargic", and also that she had been taking Tylenol and Aspirin, noting "daughter states takes a lot", suggests a history of over-the-counter medications that can break the gastric barrier and damage the gastric mucosa (lining of the stomach). Aspirin and non-steroidal anti-inflammatory drugs are known to be harmful to the gastric mucosa and are associated with an increased incidence, prevalence and complication rate for peptic ulcers. When NSAIDs irritate the gastric mucosa, they weaken the resistance to acid, causing

gastritis, ulcers, bleeding, or perforation. Adverse reactions to NSAIDs such as drowsiness, unsteady gait, slurred speech, and confusion may also be noted.

The record at OP-53 dated May 22nd of 2000 documents a recent history of urinary-(tract infection, evidenced by "Here 1 week ago for UTI. Last period on 6th of May", followed by a more recent history of "hematuria" (blood in urine) for "three days", seen at OP-54. The healthcare provider who saw her made the diagnosis of UTI. The same record documents a prescription for Cipro, for treatment of urinary-tract infection. The belated test result evidenced at OP-55, later returned a finding of "NO Growth". Routine bacterial cultures are usually NEGATIVE for pathogens, showing No Growth, however, a negative urine test can also suggest the presence of unusual bacteria, viruses, or parasites causing symptoms of UTI.

The record at OP-54 dated May 22nd of 2000 documents "large blood trace leukocytes", what are an unusually high number of white blood cells. The presence of white blood cells in the urine usually signifies a UTI; hematuria may also be noted. The presence of leukocytes in urine is referred to as pyuria (pus in the urine). Urinary tract infections due to leukocytes in urine are more common in women and the conditions can vary from cystitis (an inflammation of the urinary bladder) to severe infections of the kidneys or bladder.

Although UTIs are almost always caused by bacteria, some viruses, fungi, and parasites can infect the urinary tract as well. Parasites can also live in the vaginal area where they can cause yeast and keep it there. They can cause itching and inflammation by directly attacking the mucus lining of the vaginal area. Parasitic infection symptoms that lead to recurring yeast infections include abdominal distress. Not all fungi are yeasts. There are both parasitic fungi and parasitic yeast, based on the nature of their behavior in the body. Although rare, parasites can and do cause urinary tract infections. It is very unlikely that a GP will ever tell you that the cause of your illness is related to parasites.

Onset of menstrual period, as evidenced at OP-53, is closely related (within time frame) to illness in which symptoms of UTI result from production of toxin which may then wash backwards up through the vagina, uterus and fallopian tubes (or similar mechanism associated with toileting) to be absorbed from the colon. There can be many complications of urinary tract infections, including dehydration, sepsis, kidney failure, and death. You are more likely to develop a urinary tract infection when you are dehydrated. Dehydration can also cause the symptoms of the urinary tract infection to appear more severe.

Dehydration is the main concern with most vomiting. If the symptoms continue for days, they are usually considered severe. Further, a prolonged bout of diarrhea or vomiting can cause the body to lose more fluid than it can take in, resulting in dangerous dehydration. Loss of appetite and fatigue are early signs of dehydration. Dehydration is also the most common general complication of intestinal parasite infections. Symptoms of dehydration include headaches, dry skin, palor, lethargy, mood changes and slow responses, including dark-coloured urine, weakness, and tiredness. Dehydration occurs when water loss in the body exceeds the water intake. Most people can tolerate a three to four percent decrease in body water without difficulty. A five to eight percent decrease can cause fatigue and dizziness.

Dehydration, or not getting enough fluid, causes low blood pressure, weakness, dizziness, fatigue, and nausea.

OP-53 documents "For 2 weeks had flu, migraines", while A-5 of the record documents the presenting complaint as "headaches", accompanied by "severe stomach pain", and "abdominal pain ongoing for 2 weeks". Migraines can be associated with certain gastrointestinal problems. For the record, abdominal or stomach pain concurrent with nausea and vomiting points to the "abdomen" as the source of the problem. NO diagnosis or differential was made following the patient's admission at

that time, or at all, according to the record. Certainly, NO protocols were followed. Clearly, from the record as a whole, this patient was deliberately made to deteriorate w/o a diagnosis of her stomach pain.

Arlene Berry was still neurologically responsive when I saw her following her admission. She was able to reach and use for herself the kidney basin at her bedside table, as she occasioned to vomit more of the same flu-like "yellowish liquid" that she had done so many times on the days before, and in fact used it for herself in the presence of her family, at which time a cool cloth was provided by the nurses, evidenced by the record "family in", seen at N-6. The same record documents upwards of "100cc yellowish fluid", what I take to be frank bile, or "bilious vomit", (toxic chemicals removed from the blood by the liver flow out of the body via the bile and digestive tract). The time of that assessment was documented at 1915 hours on May 23, 2000.

The word "bilious" comes from the word cholera. The word cholera is Latin for bilious disease and has come to indicate a severe intestinal infection. The clinical difference between bilious and non-bilious vomiting (ie, vomiting yellow or green) is critical in distinguishing life threatening abnormalities. Acute symptoms include bilious vomiting, diffuse abdominal pain, and bloody stool. Although stomach flu is by far the most common cause, intestinal obstruction is also the most serious and is considered a surgical emergency, and treating the patient at the earliest is a must to avert any complications.

When a person is vomiting bile, it is pointing towards the fact that the intestine is blocked. Intestinal obstruction is typically marked by severe abdominal pain. Unlike, other inflammatory bowel diseases, where the pain is tolerable, in this case the discomfort is torturing that may subside intermittently. Abdominal pain is typically accompanied by frequent bouts of bilious vomiting. Most importantly, the person feels constipated and there is absence of bowel movement. Intestinal obstruction, especially of the proximal small bowel, produces marked nausea and vomiting of bilious material. Distention

may be lacking, but intermittent cramping abdominal pain is characteristic. People with bowel obstructions may repeatedly vomit yellow, or green colored bile and a history of frequent bilious vomiting in the presence of abdominal pain should have been a "red flag" suggesting intestinal obstruction, which should have been treated emergently.

Nausea and vomiting are common features of many GI infections. A headache that is present with an intestinal infection may also indicate signs of dehydration, which also should have been a 'red flag' suggesting the possibility of intestinal obstruction that represented a potential emergency. Transitions from one type of intestinal syndrome to another can occur and intestinal infections can give rise to extraintestinal infections. Emergency physicians have a primary obligation to treat emergency situations such as bowel obstruction. In this case it wasn't even considered.

Bilious emesis is always "abnormal" and indicates ileus or "obstruction" distal to the common bile duct. The same record documents that the patient had stated she was "very tired", whereupon she was assisted to bed, as evidenced at N-6. She also complained of being "cold" , she had the chills, and so the nurses provided her with extra blankets. Periods of feeling cold often occur during common illnesses, but in fact the chills can often be a sign of infection that has spread throughout the body. For the record, fatigue and tiredness are prominent feature of amoebic infection. Physical examination generally reveals little that is unusual. Skin sensitivity is also common: rashes, eczema-like conditions, and even serious eruptions.

Trauma from an infected surgical wound, ie., wound botulism, can also trigger shivering. Surgical site infections and disruption of sutured tissue are frequent complications following surgery. At some point, the patient would have had her surgical stitches inspected, removed, and bandages re-dressed. Neurological signs and symptoms of both food-borne and wound botulism are "identical", although the wound botulism ones may take longer to appear. Your sensory awareness (your awareness of the world around you) will be unaffected. There are also no symptoms of fever, such as a high temperature, during a botulism infection.

OP-54 also documents a "haggard appearance", usually the result of a long, harrowing or emotional ordeal. The bald truth is that this patient appeared more anemic and dysthymic, thin and undernourished than anything else.

According to the hospital record seen at A-6, Arlene Berry was admitted to the Kirkland and District Hospital on May 23rd of 2000 by Dr. Spiller for "IV fluid and Gravol". According to the record at A-12, Arlene Berry was given more than just IV and Gravol. If not Dr. Jordan, then who ordered the 30 mg MS Contin (morphine sulfate) on his watch? From that record it seems clear that either Dr. Spiller lied, or he was totally oblivious to the 30 mg administration of Morphine Sulfate, evidenced at A-13.

According to the same record, she was admitted for "vomiting", not a diagnosis, but rather a symptom of many causes. The same records documents a "soft, non-tender" abdomen, and "no masses", suggests a typical admitting physical note to express an overall, normal, negative abdomen. Although a normal abdomen may be soft and non-tender, a negative finding can also suggest hypotonia, a disorder that causes low muscle tone that results in muscle weakness. Hypotonia is often the presenting sign for many systemic diseases and diseases of the nervous system. The abdominal muscles feel "soft and doughy", also a sign of gastropareses in clinical diabetes, which also can rapidly progress to intestinal obstruction. Hypotonia in Guillain-Barre syndrome is common and can be observed with significant weakness. It is characterized by diminished resistance of the abdominal muscles, with diminished tone of the skeletal muscles. Delayed diagnosis invites tragic consequences.

On examination, the physician who saw her documented positive "bowel sounds", evidenced at A-6. Hyperactive bowel sounds provide the most immediate indication of persistent upper GI bleeding/GI hemorrhage. An accompanying crampy abdominal pain can also suggest acute bleeding. Fatigue, shortness of breath, lethargy and pallor may also be noted. Gastrointestinal bleeding ALWAYS requires prompt physician evaluation.

According to the medical record at N-6 Arlene Berry was admitted to the Kirkland and District Hospital at 18:45 hours and had spent 75 minutes in the ER. In all that time, the ED physician, Dr. Spiller, had obviously done very little, if anything at all, as evidenced by the record, seen at A-3.

What also appears to be a referral at A-6, a chart-copy from the admitting physician (Dr. Spiller), directed to the attention of the family physician (Dr. Jordan), suggests a failure or reluctance on the part of the ED physician to adequately diagnose, as evidenced by his perfunctory, careless, indifferent examination seen at A-11. He missed the obvious signs of comorbidity (existence of more than one disorder at the same time) because he declined to look past the symptom of "vomiting", and so attributed the vomiting, to lung CA without further assessment. Diagnosis involves detailed assessment and evaluation of a thourough, detailed and complete medical history of the person. Clearly, that was not done.

Not diagnosing a condition is one of the most common forms of medical negligence. Another is when they "dismiss" the presenting symptoms as temporary, minor, or otherwise not worthy of treatment. This situation may result in an exacerbation of the underlying condition or injury, causing further harm, or even death. Clearly, the etiology of the nausea and vomiting had never been established, apart from Dr. Spiller”s "a question has arisen" with respect to lung CA, as evidenced at A-3. Premature closure is the failure to consider other plausible diagnoses after an initial working diagnosis is reached. It is one of the most common clinical reasoning errors made by clinicians. For the record, a question has arisen with respect to the sobriety of the ED physician at the time of his assessment of this patient (?).

At the time of her admission to the Kirkland and District Hospital, Arlene Berry’s blood pressure was documented at "115/70 bpm, with a pulse of 79 and regular", as evidenced at A-6. Normal blood pressure is defined as a systolic (top) pressure of less than 120 mmHg, and a diastolic (bottom)

pressure of less than 80 mmHg The same record documents "mild diffuse weakness" and "difficulty ambulating", including a respiratory rate of 18, on admission. The normal adult respiration rate is 12 to 18 breaths per minute. At the time of this assessment (18:45 HOURS), Arlene Berry was found to be "alert and oriented", with "NO Focal deficits".

The word "diffuse" means widespread and refers to symptoms that are not localized to just one or a few areas. Instead, it is more or less all over, or at least in many areas. There are numerous etiologies for diffuse weakness, including infectious, metabolic, autoimmune, endocrine, and toxicologic causes. Diffuse weakness may result from polyneuropathy, myopathy, neuromuscular junction disease, or systemic fatigue. The majority of peripheral neuropathies cause mainly muscle weakness. Muscle weakness is one of the first symptoms of peripheral neuropathy and is maximized soon after the beginning of a disease or about three to four weeks after onset, such as seen in Guillain Barre syndrome (GBS). In the Miller Fisher variant of GBS, the most striking findings on examination are "diffuse weakness" with widespread loss of reflexes.

Miller Fisher syndrome involves cranial nerves, which extend from the brain to various areas of the head and neck. Miller Fisher syndrome is characterized by three features: weakness or paralysis of the muscles that move the eyes (ophthalmoplegia), problems with balance and coordination (ataxia), and areflexia. People with this condition can have other signs and symptoms common in Guillain-Barré syndrome.

A focal deficit is a specific area in which normal function isn't present. Signs of a neurological disorder include a "slurred speech", and "difficulty ambulating". These are examples of SIGNS that constitute "focal deficits". According to Dr. Spiller, there were "no focal deficits". A reasonable physician ought to know what constitutes a focal deficit, especially when documented on the face of a patient's medical record, unless perhaps he himself presented with the exact same deficits, while in the course of performing his duty?

A-23 documents a "slurred speech" as evidenced by a ✔ in the upper left corner of that document, suggestive of systemic toxicity, ie., the toxic effects of a life-threatening bowel disorder. Slurred speech can be caused by disease or damage affecting the muscle and nerves of the vocal cords, The symptoms of a brain abscess, for example, include slurred speech. In the majority of cases signs and symptoms continue for no more than two weeks before the patient is hospitalized, as in this case.

Cerebral amoebic abscess caused by Entamoeba histolytica infection, is a rare global disease, not related to immunodeficiency that causes proctocolitis with bloody dysentery, and liver abscesses and although rare, cerebral abscess through haematogenous spread has been well documented. Symptoms of cerebral abscess result from increased intracranial pressure and mass effect. Headache, nausea, vomiting, lethargy, personality changes, papilledema, and focal neurologic deficits develop over days to weeks. Fever, chills, and leukocytosis may develop before the infection is encapsulated, but they may be absent at presentation or subside over time.

The Health Management Record seen at A-22 documents the patient's sensory COGNITIVE PERCEPTUAL PATTERN as "sedated", evidenced by ✔ in the lower left corner. Sedation is the depression of a patient's awareness to the environment and reduction of his/her responsiveness to external stimulation. For the record, electrolyte derangements can mimic sedative intoxication. Sedation can cause both hypertension and hypotension. Oversedation results in obtundation, characterized primarily by reduced alertness and hypersomnia. Hypersomnia is defined as a state of sleep in excess of 25% of the expected normal.

According to Dr. Jordan, "she had presented to the ED several days before with vomiting and it was thought that she had a UTI", to rule out delay in seeking treatment. According to the hospital record at A-8 "she was given antibiotics and sent home". This is a common ploy used by doctors and

hospitals when it comes to to evading emergent complaints. The bald truth is that the Victorian Order of Nurses (VON) had to be contacted in order to get her admitted to hospital in the face of life threatening indicators. VON is Canada's largest, national, not-for-profit, charitable home and community care organization.

According to the record at A-6, Arlene Berr returned on May 23rd to the emergency department "with the very same complaints". Know that rapid evolution of illness and patient return within 24-48 hours suggests a severe illness. The healthcare provider who saw her noted that she had been "taking morphine"| for pain management, and also that she had recently "stopped" taking the morphine, evidenced by "stopped this week", noting her recent medical history and that for "2 weeks" she had the "flu". Further, the record at A-5 clearly documents "not taking any pills now".

Sometimes people mistake symptoms of stomach flu, or gastroenteritis, for the viral infection we commonly call the "flu." But the "stomach flu" is not the flu. It is a gastrointestinal illness caused by a number of factors including bacteria, viruses and parasites. Severe cases can easily result in life threatening dehydration. On the other hand, flu-like symptoms are a very common manifestation of immunopathology, ie., an immune system reaction, such as seen in sarcoidosis, including Guillain Barre syndrome (GBS). Many patients with sarcoidosis as well as GBS have a "flu-like" syndrome. For the record, Guillain Barre syndrome presenting with sarcoidosis is reported in the PubMed. Flu-like illness is also a common complication of radiation therapy which results from radiation injury of the CNS. Untoward symptoms, including low-grade fever, flu-like symptoms, headache, dizziness, nausea, vomiting are all high on the order of amoebic infection.

GBS is a catastrophic condition which can be triggered by something as innocuous as a cold or stomach upset. Even the flu jab can cause it. Triggers of GBS and its variants include known and unknown infectious agents, certain medicines, including vaccines, and autoimmune reactions. Often, it is after an infection of the lungs, or stomach and intestine. Another is that a particular medication,

substance or combination may be the real cause of these disorders, but are often misdiagnosed, or overlooked as a cause. GBS is also caused by an immune system response to a trigger, such as 'drug-drug' interaction, with possible additive effects when used in combination or concurrently as part of a drug regimen, ie., narcotics in combination with phenothiazine additives.

Phenothiazine compounds such as Stemetil should never be used in patients receiving large doses of hypnotics (opioids), due to the possibility of "potentiation". Notably, Stemetil 10mg was added to the IV at 2030 hours. The drug is sedating and a potent vasodilator, which also crosses the blood-brain barrier. Patients are usually "volume expanded" prior to its use, resulting in neurologic derangement. We already know that the patient was given 30 mg po bid MS Contin by Nurse McCrank at 2000 hours, only one half hour prior to Stemetil (a brand name for prochlorperazine).

Sudden opiate withdrawal (quitting cold turkey) leads to a syndrome called "opiate withdrawal syndrome". Opiate withdrawal (as opposed to a proper taper) refers to the wide range of symptoms that occur after stopping or dramatically reducing opiate drugs after prolonged use (several weeks or more). In addition, the symptoms include agitation, anxiety, depression, muscle aches, pupillary dilatation, abdominal cramping, and diarrhea. Complications include nausea, vomiting and breathing in stomach contents into the lungs. This is called aspiration, and can cause serious infection. Vomiting and diarrhea can cause dehydration and body chemical and mineral (electrolyte) disturbances. Opioid withdrawal syndrome may also resemble a severe flu-like illness; symptoms peak at 72 to 96 hours but last for 14 days or more. Opiate withdrawal can also result in death for unhealthy patients.

Further, constipation as an adverse effect of opioid use is almost universal: "In one study, 95% of patients interviewed by nurses in a hospital oncology unit reported "constipation as the major side effect of their opioid pain-control regimen".

According to the record at A-13, Arlene Berry was given 30 mg po bid MS Contin by Nurse McCrank at 2000 hours on May 23rd, the evening before her death, in the face of an undiagnosed and undifferentiated condition(s) associated with "severe abdominal pain". MS Contin is a brand name for Morphine Sulfate. "Contin" is a pharmaceutical industry buzzword for "continuous" release. MS Contin has widespread effects in the central nervous system and on smooth muscle and produces respiratory depression by direct action on brainstem respiratory centers. Clearly, from the record as a whole, this patient was deliberately made to deteriorate w/o a diagnosis of her stomach pain. The administration of morphine can only serve to "obscure" the diagnosis or clinical course in patients with acute abdominal conditions, and may in this instance infer criminal liabiliy.

The biggest complication is return to opiate drug use. Most opiate overdose deaths occur in people who have just withdrawn or detoxed. Because withdrawal reduces your tolerance to the drug, those who have just gone through withdrawal can overdose on a much smaller dose than they used to take. In this case, Arlene Berry was given 30 mg morphine sulfate after several days withdrawal from a 10 mg regimen of Statex. According to family, the reason that Arlene had "stopped" taking the morphine was due to increasing severity of "constipation", requiring extra laxative and tap-water enemas to assist with stool evacuation, and also due to dizziness, marked by a sense of uneasiness progressing to unsteadiness. Resulting decreases in GI motility from antidiarrheal medications may also contribute to constipation and bowel obstruction. A question of GI motility?

Notably, morphine is also "contraindicated to sedation, including increased pressure in the head or spinal cord, possible abdominal problems requiring emergent treatment or surgery, and in patients having a substantially decreased respiratory reserve". Patients with only one lung have a decreased respiratory reserve due to a "diminished lung capacity". Futhermore, narcotic analgesic drugs can produce, via inhibition of peristalsis, fluid retention great enough to "**mask** depletion of extracellular fluid and electrolytes".

CAVEAT: MS Contin overdosage may result in apnea, circulatory collapse, cardiac arrest, and breathing problems that can lead to death.

A possible cause-and-effect relationship has been posited between granuloma formation and morphine sulfate. Inflammatory masses including granulomas (some of which have resulted in serious neurologic impairment including paralysis) have been reported to occur in patients receiving continuous infusion of opioid analgesics, such as morphine sulfate. Damage caused by granulomas can include sensory loss, impaired bowel and bladder function, and permanent paralysis.

The term "granuloma" is a medical term that is used to refer to a minute collection of immune cells that are known as macrophages. It is a mass of inflamed granulation tissue from injury or infection, usually associated with ulcerated infections, or invasion by a foreign body. A granuloma is a ball of immune cells associated with various disease states including sarcoidosis, Crohn's, and tuberculosis, hence, the term "granulomatous diseases". Granulomas are caused by an extremely broad range of disease processes. Granulomas are also the pathologic hallmark of sarcoidosis. The granulomas of sarcoidosis are similar to the granulomas of tuberculosis and other infectious granulomatous diseases. Other important causes of granulomas are parasitic infections. Granulomas may also form around parasites.

Also, know that granulomatous conditions of diverse etiologies share common histologic features. Further, the coexistence of sarcoidosis and "opportunistic infection" has previously been documented. Granuloma may accompany a parasitic lung infection known as pleuropulmonary amoebiasis. The term "granulomatous" is also indicative of hemorrhagic necrotizing lesions or brain abscess with severe meningeal irritation and encephalitis. Entamoeba histolytica and Toxoplasma gondii are two of the commonest protozoa causing abscess in the brain.

In this case, while analytic reasoning may suggest sarcoidosis, pattern recognition suggests

amoebiasis, with a mixed picture. The posibily that cancer may never have existed in the first place cannot be ruled out. When found during an x-ray examination, the granuloma of sarcoidosis is often "mistaken for cancer". These granulomatous disorders are often reported as "mimicking" one another. Both of these diseases can cause a mass of granulomas in the brain or meninges, which are the membranes that cover the brain. They also can affect one or more nerves anywhere in the body. Most often, they can also affect the nerves of the face causing one side of the face to droop. Symptoms of disease in the nervous system may vary only slightly for each, or not at all. An infectious etiology of sarcoidosis has long been suspected. Aboebiasis is high on the order of having mixed infections. Some diseases are caused by the infection of more than one type of organism simultaneously. Notably, both sarcoidosis and amoebiasis have been reported as being "mimickers of multiple pulmonary metastasis".

The record at N-6 documents "telephone orders" received by the hospital from Dr. Jordan at 2030 hours for "Stemetil 10mg" by IV, x 4 daily "for control of nausea", given by the RN, as further evidenced by the physician's orders seen at A-11 of the record. Stemetil is a brand name for "prochlorperazine", a phenothiazine drug. Most drugs in this category are used as anti-psychotics (neuroleptics). Neuroleptic means "nerve seizing", and describes the paralyzing effect these drugs have on the brain and nervous system. Under normal circumstances, a typical single dose of Stemetil for a small woman with low body weight is 5mg.

It is also clear that Dr. Jordan sought to eliminate the symptom "nausea", without his attendance, as evidenced by the phone order "for control of nausea" and without addressing any possible underlying causes.

Stemetil suppresses activity in the trigger zones of the vomiting center by "paralyzing the gastrointestinal tract" which governs the vomiting reflex, which can also exacerbate dismotility. Further, the antiemetic action of Stemetil may "mask the signs and symptoms of drug overdosage

from other drugs and may obscure the diagnosis and treatment of other conditions". Increased sedation is also a serious side effect of this type of agent. Stemetil poisoning is marked by "oversedation, respiratory depression and hypotension".

Stemetil is widely distributed into body tissues and fluids. It undergoes metabolism in the gastric mucosa and on first pass through the liver where it enters the enterohepatic circulation and is excreted chiefly in the feces via the biliary tract. The drug falls in the same class of phenothiazines that have been known to suppress intestinal motility to the point of producing a paralytic ileus. Stemetil can also lead to changes in the blood-brain barrier (BBB), allowing an infectious agent to gain entry to the brain and produce lethal CNS (central nervous system = brain and spinal cord) infection.

The CNS includes the spinal cord and brain while the peripheral nervous system includes those nerves that extend into the body and are not protected by bone. Diseases of the CNS and PNS are caused by many different types of pathogens, some of these are represented by: bacteria, viruses, fungi, toxins and amoebae. Diseases of the nervous system include meningitis or encephalitis. Meningitis is the inflammation of the meninges (tissue covering the brain and spinal cord) and under certain situations lead to killing areas of the tissue. Encephalitis has symptoms similar to meningitis but includes the infection of the brain. It is also interesting to note that "amebic cerebral abscess mimicking bacterial meningitis" is reported in Springer Link.

xx

The co-administration of a narcotic analgesic (Morphine Sulfate) and a neuroleptic agent (Stemetil) will result in "neurolept-analgesia" with "drug-induced" reduction of oxygen intake, resulting in respiratory depression, which represents the principal negative variable introduced with conscious sedation that left unrecognized and untreated, is the cause of panic, including most serious

complications. The combination of these drugs is to physically "paralyze" the body, rendering the individual less able to react or to move. They produce a chemical lobotomy and a "chemical straitjacket". Their main impact is to blunt and subdue the individual.

Neurolept-analgesia is defined as a state of CNS depression, which means it can slow down the brain and cause problems such as decreased breathing, or loss of consciousness. Central nervous system (CNS) depression refers to physiological depression of the central nervous system that can result in decreased rate of breathing, decreased heart rate, and deep physiologic depression that "resembles and can mimic brain death". Although opiates usually cause constriction of the pupils of the eyes, for the record, "prolonged depressed breathing may result in extremely low blood pressure and dilated (enlarged) pupils".

The patient was sedated into a wide awake pseudocoma akin to "terminal sedation" where she became unresponsive. She was intentionally denied nutrition and liquids orally because to do so would cause aspiration of the materials into the lungs. Since this type of sedation (neurolept-analgesia) keeps the patient subdued, the fluid volume of the blood decreases to the point where the circulatory system fails, or cannot pump because there is not enough to maintain blood pressure. It is common knowledge that stealth doctors hasten death by withholding life support or using high doses of morphine in cancer patients. A combination of other drugs may also be used to obscure the clinical course. The intended outcome is the same: to hasten or bring an end to the patient's life in a way that sidesteps medical ethics, including the law itself. For the record, spontaneous breathing is evidence of responsiveness.

Terminal sedation is the practice of inducing a pseudocoma usually by means of a continuous intravenous infusion of morphine. The morphine drip becomes a STEALTH CODE for "slow euthanasia". Terminal sedation was NEVER intended to be used as a pain control method. It was mostly used in the case of agitated patients, and to a lesser extent, for patients whose pain could not

be managed well otherwise. Weither by means of deep sedation (induced coma) rendering the patient unconscious, or by making the patient appear to be unconsciousness (pseudocoma) the patient cannot make his/her own decision to terminate life. Using these types of stealth techniques, doctors and hospitals actively kill many patients without the patients' knowledge or consent.

A-15 documents the 24 hour IV fluid balance record, that between 1745 hours and 0200 hours was administered as follows: A-14 documents an "IV gid prn", meaning that fluid and medication rate of administration to be is given by IV as follows: 3.3 % dextrose (a sugar solution used in intravenous drips) and 0.3 % sodium chloride solution (referred to herein as "2/3 and 1/3") @ the rate of 100 cc/hr, together with the medications, as evidenced at A-15.

Further, it is never appropriate to use the combination of 3.3% dextrose and 0.3% sodium chloride (known as 2/3 and 1/3) as "initial fluid resuscitation" in the dehydrated patient. Its use is limited to those who have severe hyponatremia unresponsive to isotonic saline boluses or who need concomitant fluid restriction. To restrict fluid resuscitation in an already dehydrated patient is outright stupidity, and constitutes an act of wanton and reckless disregard for human life.

Dehydration – Without sufficient levels of water in the brain, ions get disrupted and the result is brain damage.

The combination of 3.3% dextrose and 0.3% sodium chloride (known as 2/3 and 1/3) contains only 51 mmol/L of sodium. Outside of the body, the osmolarity of the solution is 269 mOsmol/L (sodium and dextrose combined). Once the solution is infused, however, the dextrose is rapidly metabolized, which leaves two-thirds of the solution (667mL) as electrolyte-free water and renders the solution extremely hypotonic. The patient will suffer a decrease in the osmotic concentration of the plasma which is now hypoosmolar to red blood cells and so water enters freely by osmosis and the cells swell and eventually burst, resulting in lysis of many red blood cells and the inability to oxygenate the

brain, etc. For example, red blood cell placed in a hypotonic solution (ie, pure water) bursts immediately (hemolysis) from the influx of water. Other conditions that can cause hemolysis include immune reactions, toxins and poisons.

In addition, if there is reason to be concerned about impaired function of the brain, heart, or kidneys, it is always prudent to rehydrate more slowly. This empirically-derived approach minimizes the cerebral disturbances (e.g., seizures, cerebral edema) caused by fluid shifts that can occur if fluid is infused too rapidly. It needs to be clear that all guidelines regarding fluids and electrolytes are approximations that in no way can replace careful monitoring of the patient.

The record at N-6 documents "IV infusing well", suggestive of overzealous IV infusion. Be aware that rapid administration of hypotonic IV fluids can cause swelling of the brain cells and increased intracranial pressure. Circulatory overload can occur if IV is not regulated properly and IV fluids infuse too rapidly for the patient's body to handle. Too much water in too short of time period will actually flood your nervous system and kill off brain cells. Signs of fluid overload include "tachycardia, elevated blood pressure, dyspnea and other signs of respiratory distress".

Seriously ill patients always require accurate fluid balance monitoring because IV fluid also contains the medication(s). Rapid infusion may also lead to over-dosage. There are no further IV related entries on the record, either to indicate when or if the IV was discontinued, or to show that the rate of administration was being accurately monitored, or modified, suggestive of deliberate omission, or iatrogenic neglect.

Correction of serum sodium that is too rapid can precipitate severe neurologic complications as a result of intracerebral osmotic fluid shifts and brain edema. This neurologic symptom complex can lead to tentorial herniation with subsequent brain stem compression and respiratory arrest, resulting in death in the most severe cases. The primary cause of morbidity and death is brainstem herniation and mechanical compression of vital midbrain structures.

N-9 of the nurses' notes documents a PRECAUTION for a "resistant bacteria, evidenced by a ✓ in the upper right hand corner of that document, under the subheading for "INFECTION CONTROL PRECAUTIONS". The same precaution is also noted in the upper right hand corner of the record seen at A-21. There are no further details, suggestive of deliberate omission.

What I take to be the physician's diagnostic chart evidenced at A-3 of the record, is a "total blank", like the primary physician whose name appears on it. From that record it seems clear that nothing was entered because nothing was done. A useful maxim to remember is "Not documented means not done".

For the record, Dr. Jordan treated this patient over the telephone, "unseen", while sitting at home watching TV.

The record seen at N-6 documents the discovery by duty nurses at 0030 hours of the patient's "head against the left side bed rail with her feet under the right side rail", consistent with unconscious proprioception, or loss of sense of one's own perception of the relative position of neighboring parts of the body to each other, which is occasionally impaired spontaneously, especially with extreme fatigue. The same record documents "No response to verbal or physical stimulation, repositioned by nurses". The patient's very last words were that she was "very tired", and feeling a little better, as evidenced by "States very tired" and "States feels a little better", also seen at N-6. The same record documents "Not communicative, able to follow simple commands. Movements very slow".

N-5 documents "Pupils dilated at approx 5mm" and "very little reaction to light", at 0040 hours. The ED physician, Dr. Spiller, was up to assess the patient's condition at 0055 hours. The same record documents "Dr. Jordan phoned re pt condition No change in orders", at 0100 hours, that by 0130 hours documents "Resps becoming more soaring in nature. No change in pupils & completely unresponsive".

N-5 also documents the respirations as "deep and soaring" as early as 0220 hours, that became "Gurgly" (a sign of constriction suggestive of thoracic trauma (patients are often in shock), followed by "deep snoring without constant jaw lift", such as associated with obstructive sleep apnea. The same record documents a heart rate in the 160's (sinus tackycardia), consistent with dosage related adversities to morphine administration. Morphine distributes to skeletal muscle, kidneys, liver, intestinal tract, lungs, spleen, and brain. Tackycardia (HR 160's) can also suggest a patient awareness.

A-26 documents a blood pressure of 162/80 with an SaO2 (arterial oxygen saturation) of only 80% at 0220 hours, followed by a potentially lethal drop in blood pressure to "78/70 bpm" by 0235 hours, suggestive of clinical insult, typical of a botched intubation procedure.

CAVEAT: Systolic blood pressure <80 mm HG is the hallmark of haemodynamic instability. The term "hemodynamic instability" is most commonly associated with an abnormal or unstable blood pressure, especially hypotension, or trauma due to clinical insult. Hemodynamic instability has also been defined more broadly as global or regional perfusion that is not adequate to support normal organ function. Low blood pressure, or hypotension, occurs when blood pressure during and after each heartbeat is much lower than usual. This means the heart, brain, and other parts of the body are not getting enough blood.

The same record at A-26 documents "Family in" at 0250 hours. On seeing the patient, we found her to be propped up in the arms of two nurses, gasping for air, with only a plastic oral airway in her mouth. A reason for this , according to the duty nurse was "to keep her from swallowing her tongue". Weakness of tongue and retropharyngeal muscles causes positional airway obstruction which can occur in unconscious supine patients; difficulty with protruding tongue and difficulty swallowing indicate that bulbar involvement is significant. In my opinion, Arlene Berry appeared to be more

paralyzed or blunted than anything, with the exception of lower limb/leg contractions suggestive of possible myoclonic generalized seizure activity, such as seen in a wide variety of nervous system disorders, which rapidly subsided.

I had asked the patient twice, in the presence of her foster brother, if she could hear me to "wiggle" her toes, and indeed she did, not once but twice, to be absolutely certain. Further, an observation made by her foster brother about the same time, was the seeming appearance of the patient attempting to pull her face forward as though trying to lift her head off the pillow. The inability to lift the head off the pillow by flexing the neck is a danger sign that frequently develops simultaneously with phrenic nerve (diaphram) weakness, such as seen in thoracic outlet obstruction, or peripheral neuropathy in persons with diabetes, compromised immune systems, or those who have suffered some sort of injury to these nerves. It may also suggest an acute meningeal irritation (neck stiffness) such as seen in "meningitis and/or encephalitis". Notably, flexion of the neck causes involuntary fexion of the knees which may also explain the lower limb/leg contractions hereinbefore described.

Many cases of infectious meningitis begin with a vague prodrome. The classic meningitis triad of fever, headache, and nuchal rigidity develops over hours or days. In severe cases, attempts at neck flexion may induce flexion of the hip or knee (Brudzinski's sign), and there may be resistance to passive extension of the knee while the hip is flexed (Kernig's sign). Neck stiffness and Brudzinski's and Kernig's signs are termed meningeal signs or "meningismus"; they occur because tension on nerve roots passing through inflamed meninges causes irritation. Nuchal rigidity is the pathognomonic sign of meningeal irritation and is present when the neck resists passive flexion; passive flexion of the neck results in spontaneous flexion of the hips and knees.

Guillain-Barre syndrome (GBS) may present with a wide range of clinical pictures. The symptoms of GBS and its variants can affect each patient differently and with varying intensities, so each patient can have a unique case history. In the initial stages, the patient is likely to have few if any symptoms

(Hughes, 1995). Some cases may be so mild that medical attention is never sought, and there are case reports of patients with near total or total paralysis, and some who were only able to move a few fingers and/or wiggle some toes, retaining only a little motion in some fingers or a foot.

Babinski's sign is a prominent finding in Bickerstaff's brainstem encephalitis (BBE), including the Miller-Fisher variant of GBS. In fact, many of these patients are reported to have retained the ability to wiggle their toes or feet up and down and plantar flex. In some cases, the only way the patient could communicate was by wiggling the toes yes or no, and constitutes evidence of responsiveness.

Notably, severe forms of hepatic encephalopathy lead to a worsening level of consciousness, from lethargy to somnolence and eventually coma. In the intermediate stages, a characteristic jerking movement of the limbs is observed which disappears as the somnolence worsens. In the third stage, neurological examination may reveal clonus and positive Babinski sign. Coma and seizures represent the most advanced stage; cerebral edema (swelling of the brain tissue) leads to death if untreated.

N-5 documents a "Sudden large queery bloody emesis, reddish brown liquid" at 0255 hours, on May 24th of 2000, inconsistent with what is known in medical circles as "coffee-ground emesis" ie. dark brown tinged vomit the color and consistency of coffee-grounds. The consistency of the emesis was indistinguishable from that of a thick milkshake, "reddish brown" in color, with no odor whatsoever. The literature describes an emesis of semiliquid fluid which is said to resemble "anchovy paste" as being suggestive of "amoebic liver abscess".

Amoebic liver abscess (ALA) with or without jaundice, and with or without hepatic encephalopathy has been reported. Amoebic abscess commonly presents as an acute entity, but it can also present as a chronic type where it is covered by a capsule that remains dormant for a given peroid of time. If the infecting organism invades the liver, it causes formation of the typical "reddish brown" anchovy

paste-like fluid (liquefied liver cells) with no odor. Normal liver function tests do not exclude the diagnosis. Liver function tests may be mildly abnormal, or normal. Case reports include liver function tests having normal bilirubin and liver enzymes. A patient may present with minimal symptoms despite having liver abscesses and intra-thoracic infection due to amoebiasis.

Amebic trophozoites also cause liver abscesses with well circumscribed lesions containing dead hepatocytes and cellular debris without a preceding phase of hepatitis. A rim of connective tissue, some inflammatory cells, and a few amebic trophozoites surround the lesion, whereas the adjacent liver parenchyma is usually completely normal. Amoebiasis can cause or mimic hepatic encephalopathy, with or without jaundice. The disorder may also be triggered by any condition that results in alkalosis (alkaline blood pH), low oxygen levels in the body, use of medications that suppress the central nervous system, infections including bile duct obstruction, or any coincidental illness. Any reduction in liver function may trigger encephalopathy.

The mildest form of hepatic encephalopathy is difficult to detect clinically, but may be demonstrated on neuropsychological testing. It is experienced as forgetfulness, mild confusion, and irritability. The first stage of hepatic encephalopathy is characterised by an inverted sleep-wake pattern (sleeping by day, being awake at night), which pretty much fits Arlene Berry's sleep pattern on the week or so prior to her death. The second stage is marked by lethargy and personality changes. The third stage is marked by worsened confusion. The fourth stage is marked by a progression to coma.

Amoebic liver abscess is an **enigma** as it has been observed in people with no evidence of previous amoebic colitis or history of tropical travel. The only contact the patient had with anyone from an endemic area was the thoracic surgeon who performed the left lung pneumonectomy, namely Dr. De La Rocha.

Hepatic encephalopathy may occur suddenly in people who previously had no liver problems when

damage occurs to the liver. Hepatic encephalopathy is caused by disorders that affect the liver. These include disorders that reduce liver function and/or conditions in which blood circulation does not enter the liver. Severe forms of the encephalopathy lead to a worsening level of consciousness, from lethargy to somnolence and eventually coma. In the intermediate stages, a characteristic jerking movement of the limbs is observed; this disappears as the somnolence worsens.

The symptoms of hepatic encephalopathy may also arise from other conditions, such as cerebral haemorrhage and seizures. Notably, mimics of encephalopathy include "meningitis, and encephalitis". Amoebic cerebral abscess mimicking bacterial meningitis has also been reported in the literature.

A-19 of the record documents the mechanical charting of the patient's HEMATOLOGY, beginning with a WBC of 22.4 with a normal reference range of 4.0-11.0. The presence of an elevated WBC count is called Leukocytosis. An increase in WBCs may occur in many conditions, including infection (viral, bacterial, fungal, and parasitic), allergy, leukemia, hemorrhage, traumatic tap, encephalitis, and Guillain-Barre syndrome. When the number of WBCs in your blood increases, this is a sign of an infection somewhere in your body.

WBC’s (leukocytes) are the body’s primary defense against infection and also reflect the degree of physiologic stress. WBC’s are also elevated with dehydration, and hyperviscosity secondary to dehydration. A high WBC may also indicate that there is inflammation of the central nervous system as in meningitis.

The WBC differential helps to distinguish many of these causes. For example, viral infection is usually associated with an increase in lymphocytes, while bacterial, fungal and certain parasitic infections are associated with an increase in polymorphonuclear leukocytes (neutrophils). More precisely, migration of Polymorphonuclear leukocytes (PMNL) across epithelial linings is a hallmark

of several gastrointestinal (GI) disorders. Moreover, the presence of PMNL is noted in gastroenterocolitis induced by ischemic conditions, and by various toxic chemicals or drugs. An increase in the WBC count is also a typical response to noxious stimuli.

The histological hallmark of ulcerative colitis is "polymorphonuclear leukocyte" invasion of the mucosa and sometimes of the sub-mucosa of the colon. Ulcerative colitis is an inflammatory bowel disease that affects the lining of the large intestine, also called the colon. The disease usually causes diarrhea, blood in the stool, and abdominal pain, and can also lead to nausea, lack of appetite, weight loss, and anemia. In addition, malaise, anorexia, weight loss, moderate to severe abdominal pain, low grade fever, chills, muscle pain, signs of peritonitis, toxemia, and other systemic symptoms may also be present. Incontinence is often a problem especially when the rectum is severely inflamed.

Amoebic Colitis has been found to be associated with progressive abdominal pain, and signs of peritonitis, leukocytosis, hyponatremia, hypokalemia, bloody mucoid diarrhea and dehydration. In the early stage, the patient is usually generally well with mild or moderate abdominal pain. Symptoms often fluctuate over weeks or even months with the patient becoming debilitated.

While diarrhea, pain, and blood in the feces are common with Ulcerative colitis, the extent of disease varies greatly from person to person. Severity of disease is categorized as mild, moderate, or severe according to clinical symptoms. In severe cases, patients experience frequent episodes of bloody stools and they may become anorectic and nauseated. In severe attacks, patients may vomit and experience symptoms of anemia such as breathlessness, and fatigue. Weight loss, fever, fast heartbeat, dizziness, and severe cramping or abdominal pain can also occur with severe cases of the disease. The most common early symptoms of ulcerative colitis are constipation with passage of blood or mucus in the stools. Untreated patients may develop toxic megacolon. There is a potential for central nervous system toxicity. The use of opioids can lead to a rapid deterioration and colonic perforation.

The typical lesion doctors see in ulcerative colitis is called a "crypt abscess" and are located in the mucosa of the large intestine. The major organs of the abdomen include the small intestine, large intestine, and stomach. The presentation of abdominal abscess is similar to that of peritonitis, but the symptoms are generally milder. The most feared complication from severe ulcerative colitis is toxic megacolon. Perforations can also occur in severe ulcerative colitis even if toxic megacolon does not develop. Most perforations occur in the left colon, commonly in the sigmoid colon. Perforations tend to occur more often during first episodes of colitis. Perforations must be treated surgically. Among the possible environmental factors, no specific foods have been identified as a cause of ulcerative colitis (UC). Possible risk factors include immunologic factors, infectious agents (such as bacteria, viruses, or amoebae), and dietary factors (including chemicals and drugs).

Some people with ulcerative colitis are intolerant of cows' milk and find that dairy products may aggravate symptoms. Cigarette smoking actually reduces the risk, though what component of tobacco has a beneficial effect on the colon lining is not entirely clear. Smokers have only about 40 percent of the risk of developing ulcerative colitis of nonsmokers.

The record at A-19 documents a Neutrophil count of 92.0 H (47.0-77.0 is the normal reference range), with an Absolute Neuts of 20.0 H (1.3-6.71 is the normal). Neutrophilia (or neutrophil leukocytosis) is a condition where a person has a high number of neutrophil granulocytes in their blood. Normally, neutrophils account for 50-70% of all leukocytes. If the count exceeds this amount, the cause is usually due to an acute infection. Neutrophils, are also known as "segs", "PMNs" (polymorphonuclears), or "poly's". PMNs are the primary effector cells in the innate immune response against infection. A high neutrophil blood count is a sign that something in your body has triggered an immune response.

Neutrophilia may be due to a number of acute and chronic causes such as infection, inflammation,

emotional stimuli, drugs, metabolic hormonal, and endocrine disturbances, including hematologic abnormalities. The ratio of neutrophil and lymphocyte counts has even higher value in predicting bacteremia.

Polymorphonuclear leukocytes (granulocytes) accumulates in brain regions with low blood flow during the early postischemic period. Polymorphonuclear cells (PMNs) usually represent the predominant cell type in an inflammatory response, acting as the first line of defence against invading organisms. Severe inflammatory response may mature into a systemic response known as anaphylaxis.

Leukocytosis, especially neutrophilia, indicates "systemic infection" and is rare in the absence of "superinfection", such as bacteria, viruses or mixed infections which are resistant to antibiotics. Neutrophils are also associated significantly with the density of "parasites", such as seen in amebic infection and has been suggested that the damage observed in invasive amebiasis is related to interactions between polymorphonuclear leukocytes (PMN) and Entamoeba histolytica. If the total WBC is high due to a rise in neutrophils and eosinophils, then an allergic, or parasitic process is most likely.

Other causes of an increased neutrophil count include "cerebral abscess". Brain abscess are usually mixed infection. Ampebiasis is high on the order of "mixed infection", includng brain abscess.

There are two basic types of leukocytes. The phagocytes which are cells that chew up invading organisms, and the lymphocytes, which are cells that allow the body to remember and recognize previous invaders.

Neutrophilic leukocytosis (neutrophilia) is also high on the order of acute bacterial infections, especially pyogenic (pus producing) infections. Furthermore, amebic abscess is a common

manifestation of extraintestinal amebiasis. Laboratory findings may also reveal "leukocytosis with neutrophilia". There is neutrophilia in 90% of patients with amoebic liver abscess. When there are several abscesses, or a large abscess, leukocytosis with neutrophilia and increased fibrinogen levels develop. Untoward sequelae include peritonitis after abscess rupture into the peritoneal cavity, and sudden death from an anaphylactic or toxic reaction when there is rupture of an abscess into hepatic blood vessels.

A-19 documents a Lymphocyte count of 2.0 L (20.0-50.1 is the normal), with an absolute Lymphs count of 0.4 L (1.0-4.5 is the normal). An abnormally low level of lymphocytes in the blood is called lymphocytopenia, or lymphopenia. Lymphocytopenia can range from mild to severe. The most common causes for lymphocytopenia are autoimmune disorders. Autoimmune disorders occur if the body's immune system mistakenly attacks the body's cells and tissues. A low lymphocytes count (lymphocytopenia) results in the inability to remember and recognize and hence fails to distinguish.

An autoimmune disorder is a condition that occurs when the immune system mistakenly attacks and destroys healthy body tissue. A low lymphocyte count makes it hard for your body to fight infections. Lymphocytes help protect your body from infection.

People with lymphocytopenia have a weakened immune system and tend to get a lot of unusual infections. due to reduced antibody production (a weakened immune system is mainly caused by the presence of toxins in your body). Absolute lymphocytopenia can also be used in the prediction of infectious emergency admissions. Severe lymphocyte deficiencies can result in uncontrolled infections that can be "fatal".

A-19 of the record documents an Absolute Lymphs (lymphocytes) of 0.4 L with a normal reference range of 1.0-4.5.

Molecular mimicry refers to the situation where the pathogen and host share nearly identical antigens, which induces an antibody and T cell immune response that is cross reactive. Notably, the granulomas of sarcoidosis are caused by collections of immune system cells, particularly T cells. Further, molecular mimicry leads to autoimmunity when parasites activate lymphocytes that cross-react where a foreign antigen shares sequence or structural similarities with self-antigens. Molecular mimicry has typically been characterized on an antibody or T cell level. The most commonly proposed mechanism for the development of autoimmune disease is molecular mimicry (Yuki, 2005)..

Aggressive mimicry is a form of mimicry where predators, parasites or parasitoids share similar signals with a harmless model, allowing them to avoid being correctly identified by their prey or host .

Neutrophilia facilitates the inflammatory response, whereas when neutropenia is present, the inflammatory response to such infections is ineffective. The end result is an autoimmune reaction. The immune system fails to properly distinguish between self and non-self, and attacks part of the body. There are more than 80 different types of autoimmune disorders. If someone with lymphocytopenia gets any kind of infection, it has to be treated as an emergency. Even minor problems can progress much more seriously for people with this disorder than they would for people with normal immune systems.

Your low lymphocytes are what we expect to see in sarcoidosis. Opportunistic infections can resemble sarcoidosis. They can also co-exist with sarcoidosis. Th1 diseases are lymphopenic, which means a lack of T-cells. Lymphocytopenia can also be a side effect of radiation treatment. RadiationTherapy (RT) probably caused immunosuppression and increased susceptibility to infection. People with T cell deficiencies are particularly susceptible to intracellular pathogens. Entamoeba histolytica is the pathogen responsible for 'amoebiasis'.

People who have too few T lymphocytes or too few natural killer cells have problems controlling

certain infections, especially viral, fungal, and parasitic infections. Low levels can also indicate sepsis (blood poisoning or toxemia). Severe sepsis is associated with "organ dysfunction, perfusion abnormalities, and hypotension". Indeed, NO septic workup was ever done.

Interestingly, iatrogenic lymphocytopenia is caused by either cytotoxic chemotherapy or radiation therapy, or both, marked by a reduction in the absolute number of T cells. Lymphocytes are the most sensitive to whole body radiation and their count is the first to fall in radiation sickness. The number of lymphocytes declines within the first 12 to 48 hours after exposure. This is followed over several weeks by a decline in the number of other blood cells. The decline in lymphocytes is one of the best early signs of the severity of the radiation injury. Radiation therapy, in this case, is undoubtedly the cause of immunosuppression and an increased susceptibility to invasive bloodborne carriage of infection.

Idiopathic CD4 lymphocytopenia (ICL) is a presumed heterogenous syndrome with key element low CD4 T-cell counts (below 300/mm3) without evidence of HIV infection or other known immunodeficiency. The clinical presentation can range from serious opportunistic infections to incidentally diagnosed asymptomatic individuals.

A-19 documents an RBC (red blood cell) count of 4.30 (3.80 – 5.80 is normal), but the HCT (hematocrit) is below the normal level. HCT is also the measurement of the percentage of red blood cells (RBC's) in whole blood and in this case HCT is only 0.361 (normal reference is 0.370-0.470), with a reduction suggestive of anemia. The result is fewer red blood cells to carry oxygen. Thus anemia is present when HCT is < 1.0 x 109/L. The patient is usually noticeably pale, as in this case.

A hematocrit (HCT) test is frequently done to assess the extent of significant blood loss. A decreased hematocrit can be due to either overhydration, which increases the plasma volume, or a decrease in the number of red blood cells caused by anemias or blood loss. If a person with a normal blood cell

volume loses blood suddenly through a massive hemorrhage, the person may develop signs and symptoms of circulatory shock and the blood pressure will fall.

For the record, "parasitic infection" may also provoke significant reduction in HCT (hematocrit), and lymphocyte percentage.

A-19 also documents an RDW of 18.4 H. Red cell distribution width (abbreviated as RDW) is a measurement of the amount that red blood cells vary in size. A normal RDW level is 11.5-16.8. Red blood cells help carry oxygen in the blood. An elevated RDW (red blood cells of unequal sizes) is known as anisocytosis. This is commonly found in anemia and other blood conditions.

A-19 documents a Monocyte count of 3.0 (2.0 – 10.0 is the normal), with an Absolute Mono's of 0.60 (<1.0). The monocytes are a type of phagocyte which mature into “macrophages”; they are also important germ eating cells. Patients with a low monocyte count have a higher risk of getting sick from an infection. In this case, the Monocyte count is in the low normal range. For the record, malignant conditions such as leukemia or lung cancer can lead to increased monocyte levels; cancers tend to raise blood monocyte levels. That is NOT the case here.

A-19 also documents a Fibrinogen level of 4.67 H (the normal range is 2.00-4.00). Fibrinogen plays two essential roles in the body: it is a protein called an acute-phase reactant that becomes elevated with tissue inflammation or tissue destruction. Elevated plasma fibrinogen is called "hyperfibrinogenemia"; high levels induce a state of "hypercoagulability". Serum fibrinogen levels in a safe range is <300 mg/dL. An elevated fibrinogen level may also be seen with TRAUMA of any kind, with risk of cardiovascular disease and arterial and venous thrombosis.

Most commonly, fibrinogen is decreased in DIC; however, increased fibrinogen levels do not rule out DIC since fibrinogen is an acute phase protein synthesized in the liver and is often elevated in

inflammatory conditions. Increased fibrinogen values occur in inflammation, infections and tissue damage/trauma.

A-19 documents a D-dimer test level of 1000 H. An increase in fibrinogen and d-dimer correlates with thrombotic activity suggestive of thrombosis. Thrombosis signifies the formation of blood clotting within vessels of the brain or neck. People who are suffering from a severe infection are more likely to develop dangerous blood clots, but inappropriate combinations of medications or treatment can sometimes be the worst offenders.

Based on the patient's CBC's, the lesions are consistent with collections of purulent exudates suggested by an elevated Neutrophil count in the presence of an elevated WBC count, and/or pockets of pooled blood, as suggested by an elevated Fibrinogen in the presence of an elevated D-dimer, a hallmark of thrombus formation, ie., blood clots. The enhancement is obviously due to infection, or blood pooling, or both.

Thrombosis, particularly of the mesenteric or portal vessels, may occur during acute amebiasis, as with any other infection. Usually it is a complication of the disease, but amebae have been recovered in large numbers from thrombi within the inferior vena cava. They become an important source of thromboemboli, giving rise to pulmonary or rare cerebral abscesses.

A-19 documents a Platelet count of 544 H with a reference range of 150-450. A platelet count is used to detect a low or high number of platelets in the blood. The test is included in a complete blood count (CBC). An elevated platelet count is known as thrombocytosis. Platelets are one of the three cellular elements of the blood, whose function (along with the coagulation factors) is to stop bleeding. The final result is the clot. Platelet aggregation and thrombi form because of the increased viscosity of the blood. This can result in "decreased tissue perfusion" and the development of disseminated intravascular coagulation (DIC), a disorder characterized by procoagulant substances

entering the general circulation causing a systemic thrombotic process. The activation of the clotting mechanism may arise from any of a number of disorders. However, platelet counts in DIC are usually low.

A higher-than-normal number of platelets (thrombocytosis) may be due to anemia (too few red blood cells). The same record documents a "PLT ESTIMATE – MOD INCREASE" confirming an increase in platelet aggregation activity, blood platelets sticking together, indicating that blood thinners may be needed to prevent blood clots. Abnormally high platelet levels (thrombocytosis) may indicate either a benign reaction to an infection, surgery, or certain medications; or a disease like polycythemia vera, in which the bone marrow produces too many platelets too quickly. In addition, reactive thrombocytosis may occur, which is an elevated platelet count that may also be due to an infection, an operation or an acute blood loss. Further, reactive thrombocytosis may be caused by inflammation due to such causes as rheumatoid arthritis, or to an inflammatory bowel condition.

In certain cases, increase in platelet count can give rise to serious complications as a result of unexplained blood clotting. This can lead to deep vein thrombosis, pulmonary embolism (a clot in arteries that carry blood to the lungs) and even a heart attack or stroke. Because high platelets can be due to serious diseases, failure to treat the condition can result in serious complications and damage. Potential complications of high platelets may include: bleeding, blood clots, brain damage such as stroke, inflammatory or infectious diseases, such as connective tissue disorders, tuberculosis. Some signs and symptom of an elevated platelet count are: Bleeding from the nose mouth or rectum or unexplained bruising and heavy or prolonged menstrual cycle.

For the record, an infection is often accompanied by a raised platelet count. The other causes of an elevated platelet count may include certain types of cancer and polycythemia vera (too many red blood cells), a recent splenectomy, reactions to medicines. On the other hand, lower-than-normal number of platelets (thrombocytopenia) may be due to cancer, chemotherapy, certain medicines, or leukemia.

A-19 documents an aPTT (activated Partial Thromboplastin Time) of 60-100 seconds (23-35 is the normal). The test is used to determine the efficacy of various clotting factors used in the diagnosis of coagulation disorders. The time of that assessment was documented at 0400 hours. The aPTT is typically elevated in 90% of those with coagulopathy. Changes in the haemostatic profile - prolonged activated partial thromboplastin time, shortened thrombin time, increased concentration of D-dimer and thrombocytopenia suggest the development of disseminated intravascular coagulation, most likely due to influence of blood clotting factors secondary to dehydration, or infection, or both.

A-20 documents the O2 Sat (oxygen saturation) with an arterial oxygen saturation (SaO2) of 98.9 H . (95-98 is normal), that is slightly elevated at 1720 hours. The oxygen saturation is the amount of oxygen actually carried by the hemoglobin.

A-20 of the record also documents a pO2 (Partial Pressure of Oxygen) of 129.0 H, at 1720 hours. The PO2 measures the amount of dissolved oxygen in the blood and is measured in mmHg. The normal reference range is 78-100 mmHg. High oxygen concentrations predispose to oxygen toxicity. Oxygen toxicity is an "iatrogenic" illness caused by a high partial pressure of inspired oxygen during the course of oxygen therapy. Oxygen toxicity is a condition resulting from the harmful effects of breathing molecular oxygen (O2) at elevated partial pressures. It is also known as oxygen toxicity syndrome, oxygen intoxication, and oxygen poisoning.

Unnecessarily high levels of pO2 (generally above 120 mm Hg) can elicit "cardiac ischemia" and produce dysrhythmias. The hallmark of cardiac ischemia is an "inferior ischemia". The acute toxicity has predominant CNS effects, while chronic toxicity has predominant pulmonary effects. The first and most important method to prevent pulmonary oxygen toxicity is to limit exposure to the lowest possible pO2. If high, patient may be "over ventilated". Too much oxygen normally has harmful effects and it can sometimes lead to death. This is because it does not allow the exchange of gases to take place and it might end up overworking the lungs as well.

A-20 of the record documents a Glucose of 13.2 H mmol/L (the normal range is 4.1 – 7.8). High blood sugar usually comes on slowly. To convert mmol/l of glucose to mg/dl, multiply by 18. (13.2 x 18) = 237.6 mg/dl. Glucose is 13.2 H mmol/L = 237.6 mg/dl. Symptoms of severe high blood sugar (hyperglycemia) include drowsiness and difficulty waking up. Acute, short-term hyperglycemia may precipitate vascular occlusions by facilitating platelet activation. With infection, blood sugar levels tend to rise quickly over several hours.

Certain drugs can also affect blood sugar levels. It should also be borne in mind that certain medications such as Morphine and phenothiazine derivatives such as Stemetil actually contribute to the occurance of hyperglycemia. In fact, both have been reported to "trigger diabetes" in patients with no previous history of diabetes. See transient hyperglycemia.

A-18 of the medical record documents the consideration of an "inferior ischemia", a sign of reduced oxygen supply to vital organs due to reduced or poor blood flow to the heart. An inferior ischemia is the hallmark of "impaired organ perfusion", as it implies that, unless corrected, there may not be enough oxygen in the blood to sustain vital organs; inadequate cerebral perfusion can be avoided by removing vasoconstricting factor(s), improving peripheral blood flow, and reducing metabolic demands on the body.

The same record at A-18 documents "Sinus Tachycardia". It occurs when the sinus rhythm is faster than 100 beats per minute (bpm) ; also associated with shock, hypotension, hypoxia, congestive heart failure, and various high output states. Most often sinus tachycardia is caused by an increase in the body's demand for oxygen; sinus tachycardia can have serious consequences and put the patient at risk for iatrogenic harm.

A-20 documents a total CK (Creatine Kinase) level of only 40 units per liter (U/L) at 0400 hours. The

relative index of total CK is a parameter used to help determine health status. In females, total Creatine Kinase should be 10-79 units per liter (U/L), not 30-135. as suggested by the reference range set by the phyician who performmed this test. The creatine kinase level usually parallels a disease activity. In normal conditions, there is very little Creatine Kinase circulating in the blood of the average, healthy human being. CK is the most sensitive enzyme and in the presence of most diseases, levels can be elevated as much as 50 to 100 times the reference level. However, in certain conditions, muscle inflammation with normal or near-normal CK levels have been reported.

The hallmark of muscle wasting (or muscle damage) is elevation of CK. The wasting away of fat and muscle (cachexia), is the most visible hallmark of metastatic cancer. Persons with cancer typically have high CK levels. Elevation of CK may also be seen in stroke, extreme shock, and "brain tumors" in which CK levels can sometimes temporarily go off the scale, topping out at 50,000 to 200,000 U/L, a sign of severe muscle fiber breakdown (necrosis). CK levels may rise significantly in about 2 to 3 hours.

Significant increases in serum CK have been observed and reported in cases of "adenocarcinoma" and SCAC of the lung with proven CNS metastases, while patients with oncological conditions other than SCAC of the lung have failed to show elevation in serum CK. Further, in various cases of infectious myositis, muscle enzymes such as creatine kinase are paradoxically "normal despite muscle inflammation". In this case, CK is well within the normal range.

Serum CK is considered a useful diagnostic marker or indicator of active metastases. A normal Creatine Kinase (CK) at the time of the patient's admission to hospital on May 23rd of 2000, and in particular, the time of this assessement (1720 hours), would argue favorably against a diagnosis of metastatic CA.

For the record, there are no specific biochemical abnormalities in granulomatous myopathy. Serum

creatine kinase (CK) levels are typically normal, but mild to high elevations have been reported in both idiopathic and sarcoid myopathy. Granulomatous myopathy, in the presence or absence of sarcoidosis, is commonly associated with dysphagia (87%) and a normal serum CK [PubMED]. Overall and similar to sarcoidosis, granulomatous myositis is more frequent in women, while the idiopathic form is more often seen in men. The levels of serum CK are different in various myopathies according to the type of disease and the stage of pathology. For example, drug-induced myopathy may show only slight, or no increase in CK activity.

A-20 of the record also documents a Serum Potassium level of 3.4 L at 0400 hours with a normal reference range of 3.6-5.0 on May 24th of 2000. Hypokalemia is defined as a potassium level below 3.5 mEq/L. Hypokalemia is a metabolic disorder that occurs when the level of potassium in the blood drops too low. The two major causes for the loss of potassium from the digestive system can be vomiting and diarrhea. In addition to common symptoms such as vomiting, nausea, constipations, hypotension including an array of different symptoms, muscle weakness is the most predominant. Hypokalemia may also cause slow movement of the colon. Also, for the record, in severe cases of amoebiasis, leukocytosis with neutrophilia, and hypokalemia may occur. Notably, all three were present in this patient.

A-20 documents an Arterial pH of 7.437. A normal pH is 7.35 – 7.45. The ideal pH for blood is 7.4. With a blood pH of 7.437 the pH is optimal. The time of that assessment is documented at 0400 hours on May 24th of 2000. Notably, the kidney and the liver are two main organs responsible for the metabolic homeostasis of pH. The same record documenst a Base Excess of 1.0 with a reference range of 2.0. If the base excess is less than – 2, then there is a metabolic acidosis, which may be the compensatory process.

Hydrogen ion concentration expressed as pH "Power of Hydrogen" (Humans as organisms) scale of acidity/alkalinity. pH below 7 = acidic, pH above 7 = alkaline. Neutral pH is 7, in this case on the

alkalemic side of normal. Acids and alkalis are the chemicals at each end of the pH spectrum. The scale runs from 0 (acid) to 14 (alkali). For example, the pH of blood is normally 7.4 and that of muscle is 7.0. pH under 7 is acid; pH over 7 is basic or alkaline. The metabolic pathways of the body require a slightly alkaline environment. Ph 7.0 and higher indicates alkalinity.

Our blood pH has a very narrow range of around 7.35 to 7.45. If our body's pH deviates from this range, we will be sick or have symptoms of falling sick. a blood with a pH value of 7.45 contains 64.9% more oxygen than blood with a pH value of 7.30. The heart is normal when the pH of blood plasma is slightly alkaline, having a pH of 7.35 to 7.41. An absolute blood measurement of acidity (pH below 7.0) is incompatible with sustaining life.

Further, it is a well known fact that cancer thrives in an acidic pH environment in the body, but cannot survive in an alkaline pH environment. At a pH slightly > 7.4 cancer cells become dormant. With metastases the pH drops to 6.0 and even 5.7 or lower. Terminal cancer patients are around 1000 times more acidic than normal healthy people. The vast majority of terminal cancer patients possess a very low body pH. In the case of Arlene Berry, her pH was optimal - as good as it gets.

N-10 of the Nurses' Notes document the patient's level of care as "routine", which shows little or NO concern for patient safety. Further, NO close patient monitoring or toxicological screening was ever done or even suggested, marked by a complete absence of nursing care plan, as evidenced at A-21 of the record. In fact, NO protocols were ever followed or implemented in this case.

The record at N-6 documents the discovery by duty nurses of the patient's "head against the left side bed rail with her feet under the right side rail", usually the result of proprioception (loss of motor control), or sense of one's own perception of the relative position of neighboring parts of the body to each other, which is occasionally impaired spontaneously, especially with extreme fatigue. Loss of coordination is part of the same problem. Notably the time of that entry is documeented as being

0030 hours, and is preceded by a documented 2330 hours, suggestive of having left the patient unattended one full hour.

Could this have been the result of a transient ischaemic attack (TIA), possibly due to inappropriate treatment, or perhaps as a presenting clinical manifestation of an undiagnosed condition? In a transient ischaemic attack (TIA), your brain is temporarily starved of oxygen and nutrients by having the blood supply blocked. A TIA (Mini-Stroke) happens suddenly, without warning. The blood flow may be reduced by a narrowing or blockage of the blood vessels, or granulomas clustered around blood vessels.

The ED physician, Dr. Spiller was up to assess the patient's condition. Upon examination her eyes were documented as being "sluggish", with "pupils dilated at approx. 5 mm" with "very little reaction to light", and far from getting better, she was becoming progressively worse, as evidenced by a sense of urgency seen on the record to the attendance of the patient with increased activity between 0030 and 0055 hours, evidenced at N-5 and at N-6. The same record documents "no response to verbal or physical stimulation, repositioned by nurses in bed & placed right lateral position".

The position of the body during sleep can have a significant effect on the development of Cheyne-Stokes breathing. Data suggests that sleeping supine and at a flat head angle can significantly increase the likelihood of Cheyne-Stokes breathing.

The record at N-5 documents a physician "assessments unchanged" at 0235 hours, despite the fact that the patient had already gone into respiratory distress at that time, evidenced by "Cheyne-Stokes" respirations with periods of "apnea" lasting "5-8 seconds". Sleep apnea means cessation of breath. It is characterized by repetitive episodes of upper airway obstruction that occur during sleep, usually associated with a reduction in blood oxygen saturation, includjng pulmonary dysfunction. It occurs when something obstructs breathing in the upper airway.

N-3 of the record documents "resp noisy", "shallow", "Cheyne-stoke" at 0320 hours. Cheyne-stokes breathing is a respiratory pattern that oscillates between hypoventilation and hyperventilation, usually the result of diencephalic insult. Hyperventilation = too much gas exchange. Hypoventilation = not enough gas exchange.

A-24 of the record documents a heart rate of 174 beats per minute (bpm) at 0320 hours that is consistent with Ventricular Tachycardia (VT), a cardiac arrhythmia in which the muscles of the ventricles contract irregularly in a rapid, uncoordinated manner, impairing the normal pumping of blood, suggestive of trauma. A normal resting heart rate for adults ranges from 60 to 100 beats a minute. Ventricular tachycardia (VT or V-tach) is a potentially life-threatening cardiac arrhythmia that originates in the ventricles. It is usually a regular, wide complex tachycardia with a rate between 120 and 250 beats per minute. Ventricular tachycardia has the potential of degrading to the more serious ventricular fibrillation. Ventricular tachycardia is a common, and often lethal, complication of a myocardial infarction (heart attack). An episode of extreme terror (pain fright, extreme emotional stress), can also result in ventricular tachycardia, and potentially culminate in death.

The patient became apparently unresponsive, as evidenced at N-5, and went into respiratory distress, requiring ventilation for which she was transferred into ICU at 0320 hours, according to the record at N-3. The same record documents the time of the patient's intubation by Dr. Jordan at 0325 hours, some 5 minutes later. What I take to be the Ventilation Record at A-17 documents the arrival in the ICU of the hospital's ventilatory therapist, Helene Studholme at 0330 hours, after being "called in for patient requiring ventilation".

From these records, it is clear that the ventilatory therapist was NOT present at the time of the intubation procedure because she did not show up until 5 minutes later. Furthermore, submit that the intubation actually took place at an earlier time, such as 0320 hours. The record at A-24 documents

a heart rate (HR) of 174 bpm at 0320 hours that is consistent with an "awake intubation" (any suspicion of difficulty intubating, for any reason), marked by panic with awareness (shock), resulting in the increase in heart rate to 174 bpm.

The Vital Signs Record at A-24 documents a heart rate of 174 bpm at 0320 hours that is consistent with trauma, while the Ventilation Record seen at A-16 documents a heart rate of only 126 bpm within the same time frame, a significant difference, suggesting that the timeline for that event was in fact altered by the Ventilatory Therapist, Helen Studholm, to conceal evidence of iatrogenic trauma related injury. Further, the vital signs can offer clues on the degree of dehydration present. Tachycardia can indicate moderate dehydration, whereas hypotension is a late sign of severe dehydration. Severe dehydration (loss of 10-15% of body fluids) is a life-threatening condition that requires immediate medical care.

Know that the Vital Signs Record is a mechanical record with a run time, while the Ventilation Record, in this case, is a handwritten account, marred by having been "rewritten". Which method of recording is more likely to make entry errors, or downplay an event by omission, or to incorporate lies due to potential liability issues? Additionally, there is nothing on record to suggest that anesthesia was ever given to prepare the patient for the intubation procedure. Also, for the record, know that the earliest indication of shock is an increase in heart rate, in this case the 174 bpm at 0320 hours evidenced at A-24.

According to Dr. Jordan "the intubation proceeded uneventfully", while N-2 of the record documents the ET (endotrachial tube) was "pulled back 4 cm" (1.5748 inches) at 0425 hours. From that record it seems clear that the endotrachial tube had been malpositioned for one full hour or more before the error was discovered by one of the nurses, as to infer negligence, including failure on the part of all concerned, and Dr. Jordan, in particular, to identify an incorrectly placed airway in a timely manner, evidenced at N-2. Both myself and the patient's foster brother were present to witness this event.

When an endotrachial tube is misplaced in the esophagus and misplacement is detected late, a compromise of the patient's safety can be significant. Malpositioning of the ET tube can cause airway obstruction and may also result in tissue trauma and bleeding. Iatrogenic perforation is the leading cause of esophageal perforation.

Endotracheal tube malpositioning into a mainstem bronchus or the esophagus may result in significant hypoxemia (inadequate oxygenation of the blood). This will be evident through the assessment of several vital signs. Sympathetic responses are hypertension, tachycardia, and tachyarrhythmias. Vagal responses are laryngospasm, bradycardia, hypotension, cardiac arrest, and apnea. In some patients there is even possibility of increased intracranial pressure and increased intraocular pressure In addition to hypoxia, delayed tube repositioning can lead to unilateral pulmonary edema. When ventilation is not achievable, irreversible brain damage can result within minutes. Therefore, the maximum interval allowable for conservative airway management maneuvers is about 3 minutes.

The ambulance call report seen at N-7 documents that the patient was intubated and vented and that she was seen to be "stable", and also that she appeared to be "pale, dry and cool". Pale skin suggests decreased blood supply to the skin. Blood vessels in the body constrict to conserve blood in the body's core, making you feel cold and your skin go pale. Dry skin suggests a dehydrated patient. Cool, dry skin can also suggest late sepsis. Patients progressing from sepsis to severe sepsis become profoundly dehydrated. The skin is the first place to be robbed of water, resulting in "dry" skin. The skin turns "pale and cold".

Caveat - Sepsis causes profound hypotension. Sepsis refers to the presence of an infection, plus any two of these four criteria:

. Heart rate greater than 90 beats per minute
. Increased respiratory rate
. High (or low) white blood cell count
. Fever or low body temperature

Visible symptoms of sepsis include nausea, vomiting and chills in the presence of an infection. Other signs that can also suggest sepsis are tachycardia, tachypnea, hypotension, an identified source of infection, and toxic appearance. Nonspecific but contributing findings include leukocytosis, neutrophilia, and toxic granulations.

Hallmark of infection is fever; however, temperature can vary with severity of sepsis, typically "37.2°C" or <36°C can be indicative of severe infection.

The most frequent sites of infection leading to sepsis are the lung, urinary tract, abdomen, and pelvis. In up to 30 percent of patients, however, a definite source of infection most often cannot be identified. The course of the disease is unpredictable. Some patients quickly deteriorate, while others suffer from varying degrees of organ dysfunction or begin to recover.

A-26 documents a body temperature slightly above 37°C, at 0330 hours, suggesting a low-grade fever marked by an elevation of body temperature above the normal range of 36.5–37.5°C (98–100 °F). Pathogenic bacteria grow best at human body temperatures in the 37 C range. Evidence of toxoemia in the form of low grade fever (37.5-38°C) has been reported in the literature. For the record, a parasite is the likely source of organism that grow best in the 37°C range. Notably, all acanthamoeba capable of growth at or above 37°C are potential pathogens (more virulent amoebas).

The record at A-17 documents a complete cessation of the use of abdominal and accessory

muscles, evidenced by a "0 use of acc muscles"; and a "0 use of abd muscles"; the muscles of respiration, accessory muscles and diaphragm are affected, suggestive of respiratory and accessory muscle "paralysis" (crisis) requiring intubation and mechanical ventilation. The time of that assessement is documented at 0330 hours. Accessory muscle paralysis will result in apprehension and anxiety = panic.

N-2 documents "attempts to pull away to painful stimuli" at 0400 hours on May 24th, while N-5 documents "No response to deep pain" at 0055 hours, suggests a state of "stupor" marked by mental dullness. Stupor is an excessively long or deep sleeplike state. A person can be aroused from it only briefly by vigorous stimulation, such as repeated shaking, loud calling, pinching, or sticking with a pin. Reactions to certain drugs, dehydration, and infections are common causes of impaired consciousness; in this case, compounded by "neurolept-analgesia".

Stupor and coma are characterized by impairment of the arousal system. In stupor, a person arouses only in response to strong verbalor tactile stimuli, awakens briefly, and then lapses back into a sleep-like state after the stimulation stops. In coma, a person cannot be roused to consciousness.

Was it the doctor's belief that Arlene Berry ceased to be a human being after becoming unresponsive following undiagnosed, untreated and/or inappropriately treated conditions? Or was there a more sinister plot afoot? So much so that he decided to write her off? He showed absolutely no concern for this patient whatsoever.

A-26 of the record documents "gurgling & snoring", as evidenced in the lower left corner of that record, at 0220 hours, while the record at N-5 makes aa reference to "Gurgly resps" at the very same time. Gurgling is a bubbling sound. It usually indicates upper airway obstruction from throat secretions, or presence of fluid in the airway due to excessive pooling of oral secretions. Suctioning is the protocol used for clearing oral secretions. In this instant suctioning was not done and the

patient's airway was undoubtedly compromised by the insertion of an oral airway, evidenced by "airway inserted", without having first cleared the airway of debris. The insertion of an oral airway stimulates the gag reflex and may also stimulate airway spasm or cause the patient to retch possibly resulting in asperation.

The record at N-5 documents the respirations as "deep and soaring without constant jaw lift" as early as 0220 hours.

The record at N-4 documents "incontinent blood tinged urine" at 0305 hours. Incontinent tinged urine is consistent with dehydration, often mistaken for hematuria (blood in urine). Incontinence can also be the result of hypotonia, or neurogenic bladder. Urinary incontinence can be a symptom of both over-hydration and dehydration. Hematuria is inconsistent with a "large amount of dilute urine" documented at N-3 of the record, suggestive of either diabetes insipidus, or over-hydration; decreased or absent urine output (oliguria) suggests inadequate fluid intake.

Notably, N-3 of the record also documents a "large amount of dilute urine" (polyuria) at 0325 hours, only 20 minutes later. This finding is inconsistent with hematuria, but may suggest diabetes insipidus, or a patient demonstrated hypotonic hyponatremia with maximally dilute urine consistent with IV overload. The color and volume of urine can be reliable indicators of hydration level. The output of a large volume of dilute urine leads to extracellular dehydration. Because of the excretion of abnormally large volumes of dilute urine, you may quickly become "dehydrated". In this case, the patient demonstrated hypotonic hyponatremia with maximally dilute urine consistent with water intoxication. SIADH is water overload and not salt depletion. This leads to excess water elimination as dilute urine.

A common cause of hyponatraemia is hypotonic dehydration and iatrogenic water overload (eg overestimation of the degree of dehydration, inappropriate use of hypotonic solutions for rehydration

and/or too rapid administration of maintenance fluids). Overhydration is potentially much more dangerous than dehydration. Other causes include SIADH, especially in patients with meningitis.

SIADH stands for syndrome of inappropriate anti-diuretic hormone. SIADH is a volume-expanded state.

The abnormal production of this hormone ADH, leads to salt wasting, or hyponatremia. The result is a profound metabolic disturbance which may result in coma and death. It was originally described in people with small-cell carcinoma of the lung, but it can be caused by a number of other conditions, such as sarcoidosis, meningitis, encephalitis, brain abscess, pulmonary disorders, tuberculosis, infection, including Guillain-Barré syndrome. Head injuries and brain tumors are other possible causes. Certain drugs can also cause this condition, ie., phenothiazines, and morphine.

The record at N-2 documents "Foley draining lge amt dilute urine" again at 0425 hours, while N-1 of the record documents "Foley catheter emptied for 1200cc dilute urine" at 0450 hours that is consistent with conditions featuring osmotic diuresis, such as "diabetes insipidus" (water diabetes). It occurs in association with Na+ Disorders, primarily related to Na negligence, due to iatrogenic fluid overload.

The central causative mechanism in this case, involves a hyperglycemia-induced osmotic diuresis and resultant dehydration. Polyuria due to excess fluid intake and glucose-induced osmotic diuresis is common in patients with transient hyperglycemia. The hyperglycemia emanates from a commonly identified diabetogenic stressor, such as infection, which precipitates the onset of the syndrome, which in turn produces pseudohyponatremia commonly associated with hyperglycemia. Another is drug-induced hyperglycemia, which should not be overlooked in this case.

Arlene Berry had been scheduled for an x-ray follow-up in Sudbury on Tuesday May 30th at 2:30

PM. Sadly, she did not live long enough to meet this appointment. Ontario's healthcare system had failed her.

In sarcoidosis, the immune system starts to attack the body's own tissues, forming small lumps called granulomas. Misdiagnosis of pulmonary sarcoidosis may lead to a greater chance of dying, if not a wrongful death. People with the disease typically develop shortness of breath or a dry cough as inflammation cuts down on their lung capacity. Fatigue, weight loss, and myalgias are also frequently part of the initial presentation. Every year people are diagnosed incorrectly by their trusted physicians.

At a first meeting with the coroner held at the OPP Detachment in Kirkland Lake sometime in July of 2001, Dr. Barry A. McLellan, who was the Regional Supervising Coroner for northeastern Ontario at the time, admitted to family that there was "no evidence on record to suggest matastasis", meaning NO evidence of spread of cancer.

At a subsequent meeting, Dr. McLellan provided us with a view of a CT scan that was purportedly done in Sudbury, Ontario at the time Arlene's death on May 24thy of 2000. It definitely shows lesions, but these are nonspecific. The pathogenesis of these yet undetermined lesions remains unclear but serious infection, ie., abscess and/or a metabolic disorder seems the most plausible pathological factors. Appearances of lesions on CT with focal and/or multifocal contrast enhancement are frequently mistaken for brain tumors.

With respect to the initial CT scan hereinbefore mentioned, according to the coroner's expert "in the right occipital region there is a spot that measures less than 1 cm that is consistent in appearance with either a small hemorrhage or perhaps a small metastatic tumor". He could only speculate. Further, a paltry CT scan does NOT provide conclusive proof of metastatic brain tumors. For that it takes a biopsy and in this case that was never done. Therefore the true nature of the lesions were never established.

In my opinion, the solitary lesion is also consistent in appearance with the capsul stage "cerebritis", in the early stage of abscess development. The small lesion would have been assymptomatic. Untreated, the single capsule could easily rupture resulting in the formation of multiple abscesses.

When symptoms of amoebic infection do occur, they usually begin within 4 months after amoebas first enter the body. Symptoms often fluctuate over weeks or even months with the patient becoming debilitated. Once the infection reaches the brain, symptoms of amoebic brain abscess may develop slowly, over a period of 2 weeks, or they may develop suddenly.

Amebic brain abscesses may be single or multiple; multiple due to systemic disease. Complications may include meningitis or encephalitis of varying degrees. Early signs of encephalitis can develop in a few hours, or over a few days and can first appear as "flu-like". For the record, also know that parasite spreading can promote a systemic response that resembles bacterial sepsis. Abscesses have been observed in frontal, parietal, temporal, and occipital lobes and cerebellum in cases of cerebral amebae infection.

EVIDENCE OF ALTERED RECORDS

There are numerous material deficiencies in the related medical record of Arlene Berry which manifest a complete lack of internal consistency, ranging from out of sequence records, from the physician’s discharge note seen at A-1 and A-2, to the nurses Triage, to obviously rewritten, altered, and/or falsified medical records seen between N-1 and N-3 of the Nurses Notes, which are marred by error, inconsistency, omission, and contradiction, with A-16, and A-17 presenting similarly. These records were unsequentially numbered from back to front, and dubiously tailored to obscure and obfuscate the truth, to illustrate:

The record at A-6 documents a "history of metastatic lung cancer", while the outpatient record at OP-54 clearly documents "no metastasis" and "mediastinoscopy negative".

A-1 of the record documents "she had a left lung pneumonectomy back in October of 1999", which is erroneous. A-17 documents the very same error with "removal of left lung in 99", suggestive of having been copied. The bald truth is that Arlene Berry had the lung removed on January 13th of 2000.

A-3 of the record, what I take to be the physician's diagnostic chart is a total blank. From that record it seems clear that nothing was entered because nothing was done. The same record was filed out-of-sequence. The emergency record at A-4 was also filed out-of-sequence. Interestingly both of these records were dated using a "rubber stamp" that is consistent with backdating techniques.

A-26 of the record documents a BP (blood pressure) of 78/70 at 0235 hours, while the record at N-5 documents a BP of 98/70 at the very same time, suggestive of "scribe errors". Question is, who copied who, and/or who altered what? The answer is quite simple in that whatever answer tends to 'lessen or mitigate' the circumstance, wrongdoing, or outcome of an event (being the wrong answer), in the presence of two or more conflicting entries invaribly points to the truth. The correct answer is therefore a BP of 78/70 mm Hg, being a truly narrow pulse pressure. Low blood pressure , also called hypotension, is blood pressure that is low enough that the flow of blood to the organs of the body is inadequate. NORMAL Blood Pressure is 120/80.

A-4 of the record, what I take to be a Trauma Record, barely visible in the physician's notes situated in the lower right hand side of that record, there is an obliterated area suggesting a "white-out", or perhaps an erasure. From that record it seems clear that relevant information was deliberately removed to conceal a traumatic event. TRAUMA is defined as any insult to the body, clinical or otherwise.

The Ventillation record contains a self-serving entry, ie. "without adversities" relating to patient's intubation procedure, which took place at approximately 0325 hours on May 24th of 2000, while the record at A-17 documents patient being "suctioned for moderate amounts of coffee ground emesis" at 0330 hours, only five minutes later, which can suggest thoracic injury, or perhaps a severe gastrointestinal problem, or both. That the patient's Heart Rate soared to 174 bpm during the intubation procedure should be borne in mind. I find this to be very significant in terms of iatrogenic injury.

N-4 presents with less than half a page of documentation consistent with deliberate omission, such as having rewritten that record for the express purpose of "withholding" incriminating information, between 0305 and 0320 hours. The initials on N-4 appear as "JM", suggestive of nurse McKrank. The record at N-1 presents similarly with a documented time frame between 0450 and 0640 hours on May 24th of 2000. The signature on N-1 appears to be that of nurse Janice Chamaillard. A-10, what I take to be Progress Note presents with a documented "Femalr patient admitted tp 413' via w/c - IV infusing" and no further entries, signed by nurse S. Ferguson.

A-16 documents a blood pressure of 163/117 at 0330 hours while N-3 documents a blood pressure of 136/85 at the very same time. The same record documents a blood pressure of 121/81 at 0400 hours, while N-2 documents a blood pressure of 112/57 at the very same time.

A-26 of the record documents a BP (blood pressure) of 78/70 at 0235 hours, while N-5 documents a BP of 98/70 at the very same time, suggestive of copious error.

N-10 of the Nurses' Notes document the patient's level of care as "routine", which shows little or NO concern for patient safety. Further, NO close patient monitoring or toxicological screening was ever done or even suggested, marked by a complete absence of nursing care plan, as further evidenced

at A-21 of the record. It is clear that no course of action was charted, marked by a "routine" admission and a clinically evident inability on the part of the ED physician to adequately make a proper evaluation or to even make an appropriate or provisional diagnosis.

N-4 of the record documents that Dr. Jordan was called in at 0225 hours; the same record documents the time of his arrival at 0305 hours, while A-1 of the record documents Dr. Jordan's "I was called in later that night because the patient had become obtunded", while N-2 documents "attempts to pull away to painful stimuli" at 0400 hours (suggests a deep sleep-like state), being one hour and thirty-five minutes after Dr. Jordan claims he was called in, presumably, because the patient had become "obtunded" (unresponsive). Further, in coma, a person is highly unlikely be roused to painful stimulation.

N-10 of the record with respect to the patient's bowel routine for toileting is a complete blank, with the very same information that ought to have been recorded, also omitted at OP-53 of the outpatient record. Furthermore, the remainder of the OP (outpatient) records were omitted altogether.

The record at A-8 and A-9 documents "Medi-Vac team were due to arrive at 0435" hours, while the Ambulance Call sheet documents the time of the call event for "call received at 0620" hours, a significant difference.

There are several late dictations and I can count at least three two page documents, all of them questionable seen at A-1, A-2, A-6, A-7, A-8, and A-9, as evidenced by the times and dates upon which they were dictated and transcribed. One of them was dictated in June of 2000, and transcribed in July of 2000, some two months after the patient s death. The Cardiac Index falsely documents the patient's age at "55 years", suggestive of possible record swapping. Arlene Berry was only 41 at the time of her death.

N-7 documents a "stable" condition. Yet the same record documents a complete withdrawal of life support from a critically ill patient as evidenced by a Nature Code "0" that is consistent with No Care. Although it is clear that Arlene Berry was transferred to Sudbury with ventilator support, and although Drs. Jordan and Spiller were aware of the need for emergency care and life support, after ordering it, they canceled it, using the secretive "no code" endorsement as a pretext for evoking a declaration of death and in fact waited for the patient's death.

Within a few hours following her transfer from the Kirkland and District Hospital to the Sudbury Regional Hospital, Arlene Berry was declared as having met with "brain death criteria" , while under the care of Drs. Stephane Sauve and Andrew Adegbite. Her remains were kept in Sudbury for several days prior to being returned to Kirkland Lake.

There is insufficient evidence to determine the minimally acceptable observation period to ensure that neurologic functions had ceased irreversibly. Most sets of criteria for BD diagnosis demand a body temperature of at least 32.2°C. Because of a high risk of bias and inadequate statistical precision, there is insufficient evidence to determine what if any appropriate tests were done to identify brain death or even if they were accurately done. CNS depressant drug effects were still present at the time of brain death declaration.

Withholding life sustaining treatment from an undiagnosed patient with concurrent hyperglycemia, hypokalemia and electrolyte abnormalities in combination with a severely paralyzed motor function and who is under the influence of sedative hypnotic and tranquilizing agents is of questionable legality. Death results from respiratory paralysis and subsequent asphyxiation. Brain death is what happens when ventilator support is discontinued. Turn off the respirator and in the natural course of affairs the patient dies from lack of oxygen.

To practice euthenasia by withdrawing life support to a critically ill patient is a medical homicide; to

kill or destroy by preventing access of air or oxygen. An act of active euthenasia consists of killing someone; to do acts causing death, or by choosing not to act is also an act which determines the course and the outcome of events. Turning off a respirator is a form of passive euthanasia that is usually practiced by doctors with a family's consent. In this case, no such consent was ever given.

From the record as a whole it seems clear that the healthcare providers failed miserably in their concerted efforts to obfuscate the truth. Not only did they act together, but they acted in concert with malice and forethought and with intent to defraud the estate of the deceased of any lawful claim they may have had against them (the doctors and nurses) for their part in substandard care and civil and criminal wrongdoing. They further escalated their plot into a criminal conspiracy by their own doings.

No autopsy was performed, as I understand it, because the patient died "while under the care of a physician". Unfortunately, autopsy rates are declining for various nefarious reasons and the opportunity to measure misdiagnosis in this way is purposely reduced, leaving the door wide open to medical homicide. No appropriate period of observation and/or trial of therapy was ever undertaken. In fact, Arlene Berry was rushed to her death within five and one-half hours of her departure from the Kirkland and District Hospital to Sudbury Regional Hospital, some 220 miles away on May 24th of 2000. A family request for a formal inquest into this death was was also denied.

Are you aware that not properly diagnosing a disorder is one of the most universal kinds of medical negligence? It usually begins with iatrogenic neglect, or failing to take a proper medical history, lack of diagnostic thoroughness, incompetence, or gross medical neglince. Patients with a diagnosis of primary or metastatic brain tumor(s) associated with a CNS event should have a meticulous review of their history for other possible causes, especially iatrogenic causes.

xxxxx

Notably, fulminant Guillain-Barré Syndrome mimicking cerebral death in which the patient's pupils were dilated to 5 mm and not reactive to light is reported in PubMed. The patient appeared brain dead, showing absent brainstem reflexes, respiratory failure, and fully dilated and fixed pupils. The diagnosis of the Guillain Barré syndrome usually depends on findings such as "rapid development of muscle paralysis, areflexia, absence of fever, and a likely inciting event".

Although many conditions can "mimic brain death clinically upon examination", without excluding them you will KILL a person by homicide, or criminal negligence, despite the reversibility of brain damage.

Determine the facts. The record speaks for itself. To think critically, you must be objective and that means differentiating between what you assume, or what you think you know, and what is factual.

This Site Is Dedicated To Malpractice Prevention

(Mistreatment of a patient through ignorance, carelessness, neglect, or criminal intent)

Engage  **Contribute Spread the Word**

Malcom Everett

In Memory of Arlene H. Berry 1958-2000

Sowing The Seed For A Safer Medical Future

Education is what survives when what has been learned has been forgotten". B. F. Skinner