Redress Information & Analysis

An Enigmatic Experience Project 2000-2015

The Arlene Berry Story | Update 2015

A Shocking Portrait of Ontario's Health Care System

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**.

Over the past 15 years I have spent thousands of man hours researching this unnecessary death. Although this has been an enigmatic experience for me, I am even more convinced now than ever that Arlene Berry's death is clearly the result of gross medical negligence and in acting with wanton and reckless disregard for human life, being the hallmark of criminal negligence causing death. Determine the facts. Circumstance and speculation cannot trump fact. The record speaks for itself and is highly suggestive.

A Brief History

Arlene Berry was a robust young lady of only **41** years of age — wherever she went she was like a breath of fresh air to all who knew her. 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 fisher-girl, hiker, camper, and mother of two (a boy and a girl). She had a heart of gold, always placing the needs of her children before her own. 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, Arlene Berry still cared for her son, the youngest of the two, until her death on May 24th 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 **seasonal reforestation** or **silviculture** activities as directed, such as **"tree planting"** jobs between Matachewan, and Kirkland Lake, Ontario. 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 **she loved fishing**, **hunting**, **camping**, **hiking** and **cooking outdoors over a campfire**. A **newly found hobby had included gathering driftwood**, **pine cones**, **lichen,** and **spagnum mosses**, as well as **various ground pines, 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 **to help make ends meet**. She even did a little **rockhounding**, **wherever there were interesting rocks and minerals** to be collected. **She loved the great outdoors**.

The Facts & The Findings

(Progress Notes)

This 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 to late 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 at the west edge of the campground.

One day, while fishing along the trout stream on the southwest side of the highway, opposite the campground, **Arlene Berry had a bad fall in rugged terrain**, **landing on an old fallen tree stump**. The end result, **a left sided rib fracture**(s). It took her awhile to catch her breath, but she was rugged, never wanting to make a big deal of anything.

Within a day or so of her injury, Arlene asserted **"it feels like air leaking inside of me"**, suggestive of **pneumothorax**, or **atelectasis**. Pneumothorax occurs when **air leaks** into the space **between the lung and the chest wall**.

Atelectasis is defined as diminished volume affecting all or part of a lung. Since both conditions involve a collapsed lung, symptoms are similar and can range from mild and barely noticeable to severe or chronic. When a small portion of the lung weakens, it collapses. If this area is small, it does not always affect the functioning of lungs.

In this instance, the injury seemingly resolved just as quickly as it appeared with nothing more than a good night's rest and a daily regimen of 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.

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. A fractured rib could easily puncture the lung resulting in partial or complete collapse of the lung. 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.

The term atelectasis is derived from the Greek words *ateles* and *ektasis,* which mean incomplete expansion. Lung atelectasis occurs as a result of scarring (fibrosis) that reduces lung expansion. Atelectasis is caused by a blockage of the air passages (bronchus or bronchioles) or by pressure on the outside of the lung. Common aetiologies include granulomatous disease, necrotizing pneumonia and fibrosis, with the two most common specific etiologies being pleural effusion and pneumothorax. Further, pneumonia may also develop at any time after atelectasis in the affected lung. In pleural effusion, fluid pushes the impacted lung upwards, which generally results in the blunting of the angle on that side of the body.

Atelectasis is a collapse of lung tissue affecting part or all of one lung. In this case it was the left lung. The condition prevents normal oxygen

absorption to healthy tissues. The most common mechanism is due to sharp bony points arising from a rib fracture penetrating pleura and damaging lung tissue. Several types of atelectasis exist; each has a characteristic radiographic pattern and etiology.

Atelectasis is divided physiologically into obstructive and nonobstructive causes. Notably, in this case, the radiologist reported that there was "an area of consolidation noted in the left lung base posteriorly with blunting of the left costophrenic sulcus, suggestive of a bronchial obstructing lesion".

Causes of obstructive atelectasis include foreign body, tumor, and mucous plugging. Blood clots or scar tissue may also obstruct or block the bronchial airways. A fall, an accident or a severe injury can constrict and compress the lungs. Abnormal healing response due to injury of the lung may result in the production of excess scar tissues that interferes with lung function. Lung scarring can also result from a variety of infections.

Lung injuries leading to scar tissue development can include long-term exposure to toxins, bacterial or fungal growth, as well as viral and parasitic infection. Lung spots are more common than most people think and many are produced by harmless scarring in the lungs caused by respiratory infections in the lungs.

Pulmonary atelectasis is one of the most commonly encountered abnormalities in chest radiographs. Recognizing an abnormality due to atelectasis on chest radiographs can be crucial to understanding the underlying pathology.

Lung scarring occurs due to pathological deposition of fibrous tissue. This is a progressive disease usually and hence needs frequent periodic monitoring. The most common associated infections are chronic lung infections. Many lung infections can simulate cancer, and their differentiation, based on imaging findings, can sometimes lead to a presumptive malignant process. The infections may be fungal, mycobacterial, parasitic or, rarely viral. Most, if not all of these infections can masquerade as a primary or metastatic lung carcinoma.

Some conditions do not attack the lungs directly, but nevertheless due to their effects on tissues throughout the body, lead to scar formation. These include lupus, scleroderma, rheumatoid arthritis, dermatomyositis, polymyositis, Sjogren's syndrome, and sarcoidosis. Symptoms of lung scarring are similar and include a feeling of breathlessness, especially during or after physical activity, a dry cough, fever, chills, wheezing, chest pain, night sweats, weight loss, decreased energy level, and finger tips that become enlarged and rounded. Over time, these symptoms become progressively worse.

Chronic Atelectasis occurs when the patient has been suffering from a collapsed lung and is also dealing with other complications that include difficulty breathing, infection, and scarring of tissue or fibrosis. 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 type of lesion, reportedly "mistaken for adenocarcinoma", the most common type of lung cancer, which usually begins in the mucous-producing cells of the lung. It's also the most common type of lung cancer in women and in "people who have never smoked".

Further, 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). Pulmonary fibrosis can be caused by many conditions, including chronic inflammatory processes. Long term exposure to dust, pollens, environmental agents etc., can also cause this. Pulmonary fibrosis has also been known to "masquerade as metastatic lung cancer".

Adenocarcinoma is usually located on the outer surface of the lungs (periphery) and can also be mimicked by "pulmonary sarcoidosis", occurring usually late in the course of the disease. Sarcoidosis is a rare disease that results from inflammation. It commonly affects the lungs and skin.

Sarcoidosis as signs may **“mimic adenocarcinoma”.**

Every year people are **diagnosed** and **treated incorrectly** by their trusted physicians.

Pulmonary fibrosis occurs when the **lung tissue is damaged**. Fibrosis refers to **scar tissue** that has **replaced healthy tissue**. 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**. For the record, **pulmonary fibrosis** describes a group of diseases which produce **interstitial lung damage "mimicking lung malignancy"**.

Whatsmore, **lung infections mimicking malignancy** are **not uncommon**. A variety of lung **infections** have radiological features **“simulating cancer”,** and their differentiation, based on imaging findings can sometimes lead to a **presumptive** malignant process. Most of these are **chronic infections,** which **do not respond to routine courses of antibiotics**. The infections may be **fungal**, **myco-bacterial**, **parasitic** or, rarely, viral. **Most**, if not all of these **infections** can **masquerade** as a **primary** or **metastatic lung carcinoma**.

Misdiagnosis of **pulmonary sarcoidosis** may lead to a greater chance of dying, if not a **wrongful death**. The **most common reason** that physicians **fail to make a correct diagnosis** is that they **never take time to consider it**. Many **GP's** are, to put it politely, “**technically** and **diagnostically challenged**", **or just plain lazy**. **Mistakes** are made **when they encounter anything other than** the **most common** of **ailments**.

Mistakes are also made by **making assumptions** and **taking shortcuts**. **Labelling** is also one of the most tempting and **potentially hazardous errors** made in the **initial assessment**. Once **labelled,** everything is **fit into that diagnostic box**, anchoring all of your symptoms to that diagnosis, even ones

that don't quite fit. In particular, biases against patients that are or have been long term smokers are common and have been shown to affect a physician's judgment, practice style and level of care. In this instance you're likely to get a diagnosis of lung cancer even if you don't have it.

People with pulmonary sarcoidosis typically develop shortness of breath or a dry cough as inflammation cuts down on their lung capacity. Extreme exhaustion is one of the more common symptoms. Fatigue, weight loss, and myalgias are also frequently part of the initial presentation.

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. 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.

In sarcoidosis, the immune system starts to attack the body's own tissues, forming small lumps called granulomas. A granuloma is a small area of inflammation in tissue. The term "granuloma" is a medical term that is used to refer to a minute collection of immune cells that are known as macrophages. Granulomas are most often the result of an infection and most frequently occur in the lungs, but can occur in other parts of the body as well. They typically cause no signs or symptoms and are found incidentally on a chest X-ray done for some other reason.

Granuloma of the lung is an infection in the lung. The infection can come from a number of sources: a cold, smoking, inhaling fumes, etc. Although it sounds bad it is not cancerous but it is a serious infection. Lung granulomas are clumps of chronically inflamed tissue usually caused by a fungal or bacterial infection. A complete blood count may show a high white blood cell count, indicating the presence of an infection or inflammation. Granulomas are your body's reaction to foreign substance. Granulomas

occur in the lung in a variety of infectious and noninfectious diseases.

Granulomatous processes such as TB, fungal infections, and sarcoidosis can all resemble Nontuberculous mycobacterial granuloma (NTM) infections. Nontuberculous mycobacterial granuloma can enlarge without clinical manifestations or any satellite lesions and cavitations, leading to a misdiagnosis of lung cancer. Nontuberculous mycobacteria (NTM) are environmental organisms that are normally found in soil and water. Several patients have undergone pneumonectomy, usually partial, for presumptive lung cancer that turned out to be an infection.

Intracranial granulomatous masses presenting as space occupying lesions, although rare, have also been described in the literature. Intracranial granulomas presenting as space occupying lesions can cause focal neurology and imaging which may mimic that of tumor. Causes include infections, systemic granulomatous disorders, and iatrogenic from previous surgery, as in this case. Intracranial space occupying lesions are tumors or abscesses present within the cranium or skull. They are one of the three types of lesions that can occur; the other two are vascular (thrombosis, emboli etc) and lesions due to trauma.

Once parasites start an infection, they can effectively resist the lethal effects of macrophages and produce chronic infection that can lead to inflammation. Parasites can induce granulomatous inflammation that serves to insulate the pathogens that resist destruction. These granulomas are regulated by T cells that recognize parasite-released antigens. In the tissues macrophages accumulate and secrete chemicals that induce fibrosis and stimulate the formation of granulomatous tissue and provoke fibrosis.

As granulomas grow, they can compromise the health of an organ, including the lungs. Although non-necrotizing granulomas are the usual finding in sarcoidosis, necrosis can also occur, and is referred to as necrotizing sarcoid granulomatosis. The difference between granulomas and other types of inflammation is that granulomas form in response to antigens that are resistant to the first line of defense in the body. This consists of inflammatory cells such as neutrophils and eosinophils. The antigen causing

the formation of a granuloma is most often an infectious pathogen or a substance foreign to the body, but often the offending antigen is unknown (as in sarcoidosis).

Essentially, a granuloma is just swollen tissue. It is a mass of inflamed granulation tissue from injury or infection, usually associated with ulcerated infections, or invasion by a foreign body. Granulomas form when the immune system attempts to wall off substances that it perceives as foreign but is unable to eliminate.

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 also the pathologic hallmark of sarcoidosis, with the disease sometimes masked by other conditions or disease processes. For example, the granulomas of sarcoidosis are similar to the granulomas of tuberculosis and other granulomatous diseases.

Notably, the granulomas of sarcoidosis are caused by collections of immune system cells, particularly T cells. Other important causes of granulomas are parasitic infections.

Granulomas may also accompany a parasitic lung infection known as pleuropulmonary amoebiasis. Notably, granulomas may also form around parasites.

Know that granulomatous conditions of diverse etiologies share common histologic features. Further, the coexistence of sarcoidosis and "opportunistic infection" has previously been documented. The possible co-existence of sarcoidosis with amoebae is of interest and suggests a potential etiological relationship.

Some granulomas contain necrosis (dead cells that appear as a mass of formless debris with no nuclei present). Some also have "caseation" (literally: turning to cheese) which is a form of necrosis that without a microscope, appears cheese-like (caseous), and is typically a feature of the granulomas of tuberculosis. The identification of necrosis in granulomas is important

because granulomas with necrosis usually have infectious causes.

The CT characteristics of necrotizing granuloma are indistinguishable from those of malignant tumors. Although most cases of sarcoidosis either regress or remain stable, 10-15% progress to pulmonary fibrosis. Generally, pulmonary function worsens with an increasing stage of disease, but radiologic staging does not correlate well with the severity of pulmonary function abnormalities. Often, the radiographic abnormalities appear worse than the degree of functional impairment actually present.

Granulomas are seen in a wide variety of diseases. Infections that are characterized by granulomas include tuberculosis, leprosy, histoplasmosis, cryptococcosis, coccidioidomycosis, blastomycosis and cat scratch disease. Some more common non-infectious granulomatous diseases inculde sarcoidosis, Crohn's disease, , Wegener's granulomatosis, Churg-Strauss syndrome, pulmonary rheumatoid nodules, berylliosis, and aspiration pneumonia.

The diseases associated with granulomas each have a different preferred method of treatment, and because granulomas are so wide-spread the possibilities for treatment are almost limitless. Acute infections should be treated aggressively with antibiotics, and these can also be prescribed prophylactically to prevent infection. If an abscess forms in association with the granuloma it can be treated and drained by a surgeon.

Within 2 to 5 years, about 25% of those with Sarcoidosis will develop residual fibrosis in the lungs or elsewhere, giving rise to "residual disease". You can also get residual scarring associated with fibrosis after a lung infection. Residual fibrosis is scar tissue that is left behind after an infection, or surgery. Residual anything is a leftover. In this case, residual means the fibrosis was left behind as permanent scarring of the lungs, before and after the lung resection.

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, such as

wood smoke, etc. 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, forest products and building materials (through their antigenic or adjuvant properties) are all at a slightly increased risk of contracting sarcoidosis.

Symptoms associated with sarcoidosis can appear suddenly and then just as quickly resolve spontaneously. Sometimes, however they can continue over a lifetime. Symptoms can be related to the specific organ affected, or they can be non-specific general symptoms, including:

- weight loss
- loss of apatite
- fatigue
- fever
- chills and night sweats.

Sarcoidosis may involve one organ system or several. It usually starts in the lungs or lymph nodes in the chest. It is thought that inflammation of the alveoli (tiny sac like air spaces in lungs where carbon dioxide and oxygen are exchanged) is the start of the disease process in the lungs. This may either clear up on its own or lead to granuloma formation and fibrosis (scarring). Over 90% of patients have some type of lung problem. Once considered a rare disease, sarcoidosis is now the most common of the fibrotic lung disorders.

Central Nervous System (CNS) involvement by sarcoidosis (also termed neurosarcoidosis) is relatively common among patients with systemic sarcoidosis and has a bewildering variety of manifestations. Pupillary abnormalities, including internal ophthalmoplegia have also been described in sarcoidosis. Sarcoidosis can also cause a type of meningitis. Cases complicated by fatal meningo-encephalitis have also been reported. An association between neurosarcoidosis and Guillain-Barré polyneuropathy is also reported in the literature.

Diseases of the CNS and PNS are caused by many different types of pathogens, some of which are represented by bacteria, viruses, fungi,

parasites, and toxins. Diseases of the nervous system include meningitis or encephalitis. Neurologic symptoms of CNS infections include headache, encephalopathy, diffuse weakness, including acute flaccid paralysis.

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 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. This is typical of Red Lake as well as much of northern Ontario from east to west during the late fall and winter months.

Past studies have also noted a clustering in parts of the country where there is more logging, lumbering and sawmill activity. In particular, studies suggest that sarcoidosis cases occur twice as often where lumbering and wood milling is a principal or secondary industry. The past sarcoidosis literature should be considered carefully for the possibility that the associations with lumbering, wood milling and wood burning are surrogates for the sensitising antigens they harbour.

Arlene Berry arrived back in Red Lake during the early fall of 1998, where she had been living and working as a housekeeper at the Red Dog Inn the previous winter. The accomodations were provided but the wages were low. At some point she claimed to have pulled a muscle in her back flipping a

mattress at work. She often complained of aching discomfort in her lower back. Her back pain would come and go as it did, and had been confined primarily to the tailbone area.

While living in Red Lake, Arlene Berry had been seeing a Dr. Jinot of the Red Lake Medical Associates for a variety of ailments believed to have been work related, but obviously not considered by her doctor to be urgent enough to warrant serious medical attention. At some point she was forced to quit her job and look for less demanding work due to lower back pain including shortness of breath and soon found employment as a school-crossing guard. She moved out of the motel and into an old mid sized wood heated mobile home that had belonged to some friends. She had also enrolled in a CPR course offered locally about the same time. As I recall, it was between the late fall of 1998 and early winter, at the turn of 1999.

By mid to late February of 1999 Arlene Berry developed breathing problems due to the bitter cold and so it was decided to packed up and move back to Kirkland Lake, Ontario, where she had been living prior to the summer of 1997. Her previous 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. Pulmonary disease is usually considered the most common cause of digital clubbing. Although clubbed fingers are mostly asymptomatic, it often reflects the presence of dreadful internal illness like lung cancer, pulmonary fibrosis (lung scarring), sarcoidosis, or underlying suppurative conditions. Notably, it also occurs secondary to atelectasis, as hereinbefore mentioned.

Evaluating the service provided

On August 6th of 1999, Arlene. Berry attended the Kirkland and District

Hospital Emergency Department where a chest x-ray was finally ordered by a Dr. Beeston. As the radiology department 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. Jordan failed to get back to her with the results of her x-ray she sought him out at his office at the old hospital situated on Second Street in Kirkland Lake, Ontario, where she was told that Dr. Jordan was probably working the ER at the Kirkland and District Hospital on that particulat day. I know because I accompanied her at that time. She returned to the ED at some point during the fall 1999, thinking perhaps that the result of her x-ray was negative, since she had not heard back from anyone. According to Dr. 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". A question has arisen as to why he did not have a copy of the record? The excuse given by Dr. Jordan was that because the patient "had not attended his office" that he "made no connection" with respect to the request for a follow-up of the radiology report.

According to correspondence pertaining to the x-ray, 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 "unsuccessfully, first by phone, then via mail" asking her to "book an appointment". At that point, Arlene became infuriated 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 berate Dr. Jordan in front of everyone present in the Emergency Department at the Kirkland & District Hospital. According to one 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 and also 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 willful blindness, or iatrogenic neglect. In northeastern Ontario back in 2000 this was readily accomplished through NORTH Network, the so called tele-health 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.

A repeat chest film was finally obtained shortly after the time Dr. Jordan claims 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 a communication received by the CPSO in correspondence received from Dr. Jordan "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. Lung consolidation usually occurs when the lungs are infected and become filled with fluid. Accumulation of pus, edema and even collapse of the lung (atelectasis) may also result in "consolidation". Consolidation may be patchy in distribution and involve only certain lobules of the lung although it can be widespread and affect entire lobes of the lung. It may also be complete or incomplete.

Lung abcess due to infection can present as lung masses or nodules that may be isolated or occur within areas of consolidation. Consolidation of the lung is simply a "solidification" of the lung tissue due to accumulation of solid and liquid material in the air spaces that would have normally been filled by gas. Chest x-rays can reveal areas of opacity (seen as white) which represent consolidation.

A lesion is a broad medical term that might refer to a wound, sore, ulcer, tumor, cyst, or some other type of "tissue damage". A lung lesion is

abnormal tissue found on or in a person's lung. It can be the result of an infection or illness, which may clear up without causing the patient long-term problems. For example, some lung lesions develop because of tuberculosis or pneumonia infections. Others may be non-cancerous cysts or scar tissue.

Blunting of the costophrenic angles pertaining to the ribs and diaphragm is usually caused by a pleural effusion. Costophrenic angle is located on posterior and lateral side of the lower chest wall where diaphragm meets lower rib cage. Pleural effusion is excess fluid that accumulates in the pleural cavity, the fluid-filled space that surrounds the lungs. It is commonly known as "water on the lungs". A Pleural Effusion is often associated with atelectasis (collapse of lung tissue).

The key symptom of a pleural effusion is shortness of breath. Fluid filling the pleural space makes it hard for the lungs to fully expand, causing the patient to take many breaths so as to get enough oxygen — a blockage of the lungs may also lead to pleural effusion. Atelectasis is caused by a blockage of the air passages (bronchus or bronchioles) or by pressure on the outside of the lung. Atelectasis is also often the cause of pleural effusion.

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 CT 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 Alberto Gonzalez De La Rocha a Cardiovascular and Thoracic Surgeon at the Timmins & District Hospital. On December 20th, the patient was again seen at the Timmins & District Hospital for a "repeat bronchoscopy". Bronchoscopy is an examination of the air pasages leading to the lungs.

CAVEAT: 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. Notably, a "false positive" test can occur in the presence

of inflammation or infection. Perhaps the worst part of this issue is that false positive diagnoses are NOT altogether uncommon. In fact, it is estimated that as many as forty percent of all initial tests for cancer could be done in error. For the most part, false positive diagnoses typically occur because an improper method was used to implement a test, or the test was not analyzed properly which falls into the category of medical negligence.

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". Notably, the pulmonary artery is located directly beneath the aorta.

On January 13th of 2000, Arlene Berry was admitted to the Timmins & District Hospital and a left pneumonectomy was performed under the care of Dr. Claudio Alberto De La Rocha. Surgical entry was gained via the back under the shoulder blade. 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 adopted 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 a follow-up consisting of a CT scan, and a mediastinoscopy was done as part of her post-operative evaluation. The mediastinum is the area in the middle of the chest between the lungs. 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, believed to have been procedure related. Gone was her uniquely distinctive natural voice at that time.

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 (the procedure to examine the mediastinum inside of the upper chest between and in front of the lungs) was found to be "NEGATIVE". From that record it seems clear that NO clinically detectable metastasis was found. 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 that time.

Notably, mediastinotomy is another procedure in which the doctor inserts a tube into the chest to view the organs in the mediastinum 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 mediastinotomy or biopsy was actually ever done.

At the time of her discharge from the Timmins & District Hospital, 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 on admission to hospital, detected March 16th of 2000, with a timeline between January 13th and March 16th postoperative. Serological testing for the HIV (AIDS) was obviously done at the same time and also found to be "negative".

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 are relatively harmless and some of them trojan horses of the potentially lethal variety. There are many conditions in which cysts develop, but in most cases, they are not cancerous.

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. Cysts of this type can be spread directly from person to person or indirectly through food or water.

The asymptomatic carrier and patient with mild chronic amebiasis can develop acute symptoms at any time, especially if body resistance becomes lowered by another illness. The symptoms of amebic dysentery usually develop 2 to 4 weeks following exposure to the parasite (April 16, 2000). DOD May 24, 2000). The more important symptoms of amebic dysentery include malaise, generalized weakness, abdominal pain or cramping, diarrhea, fatigue, fever (low-grade) vomiting, bloody stool, and unintentional weight loss.

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 an approximate time-line between March 16th — to onset of flu-like symptoms beginning on or about May 10th of 2000 suggests an iatrogenic etiology. The term iatrogenic means "doctor caused". Careless patient management and poor treatment lead to iatrogenic complications.

Iatrogenic 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 "contaminated bronchoscope into the lung through the mouth", etc., resulting from medical treatment or procedures, and because NO record of it is created in the first place, the unscrupulous physician 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. This would not be unusual for a doctor with a history of "medical homicide", as in this case.

In April 1993, de la Rocha was charged with "second degree murder", 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. He admitted "dosing her with a noxious substance" — potassium chloride — as well as morphine. Killing the patient

is easier than providing good hospital care!

Dr. de la Rocha had a propensity for selective treatment (was known to discriminate against smokers), including a criminal past history of euthanasia. Ironically, de la Rocha has since moved from Timmins to Markham, Ontario where he is now practicing "colonoscopy".

Amoebiasis is the second leading cause of death from parasitic disease worldwide. In North America, 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. Notably, a high incidence of amoebic cyst-passers among food handlers in hospitals is also reported in the medical literature.

In Canada, amoebic infection is mostly encountered in small patches of population that have migrated from endemic areas. Ironically, amoebiasis was removed from national surveillance as of January 2000, about the same time Arlene Berry had her left lung removed. Notably, in Ontario, there have been between 2 and 11 cases of "amebiasis" in Simcoe Muskoka area alone every year since 2000.

Entamoeba histolytica is the pathogen (protozoan parasite) responsible for 'amoebiasis'. *E. histolytica* can also be present on the hands of an infected person. Amoebae spread by forming infective cysts. Entamoeba histolytica often burrows deep into the walls of the intestines, causing infections and abscesses. From the intestinal walls, it can travel to the spleen, liver, and all other essential parts of the body, thereby causing further infection.

E. histolytica cannot live outside the human body. It penetrates into the human gastrointestinal tract and burrows deep into the intestinal walls. It can cause abcesses and ulcers and may infect the bowel. 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.

After infection, it may take from a few days or even up to 2-4 weeks before

developing overt symptoms. However, some people may carry the parasite for several months or longer before they become ill. Thus, due to the slight variations in incubation period, tracing the cause of the illness requires that one knows what he/she ate and drank, the places traveled, and people you have come into contact with, especially immigrants from endemic countries, in the weeks/months before becoming ill.

Without treatment, E. histolytica can travel to other parts of the body such as the liver through the veins of the intestines. Other body parts that are reached by this protoa may also become infected and be decked with abcesses and ulcers. The formation of pus in the liver (liver abcess), lungs (lung abcess), and brain (brain abcess), is a sign of advanced stage of amoebiasis. Amoebiasis is a serious condition that needs immediate treatment. If not treated promptly, it can be highly fatal.

Two common antibiotics used to fight protozoal infection include:

- Metronidazole (Flagyl)
- Ampicillin

Without proper treatment, there are several complications of amoebiasis that may arise and these include liver abcess, lung abcess, and brain abcess. These complications are often characterized by the presence of pus in the liver, lung, and the brain. Other free-living opportunistic protozoa form the main differential diagnosis.

Acanthamoeba Castellani and Balamuthia Mandrellaris also develop cysts in the brain and cause a chronic granulomatous amoebic encephalitis (GAE) in humans, mostly in debilitated, malnourished immunosuppressed individuals. Acanthamoeba may also be detected in the granulomatous lesions of brain tissue as trophozoites and cysts.

Granulomatous amebic encephalitis (GAE) usually develops as a result of hematogenous spread from lesions in the lungs, upper respiratory tract, or skin. The incubation period for *Acanthamoeba* GAE could extend for weeks or months after primary inoculation. Initial screening laboratory studies are nonspecific and often demonstrate peripheral leukocytosis, hyperglycemia,

and **glucosuria** (the excretion of glucose into the urine, most commonly due to **untreated diabetes mellitus**.).

Glucosuria leads to **excessive water loss into the urine with resultant dehydration**, a process called **osmotic diuresis**. Blood cultures and peripheral blood Gram stains will be **negative for bacteria and other microorganisms**.

Nonspecific laboratory findings in peripheral blood may include the following:

o WBC count is elevated with a **neutrophilic** predominance.

o Complete metabolic panel (CMP) may show **abnormalities,** including **hyponatremia** associated with **acquired diabetes insipidus**, and **hyperglycemia**.

Brain abscess is frequently a complication of meningitis. Infection of the **central nervous system** is thought to be **hematogenous**, from sites of **primary infection in the lungs or the skin**. The infection may **mimic space-occupying lesions in CNS**, and the infected patient may present with **hemiparesis**, **aphasia** or seizures. **Rapid deterioration occurs soon after onset.**

Presenting **symptoms of GAE** are **nonspecific** and can **last for months** before becoming clinically significant. Once the **infection** involves the **CNS** (central nervous system), **death** often results **within days to weeks**. The course of the disease is **insidious** and **fatal** in most cases, mainly due to **delayed diagnosis,** GAE is an **'opportunistic' infection**, usually seen in **debilitated, malnourished** individuals.

The term "**granulomatous**" indicates **hemorrhagic necrotizing lesions** or **brain abscess** (detected by neuroimaging scans) with **severe meningeal irritation**. These **amoebas** cause a **subacute or chronic granulomatous encephalitis**. Brain **abscesses** expand over time, **placing the surrounding brain at risk**. If left **untreated,** the increasing **size** of the **abscess(s)** will **cause death**. **Rupture of an amoebic brain abscess** can lead to **shock** and **death**.

Acanthamoeba and *Balamuthia* have **two** stages, **cysts** and **trophozoites,**

in their life cycle. No flagellated stage exists as part of the life cycle. The trophozoites replicate by mitosis (nuclear membrane does not remain intact) The **trophozoites** are the **infective forms** and are believed to gain entry into the body through the **lower respiratory tract, ulcerated** or **broken skin** and invade the **CNS** (central nervous system) by **hematogenous** dissemination . *Acanthamoeba spp.* and *Balamuthia mandrillaris* **cysts and trophozoites** are found in **tissue**.

Acanthamoeba sets in with **insidious**, focal **neurologic changes** that **mimic** the clinical picture of **single or multiple space-occupying brain lesions**. Focal neurologic changes, **hemiparesis, drowsiness**, **personality changes,** and **seizures** are **common early symptoms**. **Headache** sets in early and is also **insidious**. **Nausea and vomiting** may also be early symptoms. **Fever is sporadic** and generally **low-grade**. Signs and symptoms of **brain parenchymal inflammation** develop, such as **altered mental status**, **diplopia**, **paresis**, **lethargy**, and **cerebellar ataxia**. The disease progresses **over a period of one to several weeks** and usually **ends in coma and death**.

An **altered mental state** can be **infectious, inflammatory, ischemic, traumatic, and metabolic disorders, as well as poisoning, adverse effects and dehydration**, all of which can **affect sensorium** causing anything from minor **cognitive deficits** to **agitation**, **lethargy**, **confusion**, **seizures**, **paralysis**, and **coma**.

Amoebic cysts are typically **"dormant"** and/or resistant stages in the full life cycle. **Cyst formation** is **triggered by dehydration** of **gut contents** in asymptomatic carriers. Some **cysts can remain viable for up to 4 months** or longer and here's the kicker: **"parasitic cysts of any origin may mimic primary or metastatic brain tumors"**.

If the parasite reaches the bloodstream it can spread through the body, most frequently ending up in the **liver** where it causes **amoebic liver abscesses**. **Liver abscesses can also occur without previous development of amoebic dysentery**.

As mentioned, exposure occurs through the **respiratory tract or skin lesion** with **hematogenous** spread through the **central nervous system** (CNS). Finally, **amoebae cross the blood-brain barrier** and enter into the **CNS** to produce disease. In the **CNS,** the **onset is insidious** — the infection may **mimic space-occupying lesions,** and the patient may present with **hemiparesis**, **aphasia** or **seizures**. In such instances, **head CT** and **MRI** may reveal **ring-enhancing lesions** (abscesses) **suggestive of brain tumors**.

Cutaneous amoebiasis can also occur in **skin** around sites of **surgical wound,** or **other site of infection; ashes**, **eczema-like conditions**, and even **serious eruptions** can occur. Cutaneous amebiasis is caused by infection with **pathogenic forms** of **Entamoeba histolytica**. Notably, **cutaneous and respiratory infections** can last for months but the involvement of the **CNS** can result in **fatal** consequences **within days or weeks**.

Meningitis is an **inflammation of the meninges,** the layer of tissue that **surrounds the brain and the spinal cord**. **The three types of meningitis most commonly heard of are bacterial, viral, and fungal**. You can also get **parasitic meningitis** and meningitis **from other causes**, such as from chemicals, and **injected medication that has been contaminated**, to name a few. **Encephalitis** has symptoms **similar to meningitis** but includes the **infection of the brain**.

Amoeba can also cause **disseminated infection** by entering the skin through a cut, **wound**, including a **surgical** wound. **Iatrogenic** transmission is also possible. Once inside the body, the amoebas travel through the **bloodstream** to other parts of the body, especially the **lungs, brain, and spinal cord**. The pressure exerted by a **growing cysts** can cause **paralysis** or **brain damage,** or even **blindness**. Sometimes, **cysts block the flow of cerebrospinal fluid** within the **spaces of the brain called ventricles**, putting pressure on the brain. This disorder is called **hydrocephalus**. The **increased pressure** can cause **headaches, nausea, vomiting,** and **sleepiness**.

Amebiasis is **primarily an infection** of the **colon** but **extraintestinal** (liver, kidney, bladder, skin, lung, **brain**, male or female genitalia) disease can also occur. Under certain circumstances the **colon gets affected by an**

inflammatory process medically known as **ameboma**. This inflammatory formation also known as an **amebic granuloma** causes **large local lesion of the colon** and may easily trigger **bowel obstruction**. Amebomas or **amebic granulomas** are an **unusual sequelae** of **acute amebiasis**.

Amebic granuloma (ameboma), **commonly mistaken for cancer**, can be a complication of the **chronic infection** (>6 weeks). **Pulmonary amoebiasis without liver involvement** occurs sporadically as a result of **haematogenous spread** from a **primary site,** the **colon**. **Cysts** can be seen as **single or multiple** well-defined **homogenous lesions** surrounded by otherwise normal lung parenchyma on a plain chest x-ray.

Granulomas are **tumour-like** masses that **encase** destroyed large or **parasitic eggs**. They develop most often in the colon or rectal walls but can also be found in the **lungs, liver**, **peritoneum**, and **uterus**.

The symptoms of **amoebiasis** are **similar to bacterial dysentery** as well as various forms of food poisoning, although this illness is not caused by a bacteria but rather a **parasite**. The parasite is also a cause of bloody diarrhea. Additional symptoms and signs of amebic dysentery include **abdominal pain**, **weight loss**, **fatigue,** and **dehydration** (which can be particularly harmful). **Parasitic infections** may also manifest as **systemic** disease. 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, where they form **pockets of infection** (abscesses).

Complications

Complications usually develop after the trophozoites enter the blood stream to infect other organs.

1. Ameboma growth into intestinal lumen
    1. Risk of Bowel Obstruction
    2. Risk of Intussusception
2. Toxic Megacolon
3. Pneumatosis coli
4. Abscess formation

1. Lung Abscess
2. Brain abscess
3. Liver Abscess
    1. See signs above
    2. Risk of rupture
    3. Risk factors for complication
        1. Multiple cysts or cysts >10 cm in size
        2. Superior right liver lobe involvement
        3. Left liver lobe involvement
    4. Course
        1. Spontaneous resolution by 6 months in 66%
        2. Persist >1 year in 10%

Amoebic involvement of **brain** is a **rare complication** of amoebiasis. It is **life threatening** but with the advent of newer antibiotics it **can be treated** or managed by **surgical decompression** and **amoebicidal** drugs **if diagnosed early.** The symptoms of **amoebic brain abscess resemble those of brain tumour.** Amoebic **cerebral abscesses** may be **multiple**, and **varied** in size. Clinical **symptoms of cerebral amoebiasis** are usually **preceded** by **gastrointestinal** or **hepatic** or **respiratory** symptoms. Notably, **multiple brain abscesses may not cause focal deficit** to suggest their presence.

Notably, **Dr. Claudio De La Rocha** immigrated to Canada from **Mexico**, where he graduated from the **National Autonomous University of Mexico**. Notably, **Mexico** is a **hotbed** for **amoebiasis** and is a source of **infectious cysts**. The term **amoebiasis** (when unqualified) generally refers to **E. histolytica** infection, which is common in **Mexico**. In Mexico about **50%** of the **population** is considered to have **harbored** the disease at one time or other.

Multiple abscesses are 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.**

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.

What makes amoebiasis so dangerous is that it can rapidly become invasive and pathogenic when the body becomes stressed from either physical or psychological problems. Amebic liver abscess is the most common complication of invasive amebiasis, but may affect the lung, heart, brain, urinary tract and skin.

Fulminant amoebiasis occurs in two major forms: intestinal amoebiasis and extraintestinal amoebiasis. This condition can lead to several discomforts and symptoms that affect the intestines such as diarrhea and amoebic dysentery. Extraintestinal amoebiasis, on the other hand, may affect other parts of the body other than the intestines. It can affect the liver, lungs, skin, spleen, and even the brain.

Invasive amoebiasis most often causes an amoebic liver abscess but may also affect the lung. Invasion of the intestinal lining causes amoebic dysentery or amoebic colitis. If the parasite reaches the bloodstream it can spread through the body. When Entamoeba histolytica spreads outside a patient's gut, it usually involves his liver. Here it can cause an "abscess' filled with liquid necrotic liver. To start with this is yellow or yellow-green, later it becomes a thicker dark reddish-brown and has often been described as being "anchovy sauce-like" in appearance that is consistent with the "emesis, reddish brown liquid" documented at N-5.

Factors in the patient's history that are useful in the diagnosis and management of an acute abdomen include changes in bowel habits, weight loss, bloody stool, diarrhea, menses, vomiting, clay-colored stool. An acute intra-abdominal condition of abrupt onset associated with pain may be due to inflammation, perforation, obstruction, infarction, or rupture of abdominal organs that may require emergency surgical intervention.

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**. Fever is noted in only **10%** of patients. 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**.

The onset of **amebic colitis is gradual**, with **symptoms occurring for more than one week**, distinguishing it from bacterial dysentery. Diarrhea is the most common symptom. Patients with **amebic colitis** typically present with **cramping abdominal pain**, watery or **bloody stool**, and **weight loss**. **Perforation of an amoebic ulcer** occurs in **fulminating** cases of **amoebic colitis**. Unlike duodenal ulcer perforation, but like typhoid and tuberculous ulcer perforation, **amoebic perforations are silent and painless.**

In severe cases of amoebic infection, patients experience **frequent episodes** of **bloody stools** and they may become **anorectic** and **nauseated; fecal blood** or **leukocytes** (confirming colitis) **are detectable in the stool.** In **severe** attacks, patients may **vomit** and experience symptoms of **anemia** such as **breathlessness**, and **fatigue**. **Fatiue** can also result **due to the loss of a lot of liquid** in the body. **Weight loss**, **fever** (usually low-grade), **fast heartbeat**, **dizziness**, and **severe cramping** or "**severe stomach pain"** can also occur with severe cases of the disease. **Constitutional symptoms**, such as **low-grade fever, fatigue, malaise, or anorexia**, are **nonspecific** and can be **mild or severe**. As a rule, **fever occurs only in about 1/3** of patients.

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**. If the amoebiasis is particularly severe, the patient may have **ACUTE TOXIC DILATATION OF THE COLON** (a similar condition is seen in **ulcerative colitis**). Early **recognition** and **initiation** of medical **therapy** including **treatment** are **vital to preventing catastrophic** outcomes.

In this case the detected **"cyst"** would have been **dormant** at the time it was discovered; in other words a very **serious infection waiting to happen**. A Cyst may lie **dormant** for a very long period without creating any problems. Once

the cyst becomes infected, it could lead to abscess. This was NOT cancer and Dr. De La Rocha not only identified finding a "cyst" with which he himself was familiar, transmitted to this patient by himself (directly or indirectly) he knowingly sought to pass it off to his patient as terminal cancer when it was NOT, knowing that the cyst, once unleashed, was going to be the death of her and so staged her as "T4 Nl with residual disease on the aorta".

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 Arlene Berry stayed at the Daffodil Terrace Lodge where, between appointments, she spent her leisure time fishing off the docks along the shore of Ramsey Lake. 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 discharged home to Kirkland Lake on or about the last day of April, 2000. The treatment had 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.

Within two weeks or so following radiation therapy Arlene Berry developed "flu-like" symptoms suggestive of gastrointestinal illness. It began as an upset stomach with nausea, accompanied by night sweats. She thought she had the flu due to an associated achy feeling. 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.

Further, she 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 appetite and developed a serious aversion to food. Then began the abdominal pain (upper and lower) alternating with bouts of diarrhea and constipation progressing to bloody stools, including at least one abnormally large hard painful bowel movement that was accompanied by headache, vomiting, night sweats, and general feelings of malaise.

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, facial weakness marked by a crooked smile, slurred speech, and drowsiness progressing to extreme fatigue. Her headaches became more frequent and more severe.

For the record, parasitic invasion is often mistaken for vague digestive problems, including the "flu", stomach aches, nausea, unexplained vomiting, etc. The appearance of symptoms, such as stomache pain with signs of tenesmus, low grade fever, tachycardia, hypertension, nausea, and anorexia are all suggestive of severe forms of dysentery.

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. Systemic manifestations such as nausea, headache, low grade fever and anorexia are often present. A very severe infection like this can be fatal within 24 hours.

There are two basic types of amebiasis (intestinal and extraintestinal) which may exist simultaneously. Intestinal amebiasis, also known as amoebic dysentery is an infectious disease that is characterized by inflammation of the intestine and colon. Amoebic colitis (gastrointestinal amoebiasis), is bowel infection caused by harmful parasites called Entamoeba histolutica. Amoebic colitis results when an individual has had amoebic dysentery and ulcers have developed and are infected by the parasite. Amoebic colitis is often mislabeled as ulcerative colitis.

Extra-intestinal amoebiasis is the result of dissemination in the bloodstream

which produces liver abscesses, and occasionally abscesses of the lung and brain. Pus from an amebic abscess basically is classically thick and chocolate-brown or redish-brown in color which apparently results from blood, which is more likely to enter the abscess cavity after the initial aspiration. The pus usually is thick and glutinous, but it can be thin. The features most suggestive of an amebic abscess are bacteriologically sterile, thick, relatively odorless pus regardless of its color.

An amoebic abscess of the liver will contain necrotic liver tissue at its centre. Upon aspiration this often has a dark brownish red colour called "anchovy" or "chocolate" pus, but the pus may also be yellow, grey or greenish. The pus has no offensive odour, unlike most bacterial (anaerobic) abscesses, which is an important difference. The wall of the abscess contains trophozoites, but the necrotic liver tissue itself does not.

A brown milkshake-like (or anchovy paste-like) material is often aspirated from liver abscesses. The appearance of a purulent fluid of a chocolate-like appearance following the puncture of an abscess, discharge, or through vomiting, are highly suggestive signs. The diagnosis of a hepatic abscess may be suspected from clinical findings. Leukocytosis will be high. Those with intestinal amoebiasis may likely develop a condition known as invasive amoebiasis. It can develop during the acute attack or 1–3 months later, which in this case would coincide with the left lung pneumonectomy. Further damage at the site of invasion is caused by the presence of neutrophils that comes in as a response to the invasion.

When detected in time, abscesses are usually treatable and often can be cured with a course of amoebicides, or a combination of antibiotics and a surgical procedure to drain the abscess(s). However, left untreated a liver or lung abscess can *burst* and spread the infection, leading to sepsis, a life-threatening blood infection.

Sepsis and septic shock can result from an infection anywhere in the body, such as pneumonia, urinary tract infections, and viral infections like the flu. Bacteria, fungi and various protozoa may also be responsible.

Sepsis is an illness in which the body has a **severe response to infection**. This response may be called **systemic inflammatory response**. **Cerebral ischemia is a reality in sepsis.**

Amoebiasis is both **infectious and transmissible** by **direct or indirect** contact and sooner or later follows the progress of a potentially **lethal** opportunistic **infection requiring emergent management** that, **unless diagnosed and treated in a timely manner**, can rapidly "**kill within hours or days**" once symptoms appear.

Notably, all of the signs and symptoms contained herein are common findings in amoebic infection and each and every one of them are present — scattered about on the face of this patient's medical record.

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**". **GI bleeding** is the **most serious source** of **bloody stools.** **There is nothing on record to suggest that a stool culture test was ever done.**

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 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**. A belated **test result** evidenced at **OP-55** later **returned a finding** of "**NO Growth**"; the same record documents "**SEPTRA DS GIVEN BEFORE & CIPRO GIVEN AFTER**".

Routine bacterial cultures are usually **NEGATIVE** for **pathogens**, showing **"No Growth"**, Although **white blood cells in urine signify inflammation, they do not always signify UTI.** **However,** a **negative** urine test can also suggest the presence of **unusual** bacteria, viruses, or **parasites** causing **symptoms of**

urinary tract infection (UTI). Viruses, fungi, and parasites can all cause UTIs.

UTIs occurs when bacteria or other infectious organisms invade any part of the urinary tract. Infections of the lower tract are of the urethra (urethritis) or the bladder (cystitis). Infections of the upper tract are of the kidney (pyelonephritis) or the ureters (ureteritis). Fever is usually absent during UTI, but some may feel pain in the lower abdomen (above the pubic bone) and often in the lower back as well. The urine is often cloudy, with strong odor, and may contain visible blood in some.

An untreated UTI can also cause sepsis. Sometimes called blood poisoning, sepsis is the body's often deadly response to infection or injury. The term urosepsis is usually used to describe sepsis caused by a UTI. People shouldn't die from a UTI, but if sepsis begins to take over and develops to severe sepsis and then to septic shock this is exactly what can happen.

Although rare, parasites can and do cause urinary tract infections. UTI in association with entamoeba histolytica is reported in the literature. The disease is mostly secondary to amoebic bowel disease, and amebic liver abscess.

It is very unlikely that a GP will ever tell you that the cause of your illness is related to parasites. Further, you are more likely to develop a urinary tract infection when you are "dehydrated". Dehydration can also cause the symptoms of UTI to appear more severe.

The record at OP-54 dated May 22nd of 2000 documents "large blood trace leukocytes". The presence of leukocytes in urine is referred to as pyuria (pus in the urine). White blood cells in urine is not normal. Hence if such a condition is observed it is very important to find out the cause behind it.

In the normal process of the urinary system, kidneys filter blood and prevent the WBCs from passing through the urine. But if at all a urine analysis shows that there are WBCs such as "large blood trace leukocytes", it reflects improper functioning of the urinary system.

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. There can be many complications of urinary tract infections, including dehydration, sepsis, kidney failure, and death. Notably, the onset of menstrual period evidenced at OP-53 is also within the same time frame of illness. Urinary tract infections due to Staphylococcus aureus typically occur secondary to blood-borne infections.

The record at 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 "vomiting ongoing x 2 weeks".

Dehydration is the main concern with most vomiting. If the symptoms continue for days, they are usually considered severe. 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, including body chemical and mineral (electrolyte) disturbances.

Further, a prolonged bout of vomiting alone 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, or not getting enough fluid, causes low blood pressure, weakness, dizziness, fatigue, and nausea. Other symptoms of dehydration include headaches, dry skin, palor, lethargy, mood changes and slow responses, including "incontinant tinged (dark) urine".

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

The Health Management Record seen at A-22 of the record documents the patient's sensory COGNITIVE PERCEPTUAL PATTERN as "sedated" and is evidenced by a ☑ 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, usually under the influence of a

sedative drug. Sedation can cause both hypertension and hypotension. For the record, "electrolyte derangements" can also mimic sedative intoxication.

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.

A-6 documents "headache, vomiting, increasing head pain and some difficulty ambulating due to dizziness", noting also that "she appears pale". A-1 documents the presenting complaint as "Headache, vomiting and hematuria", and "severe stomache pain". For the record, abdominal or stomach pain concurrent with nausea and vomiting points to the "abdomen" as the source of the problem, although it may not be the only problem.

According to the hospital record seen at A-6, Arlene Berry was admitted to the Kirkland and District Hospital at 1845 hours on May 23rd of 2000 by Dr. Spiller for "IV fluid and Gravol". She was given Gravol 50 ml at 1700 hours, in the ER, as evidenced at A-14; followed by 30 ml MS Contin at 2000 hours, evidenced at A-13; followed by 10mg Stemetil given at 2035 hours, as evidenced at A-11. Several hours later, the patient was *unresponsive,* with *fixed and dilated pupils.* According to the Ambuance Call Sheet, the patient was given additional Gravol by paranedics at the time of her discharge.

According to the same record, Arlene 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". Notably, "severe stomache pain" was documented at 1705 hours on May 23rd and is evidenced at A-1 and A-5 of the record. For the record "severe stomache pain" is inconsistent with a "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. Paresis is the condition describing an inefficiency that causes muscle weakness. Paresis is a condition typified by

a weakness of voluntary movement, or partial loss of voluntary movement or by impaired movement.

When used without qualifiers, paresis usually refers to the limbs, but it can also be used to describe the muscles of the eyes (ophthalmoparesis), the stomach (gastroparesis), and also the vocal cords (Vocal cord paresis). Acquired diffuse paresis in an intensive care unit (ICU) can result from critical illness myopathy or polyneuropathy.

A common cause of diffuse weakness is critical illness polyneuropathy, an axonal disorder that occurs with sepsis.

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.

Stomach paralysis, formally called gastroparesis, is a medical condition in which the muscle of the stomach is paralyzed by a disease or condition of either the stomach muscle itself or the nerves controlling the muscle. As a consequence, food and secretions do not empty normally from the stomach, and there is nausea and vomiting. The most common cause of gastro-paresis is diabetes mellitus. For the record, hyperglycemia, even short term, exacerbates gastroparesis. Other causes of gastroparesis can include medications such as narcotics and some phenothiazine derivatives.

Hypotonia (weakness of muscles) and lack of muscle tone is a symptom of an underlying condition rather than a disease entity in itself. Hypotonia is not the same as muscle weakness, although it can be difficult to use the affected muscles. Muscle weakness sometimes develops in association with hypotonia, however, depending on the cause. Hypotonia is not a specific medical disorder, but a potential manifestation of many different diseases and disorders that affect motor nerve control by the brain or muscle strength.

Patients who have brain infections such as meningitis can have hypotonia. Hypotonia indicates that the brain and nerves are not controlling the

muscles. Other diseases such as encephalitis, sepsis, wound botulism, autoimmunity disorders, metabolic disorders, central nervous system dysfunction, including infections, Guillian-Barre syndrome and cerebellar lesions can also cause hypotonia.

Hypotonia in Guillain-Barre syndrome for example, 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". This syndrome is activated by an infection, and results in the progressive weakness of the limbs, and eventual partial or complete paralysis by reason that the brain and nerves are no longer controlling the muscles. Delayed diagnosis invites catastrophic consequences.

Hypotonia has several well-defined symptoms, the most prominent among them being "reduced muscle definition". Muscle tone and movement involve the brain, spinal cord, nerves, and muscles. Hypotonia may be a sign of a problem anywhere along the pathway that controls muscle movement. Diseases such as encephalitis, sepsis, meningitis, poisons or toxins and botulism can also cause hypotonia.

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. The classic meningitis triad of fever, headache, and nuchal rigidity develops over hours or days. However, there are different types of meningitis and they don't always present the same way; although fever is almost always present, there have been instances of meningitis without fever. You can start off with a high fever and by the time you get meningitis your temperature may be "low grade". In many cases, symptoms have a biphasic pattern; the nonspecific flu-like symptoms and low-grade fever may sometimes precede neurologic symptoms.

Many cases of infectious meningitis begin with a vague prodrome. A common pattern is low-grade fever in the prodromal stage, and may also be

seen in early onset forms of meningtis. People often confuse the early signs and symptoms of meningitis with the flu. Meningitis may come on the heels of a flu-like illness or infection. Infectious causes of meningitis and encephalitis include bacteria, viruses, fungi, and parasites.

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.

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 or 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.

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 may be intense, continuous or stabbing; hepatomegaly (in some cases); intermittent fever and chills; diarrhea (in only one-third of patients); bloody diarrhea (in about 7% of cases); general discomfort (uneasiness, or ill feeling/malaise); loss of appetite; night sweats (early in the course); jaundice (rare, mostly occurs in complicated cases); 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.

Amebic liver abscess is the most common complication of invasive amebiasis. It can develop during the acute attack or 1–3 months later, which varies slightly. Severe levels of enzymes that are frequently elevated when hepatic inflammation occurs are typically within normal limits in cases of E histolytica liver abscess. In 95% of cases, onset occurs within 3-5 months of

contact (fits the time-line between mid January and mid May, 2000).

Pulmonary amoebiasis without liver involvement occurs sporadically as a result of haematogenous spread from a primary site, such as the abdomen, or colon. It can occur from hepatic lesion by haemotagenous spread and also by perforation of pleural cavity and lung. It can cause lung abscess, pulmono-pleural fistula, empyema lung and broncho-pleural fistula. It can also reach brain through blood vessel and cause amoebic brain abscess and amoebic meningoencephalitis. Pulmonary amoebiasis has also been "mistaken for bronchial carcinoma".

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. At the time of this assessment (18:45 HOURS), Arlene Berry was found to be "alert and oriented", with "NO Focal deficits". The same record documents "mild diffuse weakness" and "difficulty ambulating". There is also a documented "pulling to the right" suggestive of hemipareses, what is muscle weakness on only one side of the body — caused by diseases of the nervous system or brain.

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. It may result from polyneuropathy, myopathy, neuro-muscular 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). GBS often follows a minor infection, such as a lung infection or gastrointestinal infection. Most of the time, signs of the infection have disappeared before

the symptoms of GBS begin.

In the Miller Fisher (MF) variant of GBS, the most striking findings on examination are "diffuse weakness" with widespread loss of reflexes. MF 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 absence of reflexes (areflexia). People with this condition can have other signs and symptoms common in Guillain-Barré syndrome.

According to the hospital record at A-8 "This patient was admitted this afternoon with a history of vomiting", and "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 record at A-6, Arlene Berry returned on May 23rd to the emergency department "with the very same complaints". Rapid evolution of illness and patient return within 24-48 hours suggests a severe illness. The same record at A-6 documents "she was given antibiotics and sent home". This is a common ploy used by doctors and hospitals in northeastern Ontario when it comes to evading emergent complaints.

The bald truth is that the Victorian Order of Nurses (VON) had to be contacted in order to get this patient 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.

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 facial weakness, aphasia and facial droop, including 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. Clearly the **etiology** of the **nausea** and **vomiting** had **never been established,** apart from Dr. Spiller's **"a question has arisen with respect to metastatic CA of the brain",** as evidenced at **A-6**.

A **diagnosis involves detailed assessment** and evaluation of a **thorough**, **detailed** and **complete medical history** of the person. Your patient medical history includes **foods** you have eaten in the past few days. It also includes any recent **cuts** or other **wounds**, including **surgical wounds**, medical **procedures**, **etc**., that may have been **exposed** to **viral**, **bacterial**, or **parasitic** pathogens. During the **physical exam**, the healthcare provider will look for signs of **muscle weakness** or **paralysis**, such as **drooping eyelids**. **Blood** and **fecal tests** should also be used. Clearly, with the exception of a noted **"diffuse weakness"** and **a noted "left lung pneumonectomy"**, **there was no detailed assessement**.

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**. **Premature closure** is the **failure to consider other plausible** or **differential diagnoses after an initial working diagnosis is reached**. It is one of **the most common clinical reasoning errors constituting negligence** made by clinicians. This situation may result in an **exacerbation** of the **underlying condition or injury**, causing **further harm**, or even **death**.

NO diagnosis or differential diagnosis was made following the patient's admission at that time, or at all, according to the record. Certainly, **NO protocols were followed** as evidenced by the record. Clearly, from the record as a whole, this patient was **deliberately** made to **deteriorate** w/o so much as a diagnosis of her **stomach pain**.

For the record, a question has also arisen with respect to the sobriety of the ED physician at the time of his assessment of this patient (?)

N-6 documents **family in at 1915 hours**. 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, as evidenced at N-6. It seems clear that generally a cool cloth is provided when a mild or low grade fever is present. The patient's very last words were that she was "very tired", but 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".

The same record seen at N-6 also documents "emesis of ^ 100cc yellowish fluid" at 1915 hours on May 23rd of 2000, what I take to be "bilious emesis" or frank bile. When Red Blood Cells (RBCs) break down in the body they produce yellow pigment which is then passed to the liver and excreted into bile. Billiousness is a symptom of a disordered condition of the liver causing constipation, headache, loss of appetite, and vomiting of bile.

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. When a person is vomiting bile, it is pointing towards the fact that the intestine is blocked, meaning intestinal obstruction or gastroenteritis.

Gastroenteritis, or "stomach flu" is not actually caused by an influenza virus, but by other viruses, as well as many bacteria and parasites. Bacteria, fungi and various protozoa may also be responsible. Parasites can cause problems that often mimic other disorders and are not correctly diagnosed as being parasite related.

Throwing up yellow bile can be caused by a number of different circumstances, including a malfunctioning pyloric valve, a respiratory infection or excessive dehydration. Acute symptoms include bilious vomiting, diffuse abdominal pain, and bloody stools. 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.

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 although it may subside intermittently. When the intestine is blocked, abdominal pain is typically accompanied by frequent bouts of bilious vomiting. Most importantly, the person feels constipated and there is absence of bowel movement. When the bowel stops working, the body gets toxic.

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 obstruction 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, but was never even considered, or ignored altogether.

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 should have raised a red flag suggesting the possibility of intestinal obstruction. Instead, this patient was put on a regimen of opioids and prochlorperazine that were guaranteed to exascerbate her condition.

According to Dr. Spiller, there were "no focal deficits". A focal deficit is a specific area in which normal function isn't present. A-23 documents a "slurred speech" as evidenced by a ☑ in the upper left corner of that document, while A-6 documents "difficulty ambulating". A "slurred speech" (aphasia) can suggest a neurological deficit,; while "difficulty ambulating" suggests a motor deficit. These are examples of focal deficits. A reasonable ED physician ought to know what constitutes a focal deficit, especially when documented on the face of a patient's medical record.

N-6 of the record documents that the patient had stated she was "very tired", whereupon she was assisted to bed. She also complained of being "cold" (she had the chills) and so the nurses provided her with extra blankets. also evidenced at N-6. Periods of feeling cold often occur during illnesses, but in fact the chills can often be a sign of infection that has spread throughout the body.

The symptoms of a brain abscess 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. 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. A suddenly worsening headache, followed by emerging signs of meningism, are often associated with rupture of an abscess.

Cerebral amoebic abscess caused by Entamoeba histolytica infection, is a rare global disease, not related to immunodeficiency that causes proctocolitis with bloody dysentery, liver abscesses and although rare, cerebral abscess through haematogenous spread. Cerebral amebiasis: Headache, nausea, vomiting, and rapid mental status change with rapid progression.

Clinical symptoms of cerebral amoebiasis are usually preceded by gastro-intestinal, hepatic or respiratory symptoms. Amoebiasis should be considered in patients with history of dysenteric-like illness, in patients who have traveled to endemic areas or have recently come into contact with people from developing countries —doctors are no exception.

In severe cases of amoebiasis, "leukocytosis with neutrophilia, and hypokalemia" have all been reported to occur. Notably, all three conditions were also present in this patient. Further, fatigue and tiredness are also prominent features of amoebic infection.

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. Clearly, Dr. Jordan had elected to treat this patient over the telephone, "unseen", while sitting at home watching TV.

Stemetil is a brand name for "prochlorperazine". Prochlorperazine belongs to the group of medications known as antipsychotics, and specifically to the family of antipsychotics called phenothiazines. Most drugs in this category are used as anti-psychotics, commonly referred to as "neuroleptics". Neuroleptic means "nerve seizing", and describes the paralyzing effect these drugs have on the brain and nervous system. Increased sedation is a serious side effect of this type of agent.

Prochlorperazine (Stemetil) has neuromuscular blocking effects.. By blocking neuromuscular transmission, these agents cause paralysis until they are metabolized. Neuromuscular blockade (paralyzes all of a body's voluntary muscles, including the lungs and diaphragm) which may mask distress and result in a "gasping syndrome".

Stemetil should NOT be used where nausea and vomiting are believed to be evidence of intestinal obstruction or brain tumor. The drug is highly plasma protein bound (91-99%) and has a duration of activity from 4 to 6 hours.

Under normal circumstances, a typical single dose of Stemetil for a small woman with low body weight is 5 mg. Arlene Berry was given 10mg, x4 the recommeded dosage, together with other medications.. Notably, Stemetil 10 mg 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. 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 (PNS) includes those nerves that extend into the body and are not

protected by bone.

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. 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".

Stemetil falls in the same class of phenothiazines that have been known to suppress intestinal motility to the point of producing a paralytic ileus. The antiemetic effects of Stemetil (prochlorperazine) may "mask signs of overdosage of toxic drugs or obscure the diagnosis of conditions such as intestinal obstruction".

Stemetil should NOT be used where nausea and vomiting are believed to be evidence of intestinal obstruction. It is also contraindicated to emesis in coma, organic brain disorders, in the presence of circulatory collapse, altered states of consciousness or comatose states, particularly when these are due to intoxication with central depressant drugs. It is contraindicated in severely depressed patients, in the presence of blood dyscrasias, liver disease, renal insufficiency, or in patients with severe cardiovascular disorders or a history of hypersensitivity to phenothiazine derivatives. Blood DYSCRASIA = any abnormal condition of the blood.

Stemetil (prochlorperazine) is a "high-risk" antipsychotic-antiemetic drug to be used with caution, according to manufacturer's directives. Symptoms of overdosage include CNS depression which may vary from simple lethargy to coma. Other possible manifestations include convulsions, autonomic reactions such as hypotension, and ileus.

Stemetil suppresses activity in the trigger zones of the vomiting center by "paralyzing the gastrointestinal tract" which governs the vomiting reflex.

Sudden unexplained deaths in hospitalized patients have occurred using this

type of drug. The contraindications are put in place by the pharma industry for a reason. In this case, all of them were ignored.

In terms of the MS Contin, the biggest risk of any opioid medication use is respiratory depression that can lead to severe hypoventilation, apnea and death. Most opiate overdose deaths occur in people who have just withdrawn or detoxed. Because opiate 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 NO close monitoring or toxicological screening was done.

Acute withdrawal symptoms can be severe if morphine is stopped suddenly after regular use. 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 administration of Stemetil.

Morphine can slow or stop your breathing, especially when you start using this medicine or whenever your dose is changed. Further, sudden opiate withdrawal (quitting cold turkey) leads to a syndrome called "opiate withdrawal syndrome". In high doses, morphine can also induce seizures.

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 a week or more. In addition, the symptoms include agitation, anxiety, depression, muscle aches, pupillary dilatation, and abdominal pain or cramping. Complications include nausea, vomiting and breathing in stomach contents into the lungs. This is called aspiration, and can cause serious infection.

Further, dizziness, drowsiness, lethargy, ataxia, have all been cited with adverse events, including slurred speech, syncope, GI bleeding, constipation, nausea, vomiting, urinary incontinence , and urinary retention. These findings have also been reported in association with opiod toxicity.

Morphine overdose leads to vasodilatation

causing severe hypotension.

Acute overdosage with morphine is manifested by respiratory depression, somnolence progressing to stupor or coma, skeletal muscle flaccidity, cold and clammy skin (cold dry skin if patient is dehydrated), constricted or pinpoint pupils, and, in some cases, pulmonary edema, bradycardia, hypotension, and death.

Symptoms of *MS Contin* overdose may include: Cold skin, flaccid muscles, fluid in the lungs, lowered blood pressure, pinpoint or "dilated" pupils, sleepiness leading to stupor and coma, slowed breathing, slow pulse rate.

Hypoxic brain injury, which is caused by a lack of oxygen to the brain, is an under-reported medical consequence of morphine overdose. These brain injuries can cause coma, seizures and, in worst case scenarios, brain death. The long-term consequences of hypoxia depend on how long the brain is without adequate oxygen supply. Basically, the longer a patient is not breathing, the more potential damage is being done to the brain. In many hospital overdose cases such information is deliberately omitted from the record. Health outcomes depend on the success of damage control measures, the area and extent of brain tissue deprived of oxygen and the speed with which oxygen was restored to the brain.

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.

Opioid withdrawal syndrome may also resemble a severe flu-like illness; symptoms peak at 72 to 96 hours but can last for 14 days or more. Opiate withdrawal can also result in death for unhealthy patients.

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 of 2000 (the evening before her death), in the face of undiagnosed and undifferentiated

conditions associated with "severe abdominal pain", including "headache".

MS Contin is a brand name for Morphine Sulfate. "Contin" is a pharmaceutical industry buzzword for "continuous" release. MS Contin is a medicine used to treat moderate to severe, around-the-clock pain. MS Contin has widespread effects in the central nervous system on smooth muscle and produces respiratory depression by direct action on brainstem respiratory centers. The administration of morphine can only serve to "obscure" the diagnosis or clinical course in patients with acute abdominal conditions. MS Contin overdosage may result in apnea, circulatory collapse, cardiac arrest and breathing problems that can lead to death.

Opioids correctly titrated to provide symptom relief will not cause respiratory depression. In fact, morphine-related toxicity will be evident in sequential development of somnolence, mentall dullness or blunting. The amount of morphine that can cause an overdose or death depends on what a person's body is used to; in this case it was 10mg Statex prior to stopping the drug.

Respiratory system

- Difficulty breathing
- Slow and labored breathing
- Shallow breathing
- No breathing

A morphine overdose can be treated with a medication called naloxone (Narcan®). Naloxone, a medicine (antidote) to reverse the effects of the poison -- multiple doses may be needed. It's usually given intravenously, as this is the quickest way to get the medication into the bloodstream. Naloxone acts almost immediately to counteract the morphine. In some cases, activated charcoal is also given. In this case, since no protocols were followed, no such interventions were implemnted.

Drug interactions and warnings include: avoiding concomitant use of other CNS (central nervous system) depressants including sedatives or hypnotics, general anesthetics, phenothiazines, tranquilizers, and alcohol as these may produce additive depressant effects. The abdominal cramps and pain that are seen as morphine side effects can be especially disturbing when

morphine is used to treat abdominal pain.

Usually opioid induced constipation needs to be treated with a combination of a gentle stimulant laxative like senna and a stool softener like docusate. The additive effects of Morphine Sulfate in combination with stool softeners can only serve to exascerbate motility. In this case, Arlene Berry was given 30 mg Morphine Sulfate after a one and one half week withdrawal from a 10 mg regimen of prescribed 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.

Severe morphine side effects can include bowel problems including toxic megacolon and paralytic ileus. Toxic megacolon is a a potentially lethal life-threatening complication of ulcerative colitis. It causes widening (dilation) of the large intestine within 1 to a few days. Morphine is contraindicated.

The hallmarks of toxic megacolon (toxic colitis) is non-obstructive colonic dilatation larger than 6cm and signs of systemic toxicity.

The diagnostic criteria are as follows:

- Radiographic evidence of colonic dilatation - The classic finding is more than 6cm in the transverse colon (NO Workup DONE)
- Any 3 of the following - Fever (>101.5˚F), tachycardia (>120 beats/min), leukocytosis (>10.5 x 10^3/μL), or anemia due to blood loss in stools
- Any 1 of the following - Dehydration, altered mental status, electrolyte abnormality, or hypotension

The record speaks for itself.

For the record, 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", as in this case. Futhermore, narcotic analgesic drugs can produce, via inhibition of peristalsis, "fluid retention great enough to mask depletion of extracellular fluid and electrolytes".

A possible cause-and-effect relationship has also been posited between granuloma (swollen tissue) 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 (MS Contin).

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.

Put yourself in such a patient's position of total or near total paralysis with most of your senses blunted, unable to move, speak or even open your eyes due to a severely paralyzed motor function - you try relentlessly to free yourself until you become so overwhelmed by exhaustion, fright and panic that you go into shock. That's exactly what happened to Arlene Berry.

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. The major groups of pre-anaesthetic drugs are analgesics, particularly the opioids, such as *MS Contin;* tranquilizers of which the principal drugs in this group are the phenothiazine derivatives such as prochlorperazine, marketed under brand name *Stemetil*, and anticholinergic drugs such as dimenhydrinate, marketed under brand name

Gravol.. For the record, seizures can also occur with overdosage of these drugs.

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".

From this information, it is clear that the patient was sedated into a wide awake pseudocoma resulting in her becoming "unresponsive". Since 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.

Terminal sedation is the practice of inducing a pseudocoma usually by means of a continuous intravenous (IV) 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. Whether 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".

In this case, while analytic reasoning may suggest sarcoidosis, pattern recognition suggests amoebiasis, with a "mixed picture". 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, granulomas 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. Granulomatous disorders are often reported as "mimicking" one another.

The posibility 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". Notably, both sarcoidosis and amoebiasis have been reported as being "mimickers of multiple pulmonary metastasis".

Granulomas in the lungs or elsewhere are not considered malignant growths. Furthermore, many pathologists will mistakenly classify a biopsy of sarcoid tissue as cancer when it is not. Diagnostic errors are the most common types of medical mistakes. Faulty reasoning, low qualification, negligence or incompetence may predispose. Nevertheless, in unclear cases, biopsy or surgery and subsequent pathology analysis can help establish the final precise diagnosis.

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" at the rate of 100 cc/hr, together with the medications, as evidenced at A-15, initialed by nurse Bates, RN. The volume of maintenance fluid for 24 hours is usually 1000 ml Remember, water follows sugar (and salt). If all the sugar is in the blood, not the cells, then the patient is dehydrated.(see Glucose).

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 a dehydrated patient.

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 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 hypo-osmolar 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**.

Hypotonic fluids administered **intravenously** can cause **cell hemolysis (**from **influx of water**) and patient **death**. Other conditions that can cause **hemolysis** include **immune reactions, toxins and poisons.**

To **restrict fluid resuscitation** in an already **dehydrated** patient is outright **stupidity** and constitutes an **act of wanton and reckless disregard for human life**. The **purpose of IV fluids** and **electrolytes** is to help **prevent dehydration** and **shock**, **not** to **induce** it. Further, e**xcessive administration** of **potassium-free dextrose solutions may result in hypokalemia.** Further, **without sufficient levels of water in the brain**, ions get **disrupted** and the **result is brain damage**.

The **intravenous** (IV) **administration** of **Dextrose** and **Sodium Chloride** Injection USP **solutions** can **cause fluid** and/or **solute overloading** resulting in **dilution of serum electrolyte concentrations**, **overhydration**, **congested states** or **pulmonary edema**. The risk of **dilutional states** is inversely **proportional to the electrolyte concentrations** of the solutions. The risk of solute **overload** causing **congested states** with **peripheral** and **pulmonary edema** is directly **proportional to the electrolyte concentrations** of the solutions. **Hypersensitivity/infusion reactions**, including **anaphylaxis**, have been **reported** with **Dextrose** and **Sodium Chloride** Injection USP **solutions** (see Adverse Reactions).

That the patient was heavily **sedated** using a **combination** of **morphine** and **prochlorperazine** which causes **neurolept-analgesia** should be borne in mind. Further, Arlene Berry was given **hypotonic** solution. For the record, **deep sedation** given with **hypotonic solution** causes the **circulatory system** to **collapse** for **lack of fluid**.

Inappropriate intravenous fluid therapy is a **significant cause** of patient **morbidity and mortality** and may result from either **incorrect volume** (too

much or too little) or **incorrect type** of fluid. **Insufficient fluid** administration is readily identified by **signs and symptoms** of **inadequate circulation** and **decreased organ perfusion** (hypoperfusion. Administration of the **wrong type of fluid** results in **derangement of serum sodium concentration**, which, if **severe** enough, leads to **changes in cell volume and function**, and may **result** in **serious neurological injury**.

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** such as **seizures**, or **cerebral edema** caused by **fluid shifts** that can occur if fluid is **infused too rapidly**.

The record at **N-6** documents **"IV infusing well"** at **2330 hours**, suggesting a possible more **rapid IV infusion**, **as opposed to a slow drip**. There are **no further IV related entries,** evidenced by the last entry made at **0200 hours,** seen at **A-15**, with **nothing** 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 **iatrogenic neglect**.

Be aware that **rapid administration of hypotonic IV fluids** can cause **swelling** of the **brain cells** and **ICP** (increased intracranial pressure). **Hypotonic** solutions **should never be given** to patients who are at **risk** for **increased ICP** because of a **potential fluid shift to brain** tissue, which can **cause** or **exacerbate cerebral edema**. Further, **rapid correction of hyponatremia** may result in **brain dehydration**, **cerebral bleeding**, **demyelination**, **neurologic injury**, or **even death**. The **most disastrous complication** of **increased ICP** is **cerebral herniation**.

Seriously ill patients always **require accurate fluid balance monitoring** because **IV fluid also contains the medication(s)**. **Rapid infusion** can also lead to **overdosage**. It's when **too much is given too quickly** that the side effects of an **overdose** appear. The patient becomes **very tired,** such as evidenced at **N-6, stops talking** and eventually **becomes unresponsive,** as evidenced at **N-5.**

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 (difficulty breathing) and other signs of respiratory distress". Several late signs of fluid overload include severe edema (swelling), high blood pressure, decreased hematocrit and hemoglobin, and pulmonary congestion. All of these signs and symptoms form a part of this patient's record.

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. Alternatively, this record was swapped out for a blank page. The same record was also filed out of sequence. A useful maxim to remember is "Not documented means not done". Notably, the same record was dated by using a "rubber stamp", suggestive of backdating.

The record at A-4 was also filed out of sequence. The signature on that record appears to be that of nurse Bates RN, in addition to two other sets of illegible initials.

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 however, by 0130 hours the same record documents "Resps becoming more soaring in nature. No change in pupils & completely unresponsive", only one half hour later.

N-5 also documents the respirations as "deep and soaring" that by 0220 hours became "Gurgly", a sign of constriction suggestive of thoracic trauma (patients are often in shock); followed by "Resps. Deep snoring without constant jaw lift", suggestive of obstructive sleep apnea. 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. Obstructive sleep apnea syndrome is the hallmark of drug-induced sleep. It occurs when something obstructs breathing in the upper airway. Obstructive sleep apnea and analgesia is a potentially dangerous combination — Journal of Clinical Anesthesia, Volume 13, Issue 2, Pages 83-85 D. Cullen.

The same record at N-5 documents a heart rate in the 160's (sinus tackycardia), including a physician documented "assessments unchanged" at 0235 hours, despite the fact that the patient had already gone into respiratory distress at that time, evidenced by "Cheyne-Stokes resps" and "periods of apnea lasting 5-8 seconds". The same record documents "Pupils fixed & dilated".

Although opiates (morphine) 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". Furter, ischemia if severe, can also cause fixed dilated pupils.

Morphine distributes to skeletal muscle, kidneys, liver, intestinal tract, lungs, spleen, and brain. Notably, excessive MS Contin can also trigger heart problems. This increases a person's heart rate which leads their body into a

state of **shock**.

A-26 documents a **blood pressure** of **162/80** bpm with an **SaO2** (arterial oxygen saturation) of **80%** at **0220 hours** followed by a **potentially lethal drop in blood pressure** to **"78/70 bpm"** by **0235 hours**, suggestive of **clinical insult**. **When blood pressure drops**, the **entire brain becomes ischemic** for a brief time. **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** or **injury**. **Hemodynamic instability** has also been defined more broadly as **global** or **regional perfusion** that is **not adequate to support normal organ function** (hypoperfusion). **If the hypoperfusion is prolonged for more than two minutes irreversible brain damage begins to occur.**

A-12 of the medical record documents a blood pressure of **163/117** at **03:20 hours** that by **03:45 hours** had **dropped to 85/58,** and again to **85/52 by 3:52 hours**, over a span of **7 minutes,** as evidenced at **N-2** of the **Nurses' Notes.**

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**.

NORMAL Blood Pressure is 120/80.

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 (**labored breathing**) , 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"**, which is a **myth**. Notaby, the **tongue "blocks the airway"**. **Drug induced swallowing** or **gag reflex abnormalities may predispose.**

Medications such as prochlorperazine that affect the smooth and striated muscles of the esophagus that are involved in swallowing may cause

dysphagia. Dysphagia is the medical term for the symptom of “difficulty in swallowing”.

Further, weakness of the tongue and retropharyngeal muscles causes positional airway obstruction which can occur in “unconscious supine patients”. Gasping respirations also known as agonal respiration is an abnormal pattern of breathing characterized by shallow, slow (3-4 per minute), irregular inspirations followed by irregular pauses. Gasping breaths are also high on the order of panic attacks and general anxiety — feeling trapped. Any difficulty breathing (dyspnea) of sudden onset signifies an “impending medical emergency”.

The use of a plastic oral airway is what one might expect to see when a person is having a seizure. For example, during grand-mal seizures, injuries and accidents may occur, such as tongue biting. For the record, a plastic oral airway does NOT provide needed oxygen, although it may prevent “tongue biting”.

There are several types of generalized seizures. The most common and dramatic, and therefore the most well known, is the generalized convulsion, also called the grand-mal seizure. In this type of seizure, the patient loses consciousness and usually collapses. The loss of consciousness is followed by generalized body stiffening (called the "tonic" phase of the seizure) for 30 to 60 seconds, then by violent jerking (the "clonic" phase) for 30 to 60 seconds, after which the patient goes into a deep sleep (the "postictal" or after-seizure phase). During grand-mal seizures, injuries and accidents may occur, such as tongue biting.

A generalized tonic-clonic seizure, sometimes called a grand mal seizure, is a disturbance in the functioning of both sides of your brain. This disturbance sends out electrical signals to your muscles, nerves, or glands. These signals can make you lose consciousness and have severe muscle contractions. Tonic-clonic seizures get their name from two distinct stages. In the tonic stage of the seizure, you lose consciousness. The clonic stage consists of rapid muscle contractions, sometimes called convulsions. In general, the clonic stage lasts for about two minutes or less, before

subsiding.

What Causes Seizures?

Many conditions can provoke seizures, including:

- Stroke
- Brain tumor or Abscess
- Head injuries
- Electrolyte imbalance
- Very low or high blood sugar
- Medications, such as antipsychotics and some asthma drugs
- Withdrawal from medications, such as narcotics, or alcohol
- Use of cross-reacting drugs
- Cancer
- Brain infections, such as meningitis

Drug-related factors

- Multiple medications: drug interactions
- Use of drugs known to induce seizures
- Factors affecting CNS levels of drugs: lipid solubility, protein binding transport by endogenous systems
- Serum levels of drugs: dose, frequency and route of administration
- Overdosage
- Withdrawal

Patient-related factors

- Patients with CNS disorders without a history of previous seizures: brain tumors, stroke, or encephalopathy
- Breach of blood-brain barrier such as occurs in head injury and meningitis
- Systemic diseases affecting drug metabolism and excretion, particularly those affecting renal and hepatic functions

In my opinion, Arlene Berry appeared to be paralyzed or blunted, with the exception of lower limb/leg contractions suggestive of hyperreflexia, or

generalized tonic-clonic seizure.

Hyperreflexia is defined as overactive or over-responsive reflexes. When the reflexes are *extremely* brisk, they're called hyper-reflexes. Examples of this can include twitching or spastic tendencies, which are indicative of upper motor neuron disease. The most common cause of hyperreflexia (brisk deep tendon reflexes) is spinal cord injury, or pyramidal tract dysfunction. Brisk reflexes can also be caused by many other things, such as medications, or stimulant side effects, anxiety, electrolyte imbalance, serotonin syndrome and severe brain trauma.

A generalized tonic-clonic seizure, sometimes called a grand mal seizure, is a disturbance in the functioning of both sides of your brain. This disturbance sends out electrical signals to your muscles, nerves, or glands. These signals can make you lose consciousness and have severe muscle contractions.

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, suggestive of a downward (from the head down) paralysis, such as seen in the Miller-Fisher variant of the Guillain Barre syndrome.

The inability to lift the head off the pillow by an attempted flexing of the neck is a danger sign that frequently develops simultaneously with phrenic nerve (diaphram) weakness, such as seen in 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, an attempted flexion of the neck causes involuntary fexion of the knees.

In severe cases of meningeal irritation, 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,** which may better explain the **lower limb** leg **contractions/flexing** as described herein as **hyperreflexia.**

CAVEAT:

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**.

A-1 of the record documents **"Plantars upgoing bilaterally".** Submit that the plantar reflex is a hallmark of the **Babinski sign**, a **test** for signs of disease process in the **motor neurons of the pyramidal tract**. Babinski's sign is also a prominent finding in **Bickerstaff's brainstem encephalitis** (BBE), a variant of the **Guillain Barre syndrome** (GBS). BBE and Fisher syndrome form a continuous spectrum.

Upgoing plantar responses are a **hard sign** of **upper motor neuron pathology**. Initial **drowsiness**, **bilateral plantar responses**, and **quadriparesis, is strong clinical evidence** of **central involvement consistent with drugs** or **toxins** that affect the **basal ganglia**, **thalmus** or **brain stem.**

Babinski's reflex is a specific, **involuntary response** to a **particular stimulation** in the body. It causes a person's big toe to move toward the top of the foot while the other toes of the foot fan out **after stroking the sole**. This sign or reflex is normal in young children, though its presence after the age of two is an indication of **damage to the nerve paths** connecting the **spinal cord** and **brain**.

Babinski's sign is a prominent finding in the Miller-Fisher variant of GBS. Both diseases occur suddenly after illness and both affect the nerves. 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 further constitutes evidence of responsiveness.

Plantar response: localises to brainstem if upgoing. A Babinski's reflex that is abnormal may be permanent or temporary and can be caused by: brain tumor, amyotrophic lateral sclerosis or Lou Gehrig's disease, Friedreich's ataxia, trauma to the head, meningitis, hepatic encephalopathy, multiple sclerosis, polio, pernicious anemia, rabies, spinal cord tumor, spinal cord injury, syringomyelia, stroke and tuberculosis.

Illness or infection can cause Babinski's reflex due to stress caused to the body. This happens in some severe cases of meningitis, a viral or bacterial or parasitic infection which causes an inflammation of the membranes that cover the brain and spinal cord and is a sign of a problem in the brain or spinal cord.

N-5 documents what appears to be a "Sudden large queery bloody emesis, reddish brown liquid" at 0255 hours on May 24th of 2000 that is 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". It may also suggest that that liver abscess has ruptured into a bronchus.

A liver abscess is a collection of pus in the liver caused by bacteria, fungi, or parasites. It may occur as a single lesion or as multiple lesions of different sizes. It is commonly caused by an infection with bacteria (germs) or amoeba (the parasite that causes diarrhea). Pain on the right upper part of your abdomen, low-grade fever , chills, night sweats, anorexia, malaise and weight loss are the more common signs and symptoms. You may also have

nausea (upset stomach), and vomiting.

Amoebic liver abscess (ALA) with or without jaundice, and with or without hepatic encephalopathy has been reported in the medical literature. Encephalopathy has also 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 patient's record.

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 medications that suppress the central nervous system.

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 left untreated.

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" of 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 which can rapidly spread to the brain 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. Disorders that mimic hepatic encephalopathy, though not inclusive, include sedative overdose, subdural hamatoma, and meningitis. Further, encephalopathy with perepheral neuropathy may falsely mimic brainstem death.

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.

Mimics of encephalopathy include meningitis, encephalitis, metabolic abnormalities, subdural hamatoma and sedative overdose.

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. Claudio De La Rocha.

Further, Amoebiasis is an infection which eventuates into colitis. The parasite may cause colitis which is manifested with bouts of bloody diarrhea, abdominal pain, nausea and intermittent fever . It denudes the red blood cells. The most common clinical symptoms of amebic infection are amoebic colitis, which has various symptoms. It may result in low GI bleeding and bowel obstruction. The symptoms of gastrointestinal bleeding include black or bloody stool or vomit, dizziness, and cramping. Carefully look for the presence of blood marked by "hematemesis" preceded by "bilious vomiting". This is an indication of chronic or more severe amoebiasis.

Notably, "hematemesis" is documented on record at A-26, that was preceded by bilious vomiting, evidenced by "100cc yellowish fluid", seen at N-6 of the record. Hematemesis may also be associated with melena, which are black, tarry stools, as a result of an upper GI bleed. Hematemesis is treated as a "medical emergency". In this case it was ignored. The most vital distinction is whether there was blood loss sufficient to cause shock; as the damage of the GI tract leads to fluid and blood loss. A patient can die in this phase due to progressive circulatory failure and coma if not treated promptly.

A-19 of the record documents the mechanical charting of the patient's HEMATOLOGY, at 0400 hours on May 24th of 2000, beginning with a WBC (white blood cell) count of 22.4 with a normal reference range of 4.0-11.0. The white blood cells derive their name from the fact that all immune cells separate in a single layer upon centrifugation of blood. This layer is white in

color, hence the name leukocytes.

Leukocytes form the main part of the immune system of the body. The number of leukocytes increase when the body is fighting a disease. The presence of an elevated WBC count is called leukocytosis. Leukocytosis, is a common laboratory finding, most often due to relatively benign conditions (infections or inflammatory processes). Leukocyte recruitment is the hallmark of the inflamatory response. WBC's are the body's primary defense against infection and also reflect the degree of physiologic stress. Pathophysiologic mechanisms of leukocytosis include infection, inflammation, stress, drugs, trauma, anemia, and leukemoid reactions.

Leukocytes include five basic types of cells - neutrophils, eosinophils, basophils, lymphocytes and monocytes. These are broadly grouped into agranulocytes and granulocytes, based on the absence or presence of specific staining granules. Agranulocytes include lymphocytes and monocytes, while granulocytes include neutrophils, eosinophils and basophils. Lymphocytes are further divided into B cells, T cells and natural killer (NK) cells. Monocytes give rise to macrophages, whose main function is to ingest and destroy foreign particles and organisms.

There are two basic N-6ypes 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.

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. WBC's are also elevated with dehydration, and hyperviscosity secondary to dehydration. Because the blood has become more concentrated and thicker, it is more difficult to effectively circulate. With dehydration, blood becomes thicker and sluggish, and therefore, more prone to clotting. Dehydration interrupts blood flow which causes clots, cutting off the supply of oxygen to various parts of the body. A high WBC may also indicate that there is inflammation of the central nervous system as in meningitis. An increase in the WBC count is also a

typical response to noxious stimuli.

White blood cells (WBCs) are categorized into five distinct types: neutrophils, monocytes, lymphocytes, eosinophils and basophils. Each type plays its own role in fighting viral, fungal, bacterial and parasitic infections. The relative frequency of each cause usually relates to the clinical setting. Eosinophils are white blood cells that participate in immunologic and allergic events and are are responsible for fighting against infections or inflammations..

An increase in eosinophil count is called eosinophilia. Several causes are known, with the most common being some form of allergic reaction or parasitic infection. Notably, the gastrointestinal (GI) tract typically has the highest number of eosinophils relative to other organs.

Drug reactions commonly cause an increased eosinophil count in hospitalized patients. Dermatologists frequently find eosinophilia in patients with skin rashes. Eosinophils also secrete chemical mediators that can cause broncho-constriction in asthma and in those having a diminished lung capacity. Pulmonologists often see elevated numbers of eosinophils in conjunction with pulmonary infiltrates and bronchoallergic reactions. Other conditions that can cause a rise in eosinophils include ulcerative colitis, and sarcoidosis.

An abnormal increase in the number of eosinophils in the blood is characteristic of allergic states and various parasitic infections. Further, lung disease and certain medications can cause an increase in the count. Drug interactions or allergic response to a particular drug can also lead to eosinophilia. Interestingly, eosinophils can secrete substances which turn off chemicals that mediate infections, and can destroy cancer cells.

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 neutrophils (polymorphonuclear leukocytes). More precisely, migration of polymorphonuclear leukocytes (PMNL) across epithelial linings is a hallmark of several gastrointestinal (GI) disorders. The

presence of PMNL is also noted in gastroenterocolitis induced by ischemic (having inadequate blood flow) conditions, and by various toxic chemicals or drugs. Further, PMNs (polymorphonuclears) accumulates in brain regions with low blood flow during the early postischemic period.

Leukocytosis, especially neutrophilia, indicates "systemic infection" and is rare in the absence of superinfection (also known as super-bugs) such as bacteria, viruses, parasites or mixed infections which are resistant to antibiotics. Some diseases are caused by the infection of more than one type of organism simultaneously. Notably, amoebiasis is high on the order of having mixed infections.

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, abdominal pain and can also lead to nausea, lack of appetite, weight loss, and anemia.

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 (a severely enlarged bowel that can rupture). Toxic megacolon is the clinical term for an acute toxic colitis with dilatation of the colon. The dilatation can be either total or segmental.

Toxic megacolon occurs as a complication of inflammatory bowel disease, such as ulcerative colitis, Crohn's disease, and infections of the colon. The term "toxic" means that this complication occurs with infection or inflammation and is very dangerous.

A more contemporary term for toxic megacolon is simply toxic colitis, because patients may develop toxicity without megacolon. Thus, toxicity or megacolon can occur exclusively of each other. 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. Though it is important for everyone to drink plenty of water, it is essential that those with amebic colitis remain well-hydrated.

The opposite of leukocytosis is called leukopenia (or leukocytopenia). Leukopenia is defined as a decreased WBC count. It is a blood disease in which the number of circulating white blood cells diminishes to a great extent. Leukopenia is usually caused by a decrease in the granulocyte numbers, particularly the blood neutrophils. That is NOT the case here. When the number of WBCs in your blood increases, such as in this case, this is a sure sign of infection somewhere in your body.

On the week before her death, Arlene had become severely constipated leading to a massive stool to the point of clogging the toilet, suggestive of megacolon. Megacolon is usually characterized and preceded by severe constipation. There is a potential for CNS (central nervous system) toxicity. Further, the use of opioids can lead to a rapid deterioration and colonic perforation.

Further, findings suggest that constipation actually gives rise to a process of self-poisoning. Thus, auto-intoxication is the process whereby the body literally poisons itself by maintaining a cesspool of decaying matter in its colon. During fasting, the concentration of toxins expunged from the body and appearing in the blood can increase ten times above normal concentrations. The released toxins can either exacerbate the symptoms being treated or create their own symptoms.

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. Guillain-Barre and Miller Fisher syndromes in association with ulcerative colitis have been successfully treated with infliximab (is reported in the literature).

The record at A-19 documents a Neutrophil count of 92.0 H with a normal reference range of 47.0-77.0), with an Absolute Neuts of 20.0 H having a normal of 1.3-6.71. Neutrophilia (or neutrophil leukocytosis) is the condition where a person has a high number of neutrophil granulocytes in their blood. Normally, neutrophils account for 50-70% of all leukocytes. About 55 to 75 percent of the total WBC count in the blood is made up of neutrophils. They play a crucial role in fighting infection. Neutrophil accumulation in tissue is the hallmark of inflammation and is associated with a variety of pathological conditions. Inflammatory diseases of the brain include abscess, meningitis or cerebrospinal meningitis, encephalitis, and vasculitis.

The most common and important cause of neutrophilia is infection, and most infections cause neutrophilia. The degree of elevation often indicates the severity of the infection.

When an infection occurs, neutrophils traveling in the blood vessels close to the site of infection are attracted to the site by chemicals released by the microbe as well as by other immune cells. After reaching the site, neutrophils surround and ingest the microbe. The granules present in neutrophils contain several chemicals, mostly enzymes, for destroying ingested microbes.

Neutrophils, are also known as "segs", "PMNs" (polymorphonuclears), or "poly's". PMNs are the primary effector cells in the innate immune response against infection. 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.

Neutrophilia facilitates the "inflammatory response", whereas when neutropenia (the opposite of neutrophiia) is present, the inflammatory

response to such infections is **ineffective**. The end result is an **autoimmune reaction**. A **high neutrophil blood countt** is a sign that something in your body has triggered an **immune response**. The **immune system fails to properly distinguish between self and non-self**, and **attacks part of the body**.

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**.

Polymorphonuclear leukocytes (granulocytes) usually represent the **predominant cell type** in an **inflammatory response** acting as the first line of defence against invading organisms. PMN infiltration intensity as consequence of **Entamoeba histolytica** density in **amebic colitis** is reported in **PubMed**, Surgical Infect., 2: 91-97. Guerrant et al. (1981) studied the interaction between *E. histolytica* and PMN phagocytes. A **higher density** of **PMN infiltration** has been observed in **severe cases**.

Furthermore, **granulocytes** are subdivided into three types called as **eosinophils**, **neutrophils** and **basophils**. Each type plays its own role in fighting viral, fungal, bacterial and parasitic infections.

Eosinophils are a late white blood cell of the polymorphonuclear leukocytes subgroup. Basophilia and eosinophilia may be found with neutrophilic PMN leukocytes. **Parasitic diseases** and **allergic reactions** to medication are among the more **common causes of eosinophilia**. Eosinophils gather wherever there is a **parasite infection** or an **allergic reaction** and then release their **toxins**.

Eosinophils are **white blood cells** that **participate in immunologic** and **allergic events.** More precisely, **eosinophils** belong with **neutrophils** and **basophils**, in the category of **granulocytes**, once called **polynuclear cells"** known as **polymorphonuclear leukocytes**. The relative frequency of each cause usually relates to the clinical setting. For example, **parasitic infections** are often

responsible for **eosinophilia**. **Basophils** are commonly associated with **immediate immune reaction** against **foreign particles in the bloodstream**.

Drug reactions commonly cause an **increased eosinophil count** in hospitalized patients. **Several causes are known, with the most common being some form of allergic reaction or parasitic infection** Other conditions that can cause a **rise in eosinophils** include **ulcerative colitis**, and **sarcoidosis**. Notably, the **gastrointestinal (GI) tract** typically has the **highest number** of **eosinophils** relative to other organs.

An abnormal increase in the number of **eosinophils** in the blood is characteristic of **allergic states** and various **parasitic infections**. They are active against nematodes and other parasites as well as against **protozoa**, such as the **amoebae**. Eosinophils protect the body killing bacteria and parasites, but can **cause problems when they react incorrectly** and cause **allergies and other inflammatory reactions** in the body. Since parasitic infestations **provoke** strong **allergic reactions** in the body, they are associated with high Immunoglobulin G (IgG) numbers.

Dermatologists frequently find eosinophilia in patients with skin rashes. Eosinophils also secrete chemical mediators that can cause **broncho-constriction** in asthma and in those having a **diminished lung capacity**. Pulmonologists often see elevated numbers of eosinophils in conjunction with **pulmonary infiltrates** and **bronchoallergic reactions**.

Interestingly, **eosinophils** can also secrete **substances** which **turn off chemicals that mediate infections,** and can **destroy cancer** cells. Although both the **eosinophils** and **basophils** are an integral part of the **CBCs,** in this case, they were **omitted from the the record altogether (**not counted at all), giving rise to a **false impression** that perhaps there were none.

Neutrophilic leukocytosis (neutrophilia) is also **high on the order of acute bacterial infections**, **especially pyogenic or pus producing infections**. Other causes of an **increased neutrophil** count include **"cerebral abscess"**. Amoebiasis is high on the order of **"mixed infection", including brain abscess**. Brain abscesses are usually **mixed** infection. **Entamoeba histolytica**

and Toxoplasma gondii are two of the commonest protozoa causing abscess in the brain. Brain abscesses can cause brainstem herniation and can rupture into the ventricular system. Increased intracranial pressure, stupor, and papilledema are also signs of brain abscess.

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, SUCH AS IN THIS CASE.

The diagnosis of amoebic brain abscess is considered when the patient develops symptoms of amoebic dysentery. Some of the symptoms of amoebic dysentery resemble symptoms of the flu. Another symptom that becomes noticeable over time is a change in the color of your skin. You may develop a chalky or pale color to your skin that will take over your entire body. A brain abscess can grow very quickly, typically becoming fully formed within about two weeks. The symptoms of a brain abscess may also resemble those of brain tumour.

Other symptoms of amoebic dysentery involve bleeding that is immediately noticeable. The more common instances of bleeding during amoebic dysentery are rectal bleeding, and you may begin bleeding within your intestines, which will show itself in the form of a bloody discharge when you are trying to make a bowel movement or as blood in your stool. Another

You may also experience anemia, which is a condition that prevents the proper production of red blood cells to carry oxygen to the internal organs. This will make you feel tired. Anemia is present when HCT (hematocrit) is below 1.0 x 109/L. The patient is usually noticeably pale.

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). Lymphocytes help protect your body from infection. 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 and destroys healthy

body tissue. A low lymphocyte count makes it hard for your body to fight infections. A low lymphocytes count (lymphocytopenia) results in the inability to remember and recognize invaders and hence fails to distinguish.

Many disorders can decrease the number of lymphocytes in the blood, but AIDS and undernutrition are the most common. Abnormally low lymphocyte counts are also characteristic of prolonged (chronic) illness or immunosuppression, including that caused by HIV infection and drug therapies.

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). Initially, lymphocytopenia has been described in case reports concerning infectious emergencies such as Toxic Shock syndrome.

A-19 of the record documents an Absolute Lymphs (lymphocytes) of 0.4 L with a normal reference range of 1.0-4.5. Your low lymphocytes are what we expect to see in sarcoidosis. An infectious etiology of sarcoidosis has long been suspected. Opportunistic infections can even resemble sarcoidosis. They can also co-exist with sarcoidosis. Notably, an infectious etiology of sarcoidosis has also long been suspected.

Absolute lymphocytopenia can also be used in the prediction of infectious emergency admissions. Drastically reduced numbers of lymphocytes are high on the order of infections with bacteria, viruses, fungi, and parasites. Severe lymphocyte deficiencies can result in uncontrolled infections that can be "fatal". Absolute lymphocytopenia is a predictor of bacteremia. The ratio of neutrophil and lymphocyte counts has even higher value in predicting bacteremia.

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.

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. Th1 diseases are lymphopenic, which means a lack of T-cells. People with T cell deficiencies are particularly susceptible to intracellular pathogens. People who have too few T lymphocytes or too few natural killer cells have problems controlling certain infections, especially parasitic infections.

Lymphocytopenia can also be a side effect of radiation treatment. Notably, iatrogenic lymphocytopenia can be caused by either cytotoxic chemotherapy or radiation therapy or both, marked by a reduction in the absolute number of T cells.

Radiation Therapy (RT) may cause immunosuppression and increase susceptibility to infection. Lymphocytes are the most sensitive to whole body radiation and their count is the first to fall in radiation sickness or injury. The number of lymphocytes declines within the first 12 to 48 hours after exposure. This may be followed over several weeks by a decline in the number of lymphocytes. 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. Low lymphocyte levels can also indicate sepsis

(blood poisoning or toxemia).

Sepsis is a clinical syndrome that complicates severe infection. It is characterized by the cardinal signs of inflammation (vasodilation, leukocyte accumulation). Severe sepsis is associated with organ dysfunction, perfusion abnormalities, and hypotension. Sepsis and septic shock can result from an infection anywhere in the body, such as pneumonia, urinary tract infections, and viral infections like the flu. Indeed, NO septic workup was ever done in this case.

Worthy of mention is radiation necrosis, a process of scarring, inflammation, and swelling which can occur in areas of normal brain after treatment with radiation. The typical appearance of brain radiation injury is similar to that of brain tumors, with a contrast enhancing mass surrounded by edema and mass effect. Although radiation-induced necrosis (RIN) is not a tumor in itself, the lesion progressively enlarges with mass effects and diffuse peritumoral edema in a way that resembles neoplasm. It can occur secondary to any form of radiotherapy modality or regimen. MRI signal changes in radiation necrosis cannot be differentiated from tumor related changes.

A-19 documents a Red Blood Cell (RBC) count of 4.30 with a normal reference range of 3.80 – 5.80, but the HCT (hematocrit) is below the normal level giving the "false impression" that the RBCs are perhaps normal when they are not. 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 having a normal reference of 0.370-0.470, with a reduction suggestive of anemia. The result is fewer red blood cells to carry oxygen.

Erythrocytes, or red blood cells, are the most common type of blood cells. Erythrocytes give the characteristic red color to the blood. Their principle function is to deliver oxygen (O2) to the different parts of the body via blood flow through the circulatory system. Red Blood Cells take up oxygen in the lungs and release it into tissues while squeezing through the body's capillaries.

Anemia is a condition that is characterized by a reduction in the oxygen carrying capacity of the blood. This reduction is caused by inadequate levels of hemoglobin, inadequate numbers of erythrocytes (low hematocrit) or both. Thus, a deficiency of iron and therefore of hemoglobin leads to anemia and poor oxygenation of the body tissues.

Anemia (lack of red blood cells) can cause fatigue, pale skin, weakness, dizziness, headache and more. 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.

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. Even 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. The hematocrit is defined as the percentage of whole blood made up of erythrocytes.

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

A-19 documents an RDW (Red Cell Distribution Width) of 18.4 H. The 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 is known as anisocytosis. It can be seen in various types of anemia and vitamin deficiencies.

Anisocytosis manifests in various symptoms although the characteristic manifestation of anisocytosis is influenced by the oxygen supply in the body which is carried insufficiently (inefficiency in oxygen distribution is influenced by variation in the size of RBCs (red blood cells) of unequal sizes while most of the symptoms are similar to the symptoms of anemia or heart failure); prompt treatment is required.

Breathlessness is usually experienced by patients suffering from anisocytosis even with minimal activities and which may be experienced often. Tiredness and exhaustion is the hallmark of anisocytosis in people suffering from it. Patient easily gets tired and lacks in energy to go about daily activities or even finish a task.

The same record at A-19 documents a Monocyte count of 3.0 with a normal reference range of 2.0–10.0 and an Absolute Mono's of 0.60 (<1.0). Monocytes give rise to macrophages, whose main function is to ingest and destroy foreign particles and organisms. 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. Notably, malignant conditions such as leukemia or lung cancer can lead to increased monocyte levels. For the record 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 disseminated intravascular coagulation (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. normal is < 500. An elevated D- dimer is a blood test that suggests a blood clot. 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. Abnormal D-dimer values are indicators of acute diseases or medical conditions that call for immediate action. In this case, according to the record, NONE WAS TAKEN.

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 amoebae 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. The main cause of thrombocytosis is infection. Thrombocytosis is usually categorised into mild, moderate, severe or extreme. The same record documents a modest increase.

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" (hypoperfusion) and the development of DIC (disseminated intravascular coagulation) 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. A higher-than-normal number of platelets (thrombocytosis) may be due to anemia (too few red blood cells). Further, acute, short-term hyperglycemia may precipitate vascular occlusions by facilitating platelet activation. Common conditions include tissue damage from surgery, infection, some forms of malignancy, asplenia, drugs, and chronic inflammatory disorders. Thrombocytosis may also be caused by inflammation due to an inflammatory bowel condition. Infections can also

cause a high or low platelet count.

Thrombosis occurs more frequently in arterial vessels but also occurs in large veins, potentially resulting in deep vein thrombosis, portal vein thrombosis, or pulmonary embolus. The most important medical complications of thrombocytosis are hemorrhage and thrombotic events. Thrombocytosis is frequently present without the symptoms of hemorrhage and thrombotic events.

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.

The same record at A-19 also documents a "PLT ESTIMATE – MOD INCREASE" confirming an increase in platelet aggregation activity or blood platelets sticking together, indicating that blood thinners may be needed to prevent blood clots. An elevated platelet count may also be due to an infection, an operation or an acute blood loss.

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. The most common location for an arterial thrombus is in the brain. Non-specific symptoms may include headache and paresthesias.

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.

For the record, an infection is also often accompanied by a raised platelet count. On the other hand, a lower-than-normal number of platelets (thrombocytopenia) may be due to cancer, chemotherapy, certain medicines, or leukemia, but that is NOT the case here.

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. For the record, 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 DIC (disseminated intravascular coagulation), most likely due to influence of blood clotting factors secondary to dehydration, or infection or both. Because the blood has become more concentrated and thicker, it is more difficult to effectively circulate. When blood pools it causes overall blood pressure to drop and symptoms to occur.

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

A-20 of the record also documents an arterial 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 levels of pO2 are seen in people who are receiving oxygen treatment through a mask or nasal cannula. Usually that high of a pO2 is seen during 100% bagging as a result of over-oxgenating a patient by bagging, extra breaths before suctioning. Notably, an increased arterial pCO2 (hypercapnea) causes cerebral dilation.

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". Oxygen is vital for life but in its pure form it has harmful effect to humans. 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 is toxic because of its propensity to undergo univalent reduction

leading to the generation of reactive oxygen species. Oxygen toxicity occurs when reactive oxygen species overwhelm the natural antioxidant defense system. As the lung is exposed directly to the highest partial pressure of inspired oxygen, it is the primary target organ for oxygen-induced injury. The clinical syndrome of pulmonary oxygen toxicity is that of tracheo-bronchitis and acute respiratory distress syndrome.

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", and is evidenced at A-18 of the record. The acute toxicity has predominant CNS effects, while chronic toxicity has predominant pulmonary effects.

Oxygen toxicity is actually severe hypoxia that is caused by inhaling oxygen at elevated partial pressures (See: pO2). It is also known as oxygen toxicity syndrome, oxygen intoxication and oxygen poisoning. Oxygen toxicity is associated with number of symptoms. This condition affects different body systems and organs including the central nervous system, lungs and eyes. In severe cases, oxygen toxicity can result in cell damage and even death.

The first and most important method to prevent pulmonary oxygen toxicity is to limit exposure to the lowest possible pO2. If high, patient is likely "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. Inadequate cerebral perfusion can only be avoided by removing vasoconstricting factor(s), improving peripheral blood flow, and reducing metabolic demands on the body.

N-3 of the record at 0320 hours documents "Respirations assisted by ambu-bag @ O2 100 %". The same record documents "Intubated #70 ET tube by Dr. Jordan, bagged @100% O2 sats 95-97%".

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.

The most common complications associated with high glucose levels are dehydration and electrolyte imbalances. High levels of glucose will cause a shift in sodium and potassium, which can lead to low blood pressure, renal failure and abnormal heart beats in addition to nerve and muscle dysfunction, drowsiness and difficulty waking up.

Further, sugar solution in IV creates gaps in the blood-brain barrier (BBB) allowing chemicals to enter. Infected material can block the blood vessels to the brain, and drugs that cross the BBB can help shuttle it directly into the brain and CNS. Once across the blood-brain barrier, the infection enters neural cells, with resultant disruption in cell functioning, perivascular congestion, hemorrhage, and inflammatory response diffusely affecting gray matter disproportionately to white matter.

With infection, blood sugar levels tend to rise quicklyl over several hours. Hyperglycemia affects the immune system -- the system that defends your body against infections. An active blood infection, known as sepsis is often seen with blood sugar levels that are high. Certain drugs can affect blood sugar levels. It should also be borne in mind that Morphine and phenothiazine derivatives, such as Stemetil, actually contribute to the occurance of high blood sugar levels (hyperglycemia). In fact, both have been reported to "trigger diabetes" in patients with no previous history of diabetes.

Infection with hyperglycemia is a complex process that involves a cascade of symptoms and systems. Further, a "diabetes-amoeba" association has been reported in the literature. Retrospective studies show that diabetics are at increased risk of suffering severe complications following amoebic infection. Further, plasma glucose >120 mg/dl in the absence of diabetes = clinical sign of sepsis. Many different microbes can cause sepsis. Infections that develop after surgergical procedures can also lead to sepsis.

A-18 of the medical record documents 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. This is called '**ischemic** (having inadequate blood flow) **hypoxia**'. This can include an **embolic event**, a **heart attack** that **decreases overall blood flow,** or **trauma** to a **tissue** that results in damage. **Reduction in blood flow** (relative ischemia) **impairs O2** delivery and causes **cerebral hypoxia**.

The same record at **A-18** documents a **"Sinus Tachycardia"**. It occurs when the sinus rhythm is **faster than 100 beats per minute** (bpm) and is 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** (doctor caused) **harm**, ie **right bundle branch block** (RBBB). The right bundle branch receives **most of its blood supply from septal branches of the left anterior descending coronary artery**, particularly in its initial course.

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 **Creatine Kinase** is a parameter **used to help determine health status**. In females, **total CK** should be **10-79 units per liter** (U/L), **not 30-135**. a**s suggested by the reference** range **set by the physician who performed this test**.

Serum CK is considered a useful diagnostic marker or indicator of active metastases. In this case, **CK is well within the normal range**. 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 patient's assessment (1720 hours), would argue favorably **against** a **diagnosis of metastatic CA**.

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**.

Notably, metastatic CA of the brain is NOT one of them.

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.

The levels of serum CK are different in various myopathies according to the type of disease and the stage of pathology. Notably, drug-induced myopathy may show only slight, or no increase in CK activity.

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".

There are no specific biochemical abnormalities in granulomatous myopathy. Granulomatous myopathy, in the presence or absence of sarcoidosis is commonly associated with dysphagia (87%) and a normal serum CK [PubMED].

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 as anything 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, constipation, hypotension including an array of different symptoms; muscle weakness is the most predominant. Hypokalemia may

also cause slow movement of the colon.

Hypokalemia can cause prolonged neuromuscular block which causes periodic paralysis. Hypokalaemic periodic paralysis is characterized by attacks of muscle weakness. Residual neuromuscular blockade also results in paralysis which may be perceived as unresponsiveness though the patient may be fully conscious and aware. This may occur secondary to overdose, or virtually any medication capable of causing neuromuscular blockade.

In severe cases of amoebiasis, "leukocytosis with neutrophilia, and hypokalemia" have all been reported to occur. Notably, all three were also present in this patient. For the record, both leukocytosis (WBC) and neutrophilia (Neutrophils) are evidenced at A-19, and Hypokalemia (Serum Potassium level of 3.4 L) count seen at A-20.

A-20 documents an Arterial pH of 7.37. The ideal pH for blood is 7.4. A normal pH is 7.35 – 7.45. 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. Hydrogen ion concentration expressed as pH "Power of Hydrogen" (Humans as organisms) scale of acidity/alkalinity. pH below 7 = acidic, and pH above 7 = alkaline.

The vast majority of terminal cancer patients possess a very low body pH. In fact, terminal cancer patients are around 1000 times more acidic than normal healthy people. In the case of Arlene Berry, her pH was optimal — as good as it gets.

Neutral pH is 7, 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.

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**. Notably, 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**. Thus, **tumors tend to exhibit a more acid pH.**

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",** suggestive of **unconscious proprioception,** or possibly the result of a **convulsion**, **seizure**, or **stroke**.

A **seizure is the physical findings** or **changes in behavior that occur after an episode of abnormal electrical activity in the brain**. Notably, the term **"seizure"** is often used interchangeably with **"convulsion"** and so must also be considered. **During convulsions**, the **person's muscles contract and relax repeatedly**. Some have **mild symptoms without shaking.** Most seizures **stop by themselves**. But **during a seizure, the person can be hurt** or injured. An example is **“tongue biting”**.

Stroke, also called a “brain attack” is a serious **medical emergency** affecting **blood flow to the brain**. **Stroke mimics have similar effect**s. The best examples are **migraine, seizures** or having either low or **elevated blood sugar**. Strokes occur when **a part of the brain is deprived of oxygen** and nutrients. **Eighty percent of strokes** are **"ischemic,"** caused by the **narrowing of the large or small arteries of the brain**, or by **clots** that **block blood flow to the brain**.

A **stroke-like episode** is an **acute event** that very much **resembles a stroke**. A stroke is a **sudden loss of consciousness** due to an **acute vascular disturbance** caused by the **rupture of an artery in the brain** or its **obstruction** by a **blood clot** (embolism or thrombosis). It can present in **several ways**:

drowsiness, dulness, subcoma, coma, hemiparesis (paralysis on one side of the body), or paralysis on both sides), loss of vision.

Unconscious proprioception is the 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. On the other hand, a convulsion suggests a contortion of the body caused by violent, involuntary muscular contractions of the extremities, trunk, and head".

A convulsion is the result of abnormal electrical activity in the brain and may be followed by deep sleep. The same record at N-6 documents "No response to verbal or physical stimulation, repositioned by nurses". Notably the time of that entry is documented as being 0030 hours when the duty Nurses finally made their rounds and is preceded by a documented 2330 hours after having made the previous round, suggestive of having left the patient unattended for several hours before looking in on her.

During convulsions, the person's muscles contract and relax repeatedly. There are many different types of seizures. Some have mild symptoms without shaking. A seizure is the physical findings or changes in behavior that occur after an episode of abnormal electrical activity in the brain. A seizure usually lasts no longer than a minute or two. But during a seizure, the person can be hurt or injured. Notably, the term "seizure" is often used interchangeably with "convulsion" and so must also be considered because the patient had been left unattended.

A generalized tonic-clonic seizure, sometimes called a grand mal seizure, is a disturbance in the functioning of both sides of your brain. This disturbance sends out electrical signals to your muscles, nerves, or glands. These signals can make you lose consciousness and have severe muscle contractions.

The ED physician, Dr. Spiller was up to assess the patient's condition. Upon examination the patient's eyes were documented as being "sluggish", with "pupils dilated at approx. 5 mm" with "very little reaction to light". 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".

N-3 of the record documents "resp noisy", "shallow", "Cheyne-stoke patten" 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**.

A-17, what I take to be the **Ventilation Record** 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**. Further submit that **the intubation may actually have taken place at an even earlier time,** such as **0320 hours** as evidenced by the record at **A-24**, which documents a **heart rate** (HR) of **"174 bpm"** at **0320 hrs** that is consistent with an **"awake intubation"** (any suspicion of difficulty intubating, for any reason), marked by **panic with awareness,** or **shock** resulting in the **increase in HR** (heart rate) to **174 bpm** at that time.

The Vital Signs Record at **A-24** which documents a **HR of 174 bpm** at **0320 hours** is consistent with **trauma**, although is **contradicted** by the **Ventilation Record** seen at **A-16,** which documents a **HR** 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**.

The **vital signs** can also 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 attention**.

Also know that the **Vital Signs Record** is a **mechanical record** with a **run time**, while the **Ventilation Record** is a **handwritten** account, in this case **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 to **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"** 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 duty nurses, as to infer **negligence** including **failure on the part of Dr. Jordan to properly perform the intubation or 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. Further, it is also clear that the Ventilation Therapist, Helen Studholm was **not** present **to oversee the procedure** in a timely manner.

When an **endotrachial tube** (ET) is **misplaced** in the **esophagus** and **misplacement is detected late**, a **compromise** of the **patient's safety** can be significant. **Malpositioning** of the **ET** can cause **airway obstruction** and may also result in **tissue trauma** and **bleeding**. Further, **Iatrogenic perforation** is the **leading cause** of **esophageal perforation**. The possibility of ***iatrogenic*** **perforation** thus **cannot be ruled out**.

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 **may include laryngospasm**, **bradycardia**, **hypotension**, **cardiac arrest**, and **apnea**. In some patients there is even the possibility of **increased intracranial pressure** (ICP) and **increased intraocular pressure**.

In addition to **hypoxia**, **delayed tube repositioning** can **lead to** unilateral **pulmonary edema**. **When ventilation is not achieved in a timely manner**,

irreversible brain damage can result within minutes. Therefore, the **maximum interval allowable for conservative airway management maneuvers is about 3 minutes.** That was **NOT** the case here.

The **ambulance call** report seen at **N-7** documents that the patient was intubated and vented and that she was seen to be **"stable"**; also that she appeared to be **"pale, dry, and cool"**. Pale skin suggests **decreased blood supply** to the skin. The **paleness** is relative to **insufficiency in oxygen supply** distributed to the tissue of the body. Blood vessels in the body **constrict** to conserve blood in the body's core, making you **feel cold** and your skin go **pale**. If a tissue is **not** being **perfused properly**, it may feel **cold** and appear **pale**. Further, the **skin** is the **first place to be robbed of water**, resulting in **"dry"** skin. **Dry skin** suggests a **dehydrated** patient. The skin turns **"pale and cold"**.

Cool, dry skin can also suggest **late sepsis**. Patients progressing from **sepsis** to **severe sepsis** become profoundly **dehydrated**. While **sepsis** is triggered by a **hyper activation of the immune response**, most sepsis patients **actually die from immune paralysis**.

Sepsis is caused by the **immune system's response to a serious infection**, most commonly bacteria, but also fungi, viruses, and **parasites** in the **blood, urinary tract**, **lungs**, **skin, or other tissues**. **Sepsis** is often called **septicaemia** or **blood poisoning** when the body is **fighting** a **severe infection** that has **spread via the bloodstream**.

Septicaemia can occur with or without meningitis. Meningitis and septicaemia can kill in hours.

Caveat - **Sepsis** causes **profound hypotension**. Notably, **sepsis** refers to the presence of an **infection**, **plus** any **two** of these **four** criteria (three of the four have been met):

. 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, and toxic appearance. Nonspecific but contributing findings include leukocytosis, neutrophilia, and toxic granulations. Hallmark of infection is fever although temperature can vary with severity of sepsis, typically "37.2°C" or <36°C can be indicative of severe infection. Tachypnea is a medical term that means "rapid breathing". Tachypnea may or may not involve labored breathing.

A-8 of the record documents "patient was unconscious with respirations of approximately 30 and laboured", that is consistent with dyspnea - difficult or labored respiration. Dyspnea is breathlessness due to high filling pressures and pulmonary congestion/edema, i.e. shortness of breath, a smothering feeling, inability to get enough air, and suffocation.

A-26 of the record documents a BP (blood pressure) of 78/70 at 0235 hrs. Notably, Septic shock is sepsis with hypotension (systolic BP less than 90 (<90) mm Hg or a reduction of 40 mm Hg from baseline) despite adequate fluid resuscitation. In severe sepsis, if blood pressure drops to dangerous levels, organs are prevented from getting enough oxygenated blood, and this is called 'septic shock'.

Septic encephalopathy is associated with breakdown of the blood brain barrier and cerebral oedema. Septic encephalopathy has been defined as "brain dysfunction secondary to sepsis". Notably,the record at OP-53 documents a body temperature of "37.3°C" that is also consistent with sepsis.

The most frequent sites of infection leading to sepsis are the lung, urinary tract, abdomen, and pelvis. In up to 30 percent of patients, 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. The most dramatic manifestation of sepsis on the lung is acute respiratory distress syndrome.

Further, know that pathogenic bacteria grow best at human body temperatures in the 37°C range. For the record, also know that a parasite is the likely source of organism that also grow best in the 37°C range. Notably, parasites capable of growth at or above 37°C are potential pathogens — more virulent. Amoeba only multiplies rapidly if a person is very run down by an immune deficiency, illness or infection.

Notably, a number of non-pathogenic strains of amoeba have been isolated from human carriers. These amoeba can be cultured at room temperature at 37°C and will grow in hypotonic media, whereas pathogenic amoeba require isotonic media and 37°C as well for growth.

The record at A-17 documents a complete cessation of accessory and abdominal muscles, evidenced by a "0 use of acc muscles" and a "0 use of abd muscles" (accessory muscles and diaphragm are affected). The time of that assessment is documented at 0330 hours and may suggest a rapid weakening or loss of abdoninal and accessory muscle function.

The accessory and abdominal muscles include the muscles of respiration. These muscles are the paired muscles of the flank and stomach that surround and support the abdominal viscera. The abdomen contains the stomach (the stomach is a part of the abdomen), small intestine, colon, rectum, liver, spleen, pancreas, kidney, appendix, gallbladder and bladder. When the muscle of the stomach is weakened or paralyzed by a disease of either the stomach muscle itself, or the nerves controlling the muscles, as a consequence, food and secretions do not empty normally from the stomach, and there is nausea and vomiting.

The abdominal muscles are a group muscles in a person's upper body. They run from a person's ribs all the way down to the pelvis. They also assist in posture, breathing, and movement of the legs. These muscles facilitate the spine's forward movement and flexing which make it possible for a person to

move his upper body in all sorts of directions. **Loss of muscle function** can be **permanent** or **temporary,** depending on the cause.

Accessory muscles assume some or all of the work of **breathing**. The function of the **accessory muscles** is also to **stabilize the thorax** during ventilation. Weakening of **accessory muscles** can lead to **ventilatory failure** due to **fatigue. Paralysis** of the **abdominal musculature** can be both devastating and **catastrophic**.

Muscle tone and movement involve the **brain, spinal cord, nerves**, and **muscles**. The accessory **muscles of ventilation** face **greater load and gas exchange** when **deranged** due to **poor matching of ventilation to perfusion** in the **affected areas** which in turn **may lead to hypoxemia**, at rest, and more so **during sleep**. The **function** of the **accessory muscle** is to **stabilize** the **thorax** during **ventilation. Respiratory and accessory muscle "paralysis"** (crisis) **requiring intubation** and **mechanical ventilation** can be **catastrophic**. **Accessory muscle paralysis** will result in **apprehension and anxiety** = **PANIC** The **loss of function** in **involuntary muscles** can be **fatal.**

Muscle function tends to occur in what are called **voluntary muscles**. Voluntary muscles are **skeletal muscles** that you have full control over. **Involuntary muscles**, such as the **heart** and **intestinal smooth muscles** are **not within your conscious control**. However, they too can **cease functioning**. Common causes for the loss of **voluntary muscle function** are **diseases of the muscles** and **diseases of the nervous system**.

Muscle paralysis happens when something goes awry with the passage of **messages** between your **brain** and **muscles**. **Muscle paralysis** is caused by a **loss** or **impairment** to a **neural** or **muscular** mechanism. A complete **cessation** of **abdominal** and **accessory muscles** indicates that the **brain and nerves are no longer controlling the muscles**. In the absence of a robust immune response, **infection** can cross the **blood-brain barrier** and initiate a devastating **neuroinvasive illness** than can cause a **flaccid paralysis**. **Panic**, hysteria or a **severe emotional trauma** may also cause paralysis.

Paralysis is most often caused by **damage** to the **nervous system** or **brain,**

especially the spinal cord while the muscles themselves stay intact. There are several possible causes of paralysis and the major ones are stroke, trauma, poliomyelitis, amyotrophic lateral sclerosis, botulism, multiple sclerosis, and Guillain-Barré syndrome. Also, for the record, one of the most severe notable effects associated with adversities of morphine is skeletal muscle flaccidity.

N-5 documents "No response to deep pain" at 0055 hours, suggests a state of "stupor" marked by mental dullness. Notably, N-2 documents "attempts to pull away to painful stimuli" at 0400 hours on May 24th. A delayed reaction to external stimuli suggests a deep sleep-like state.

Stupor is an excessively long or deep sleep-like state marked by mental dullness. 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 verbal or 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.

A-27 documents a falling pulse rate between 0320 and 0415 hours. The same record documents a Motor Response of "4 Withdraws" for both the arms and legs at 0415 hours, where earlier at 0320 hours there was no response at all, although the pupils were documented as being dialated and fixed at 6 on the pupil scale. Marked mydriasis (pupil dilation) rather than miosis may be seen due to severe hypoxia (brain injury caused by a lack of oxygen to the brain) in overdose situations.

Hypoxia is caused by a lack of oxygen in the tissues and organs of the body. The condition is treated by improving oxygenation and increasing the pO2 (partial pressure of oxygen) in the blood. Hypoxia can result from a failure at any stage in the delivery of oxygen to cells.

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 a reference to "Gurgly resps" at the very same time. Gasping has also been described as gurgling, agonal, or labored breathing.

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. Bubbling, or crackling sounds when breathing can also suggest pneumonia. Gurgling sounds are suggestive of pulmonary edema (Thelan, et al.1996) .

Suctioning is the protocol used for clearing oral secretions. In this instant suctioning was not done first and the patient's airway was undoubtedly compromised by the insertion of the 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-4 documents "incontinent blood tinged urine" at 0305 hours. Incontinent tinged urine is also consistent with dehydration, often mistaken for hematuria (blood in urine). Hematuria is inconsistent with a "large amount of dilute urine" documented at N-3, suggestive of either diabetes insipidus, or over-hydration, as occurs with overzealous IV administration.

Incontinence can also be the result of hypotonia, or neurogenic bladder. The color and volume of urine can be reliable indicators of hydration level. Urinary incontinence can be a symptom of both over-hydration and dehydration. A small amount of dark tinged urine suggests dehydration, while a large amount of dilute urine suggests over-hydration. Prolonged or severe dehydration leads to abnormally dark tinged urine.

N-3 of the record also documents a "large amount of dilute urine" (polyuria) at 0325 hours, only 20 minutes later, suggests diabetes insipidus, or a patient demonstrated hypotonic hyponatremia with maximally dilute urine that is consistent with IV overload. Inappropriate use of hypotonic fluid can

cause hyponatremia. A description of the mechanism by which hypotonic fluid can cause hyponatremia is reported in the literature. In acute hyponatremia, the brain cells are unable to compensate for the rapid decrease in serum osmolality; as such, minor increases in electrolyte-free water can lead to disproportionately large increases in intracranial pressure due to swelling of the brain cells. There follows a review of the extensive clinical evidence linking hyponatremic-induced brain injury and deaths with administration of hypotonic fluids.

Hypotonic dehydration causes the amount of deficit to be overestimated by physical examination. It is important to note that hyponatremia more often represents excess of water than insufficiency of sodium. Adequate sodium balance is necessary for transmitting nerve impulses and proper muscle function, and even a slight depletion of this concentration can cause problems.

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 is also consistent with water intoxication.

A common cause of hyponatraemia is hypotonic dehydration and iatrogenic water overload (overestimation of the degree of dehydration), inappropriate use of hypotonic solutions for rehydration and/or too rapid administration of maintenance fluids. Hyponatraemia also occurs because high plasma glucose increases serum osmolarity, causing a shift of water from the intracellular space into extracellular fluid. Other causes include SIADH, especially in patients with meningitis. SIADH is a volume-expanded state.

SIADH (syndrome of anti-diuretic hormone) is water overload and not salt depletion. This leads to excess water elimination as dilute urine. Use of hypotonic fluids in presence of circulating ADH can causes free water retention resulting in hyponatremia. Other nonosmotic stimuli for the release of ADH (anti-diuretic hormone) include pain, stress, fear gastro-enteritis, hypoxia, positive pressure ventilation, trauma, and medications

such as opioids.

Conditions with impaired free water excretion and high anti-diuretic hormone levels include

- Meningitis, encephalitis, pneumonia, bronchiolitis, sepsis
- Surgery, pain, nausea and vomiting

Irreparable harm can befall the patient when abnormal serum sodium levels are corrected too quickly or too slowly. Rapid correction of hyponatremia, even mild hyponatremia, risks neurologic complications.

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, infection, tuberculosis, including Guillain-Barré syndrome. Certain drugs can also cause this condition, ie., phenothiazines, and morphine sulfate. Severe hyponatremia can lead to encephalopathy, brain damage, and death; young women are at highest risk.

Hyponatremia may occur in up to one-third of patients with Guillain-Barre syndrome (GBS) and the syndrome of inappropriate antidiuretic hormone (SIADH) has been reported to accompany the neurologic manifestations of the Guillain-Barre syndrome. An association between SIADH and diabetes mellitus has also been reported; the basic fluid problem in SIADH is water overload and not salt depletion. One of the most potent stimuli for ADH release is nausea and vomiting.

Hyponatremia exerts most of its clinical effects on the brain. Brain volume is regulated by equal osmolality of extracellular and intracellular fluid. When extracellular osmolality decreases, water influx occurs in the brain resulting in cerebral edema due to electrolyte-free water moving into the brain cells. Cerebral edema is responsible for symptoms such as headache, nausea,

vomiting, irritability, and seizures.

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 (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. Polyuria has been observed in a severe case of GBS. Polyuria in GBS is multifactorial and would be partly due to a dysregulation of osmoreceptors. Besides dangerous cardiac manifestations, neuro-endocrine changes could induce electrolytes and fluid balance impairments. The hyperglycemia emanates from a commonly identified diabetogenic stressor, such as infection, which precipitates the onset of the syndrome which in turn produces pseudo-hyponatremia commonly associated with hyperglycemia. Another is drug-induced hyperglycemia, which should not be overlooked in this case.

CAVEAT: Hyperglycemia can lower the serum sodium concentration by 1.6 mEq/L for each 100 mg/dl, giving rise to a false test. There is undoubtedly an involvement of metabolic derangement and neuronal injury in the detrimental effects of hyperglycemia. Notably, hyperglycemia is particularly detrimental in ischemia/reperfusion.

Hyperglycemia is ALSO reported to trigger massive neutrophil deposition in the brain following transient ischemia.

Hyponatremia is decrease in serum Na (natrium) concentration < 136 mEq/L caused by an excess of water relative to solute. Clinical manifestations are primarily neurologic (due to an osmotic shift of water into brain cells causing edema), especially in acute hyponatremia, and include headache, confusion, and stupor; seizures and coma may also occur. The diagnosis is made by

measuring serum Na. Serum and urine electrolytes and osmolality help determine the cause. Treatment involves restricting water intake and promoting its loss, replacing any Na deficit, and correcting the underlying cause. Symptoms mainly involve CNS dysfunction and occasionally osmotic demyelination syndrome or brain stem herniation.

Serum Na may be low when severe hyperglycemia increases osmolality and water moves out of cells into the ECF. Serum Na concentration falls about 1.6 mEq/L for every 100-mg/dL (5.55-mmol/L) rise in the serum glucose concentration above normal.

Overloading the circulatory system with excessive IV fluids causes increased blood pressure and central venous pressure. Signs and symptoms of fluid overload include moist crackles on auscultation of the lungs, edema, dyspnea, and respirations that are shallow and have an increased rate.

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

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.

In my opinion, the solitary lesion is also consistent in appearance with an amoebic cyst, or capsul stage of abscess development. The small lesion would have been asymptomatic at that time. Cerebral abscess formation results from invasion of cerebral capillary endothelium by infected macrophages characteristic of cerebritis (a local area of inflamed, infected brain tissue) in the early developmental stages of abscess formation.

The opionated expert (whose name was erased) goes on to state "She was then transferred with ventilatory support to Sudbury under the care of Dr. Adegbite where a CT scan was done. It shows several large metastic tumors

with massive oedema of the right cerebral hemisphere, a 1 cm shift of the midline structure from right to left and evidence of extreme intracranial hypertension (compression of the third ventricle, obliteration of the ambient cystern and decreased attenuation through much of both cerebral hemispheres suggesting no cerebral perfusion). She was declared brain dead."

"It is clear although she remained relatively asymptomatic until the last week or two of her life, Arlene Berry had a particularily aggressive tumor with rapid evolution of several brain metastasis. The largest measures nearly 3 cms in diameter and I can count a total of four distinct tumors. There may, in fact, be more but with the impaired cerebral perfusion due to the high intracranial pressure they are not well opacified."

"Dr. Jordans discharge note documents a rapid deterioration that by 0245 hrs had resulted in complete cessation of motor response, verbal response and occular response, that is, Arlene Berry had a Glasgow Coma Scale of 3. He documents also that her pupils were midsize and fixed, a sign of brainstem malfunction. She was still breathing spontaneously."

"Had she been started on decadron she might have enjoyed respite from her headache and might have lived a few weeks longer, but with multiple metastatic tumors , she would have been inoperable and, have been pallative. Whole brain radiotherapy would have extended her life a very few months."

"In my opinion, the physicians who looked after Arlene Berry met a reasonable standard of care."

OPINION REBUTTAL

For the record, **cerebral perfusion pressure (**CPP) is the **net pressure gradient causing cerebral blood flow to the brain** (brain perfusion). It **must be maintained within narrow limits** because **too little** pressure **could cause brain tissue to become ischemic** (having inadequate blood flow), and **too much** could **raise intracranial pressure** (ICP).

Once infection has established a foothold on the membranes surrounding the brain, it triggers inflammation severe enough to cut off the blood supply resulting in decreased cerebral perfusion which causes swelling in the brain. Once cerebral flow ceases, the nerve cells and the capillary bed are damaged irreversibly.

The decreased attenuation throughout the cereberal hemispheres may result in stroke symptoms that include paralysis, which if left untreated, can result in herniation or massive hemorrhage into brain substance.

Clinical correlation with patient symptoms and white blood cell count are necessary to understand the etiology of the brain lesion. Clinical presentation of brain abscess is usually similar to other intracranial space occupying lesions but the symptoms of an abscess(s) tend to be more rapidly progressive than those associated with neoplasm. Another method that could help in distinguishing between abscess and tumor is perfusion imaging.

Massive haemorrhage into brain substance is characterized clinically by an abrupt onset and rapid evolution. Intracranial hypertension (IH) is high pressure inside the skull, which may happen suddenly or build up gradually over time. It's a relatively common condition with many different possible causes. Acute IH occurs when the condition comes on rapidly as the result of a severe head injury, stroke or brain abscess, for example.

Some medical conditions, such as the following, can cause chronic IH:

- a brain tumor such as a glioma or meningioma
- a brain infection such as meningitis or encephalitis
- hydrocephalus, which is a build-up of fluid in the cavities of the brain
- blood vessel abnormalities such as an arteriovenous fistula (an abnormal connection between an artery and a vein)
- blood clotting in one of the large veins of the brain known as a venous sinus thrombosis, usually caused by infection or severe dehydration

For the record, hypertonic saline is used for treating intracranial hypertension.

With meningitis, intracranial infection can result in cerebral abscesses which can lead to brain herniation and shift of midline structures. ICP itself can be responsible for further damage to the CNS by decreasing blood flow to the brain causing the brain to herniate (push through) the opening in the back of the skull where the spinal cord is attached . With meningitis, CT of the brain often shows obliteration of the cisternae surrounding the midbrain and of the subarachnoid space over the cerebral hemispheres (is reported throughout the medical literature).

At a subsequent meeting, Dr. McLellan provided us with a view of a second CT scan that was purportedly done in Sudbury at the time Arlene's death on May 24thy of 2000. It shows multiple lesions, but these are nonspecific. Based on the record and the findings contained herein, multiple cerebral abscesses secondary to an untreated brain abscess and hematogenous spread is the most plausible explanation. Appearances of lesions on CT with focal and/or multifocal contrast enhancement are frequently mistaken for brain tumors.

Contrast cerebral CT and MRI evaluation increase the specificity of diagnosis but none of them can differentiate between the lesion types. Nevertheless, in unclear cases, biopsy or surgery and subsequent pathology analysis can help establish the final precise diagnosis.

In terms of the second CT that was done in Sudbury, it shows cystic lesions consistent with multiple abscesses (dark areas with rims of enhancement). Tumors may show increased perfusion, whereas abscesses generally have "decreased perfusion", as in this case. SOURCE:

Further, microabscesses in the brain are not well opacified. The meninges overlying these foci may be cloudy. Micro-abscesses in the brain may develop into meningitis. Further, with multiple abscesses the meninges typically show a purulent exudate that obscures the sulci making radiographic appearance of microabscesses less visible, hence they are not well opacified.

Brain abscesses can produce "purulent meningitis" associated with signs of

neurologic damage or "brainstem malfunction". In severe meningitis, the basal cisterns may become completely "obliterated" and that is what happened here. Spontaneous meningitis can kill in 24 hrs if left untreated.

Brain abscess may also occur in a previous hemorrhage or infarction area as a complication of systemic infection. Brain abscess is a focal intracerebral infection, beginning with a localized area of cerebritis and developing into a collection of pus-like material surrounded by a well defined capsule.

Multiple brain abscesses secondary to an untreated brain abscess and hemato-genous spread is not uncommon. Single or multiple abscesses within the brain may mimic brain tumors but evolve more rapidly, from days to a few weeks. Amoebic abscess of the brain is commonly an extension of pre-existing amoebiasis in extra-intestinal viscera but may be a direct extension from the colon.

Although it may appear that this patient developed a brain abscess as infective transformation of a preceding intracerebral haemorrhage after urinary tract infection, it seems likely that infection with E histolytica played a key role in the development of multiple brain abscess formation.

Also, know now that a paltry CT scan does NOT provide conclusive proof of metastatic brain tumors. For that it takes a biopsy and in this case NO biopsy was done. Even when the imaging characteristics are very suggestive of tumor, a biopsy is the only way a precise diagnosis can be made. Therefore the true nature of the lesions were never truly established.

Clinically, many conditions producing increased intracranial pressure or progressive neurologic deficits mimic brain tumors. These include subdural hematomas, brain abscesses,hydrocephalus, benign intracranial hyper-tension, progressive multifocal leukoencephalopathy, multiple sclerosis, vascular malformations, cerebral infarctions, Alzheimer's disease, and some congenital anomalies.

Many of these conditions have characteristic radiologic appearances that enable them to be differentiated from brain tumors. However, some of them,

brain abscesses and certain inflammatory lesions, demyelinating disease, hamartomas, and congenital anomalies cannot be distinguished from brain tumors on the basis of their radiologic appearances alone, and a definite diagnosis often requires biopsy.

Even when the imaging characteristics of a lesion are very suggestive of a tumor, a biopsy is usually indicated to obtain tissue for precise histologic diagnosis and grading of the tumor since these factors will have an important bearing on treatment.

When symptoms of amoebic infection do occur, they usually begin within 4 months after amoebas first enter the body, which coincides with the left lung pneumonectomy that was performed on January 13th of 2000. 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. Untreated, a single capsule could easily rupture resulting in the formation of multiple abscesses.

E. histolytica rarely infects the CNS and when it does, it tends to cause abscess(s) with a fulminant clinical course culminating in the patient's death within 12-72 hours untreated, which explains Arlene Berry's sudden and unexpected demise.

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". The amoeba generally only causes encephalitis in people who are immunocompromised or have a chronic disease such as diabetes. For the record, also know that parasite spreading can promote a systemic response that resembles bacterial sepsis. Severe inflammatory response may also mature into a systemic response known as anaphylaxis..

Abscesses have been observed in frontal, parietal, temporal, and occipital lobes and cerebellum in cases of cerebral amebae infection. The tissue

sections from the brain, particularly at the **right occipital lobe**, may also reveal **acute suppurative meningitis** with the presence of **degenerated *E. Histolytica.*** Signs vary in severity. There may be a **tendency to deviate** or **fall to the side of the lesion while walking**, for example, **"pulling to the right"**. This is called **hemipareses**.

Morbidity due to a **brain abscess** generally results from **brain herniation** due to **mass effect**.

Submit, from the record there was every indication that Arlene Berry was about to suffer a **catastrophic decline**, at least from **foreseable "dehydration"** due to **decreased oral/water intake** and **malnutritian** from **excessive vomiting over the previous week or more**, which **should have prompted immediate medical attention, but did NOT**.

Submit also that **dehydration**, which **interupts blood flow** and **causes blood clots**, **cutting off the supply of oxygen** to **various parts of the body**, the **result** of **poor oxygen exchange,** marked by a **slurred speech**, a **sedated** and **haggard appearance**, and **drowsiness** together with **constitutional symptoms** documented on the record to include **headache** and **vomiting** with **severe stomach pain** evidenced by the **"abdominal pain ongoing for 2 weeks"** documented at **A-5,** and at **A-8** of the medical record suggests a **"life-threatening medical emergency"** that was basically ignored.

From **imprudent doctors and nurses**, to **lack of diagnostic thoroughness**, to **medication errors**, to **negligent and callous nursing care**, to **medical omission,** to **ignoring symptoms and outcomes to fatal conclusions**, to **acting with wanton and reckless disregard for human life**, to **terminal sedation**, to **altered and falsified medical records,** to **medical homicide**, **shows a recklessly careless scatomatous state of ignorance associated with a gross dereliction of duty that cannot support a reasonable standard of care**.

Over 50% of amebic brain abscess are associated with intestinal symptoms (Lombardo et al, 1964). Orbison et al (1951) collected 64 cases of amebic brain abscess from the literature and reported five more cases. More cases of amebic brain abscess have been documented since then (Lombardo et al, 1964; Hughes et al, 1975; **Becker** et al, 1980; Banerjee et al, 1983; Schmutzhard et al, 1986; Ohnishi et al, 1994; Shah et al, 1994; De Villiers and Durra, 1998; Di Rocco et al, 2004; Sundaram et al, 2004;

Solaymani-Mohammadi et al, 2007; Sayhan Emil et al, 2008).

EVIDENCE OF ALTERED MEDICAL RECORDS

Accurate record keeping is paramount to good patient care. In fact the medical record mirrors the degree of quality provided in healthcare.

Fraudulent addition or omission to a medical record for the purposes of covering up an incident can be detected by current technology. Some clues used to detect altered records include:

Clues to altered records:

- writing crowded around existing entries
- changes in slant, pressure
- uniformity or other differences in handwriting
- erasure or obliteration
- use of different pens or typewriters to write one entry
- misaligned typed notation, impressions or lack of impressions from writing instruments on the following pages
- ink offsets or lack of offsets on the back side of the preceding page, and
- additions on different dates written in the same ink, while original entries were written in different ink
- using a dating devise of rubber stamp, while original entries were written in ink

A medical record is considered complete only if it contains sufficient information to identify the patient; support the diagnosis/condition; justify the care, treatment, and services; document the course and results of care, treatment, and services; and promote continuity of care among providers. With the exception of sufficient information to identify the patient, clearly, that is NOT the case here.

All health care professionals should be aware that *falsification* of *medical records* is grounds

for criminal indictment.

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 at **N-1, N-2** and **N-3** of the **Nurses Notes,** and elsewhere, which are **marred by error, inconsistency, omission, and contradiction,** with **A-16**, and **A-17** presenting similarly.

The medical record for May 23rd and 24th of 2000 was **un sequentialy 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"**. There was **NO history of metastasis** and there is **nothing on record to prove** otherwise.

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 **copious error**. The bald truth is that Arlene Berry **had the left lung removed on January 13th of 2000** at the **Timmins & District Hospital** under the care of **Dr. Claudio De La Rocha**.

A-5 of the record was dated using a **"rubber stamp"** that is consistent with **backdating** techniques. The same record contains a very brief "**Triage Assessment**" with the "**Medications Administered**" **omitted. Notably,** in the **lower right center of the same document** there is an **obliterated** (barely visible) "**Trauma Legend**" suggesting a "**white-out**", or perhaps an **erasure**. Since **TRAUMA** is defined as **any insult to the body (**clinical or otherwise), it seems clear that **relevant information was deliberately removed** to **conceal** a **traumatic event**. The **signature** on this record also appears to be that of **nurse Bates RN**.

Several of the records contain **write-overs**, primarily with respect to **date and time**, also consistent with **making changes at a later date**.

The primary purpose of *backdating* is done with intent to absolve oneself of civil and/or criminal wrongdoing.

The Ventillation Record contains a **self-serving entry**, ie. "**without adversities**" relating to patient's **intubation** procedure which took place at about **0320 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 minutes later) which can suggest **thoracic injury**, **essophagal perforation.** 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.**

According to Dr. Jordan, **"The intubation was performed and at approximately 04:35 hours",** while **N-3** documents the **time of intubation** as being **0325 hours**, a significant difference.

N-4 presents with **less than half a page** of documentation **consistent with deliberate omission**, such as having **rewritten** the course of events between **0305** and **0320 hours,** for the express purpose of **removing incriminating information**. The **initials on the record at N-4** appear to be **"JM",** suggestive of **nurse McKrank**. The record at **N-1** presents **similarly**. The **signature** on **N-1** appears to be that of nurse **Janice Chamaillard**.

A-10, what I take to be **Progress Notes,** presents with a documented "**Female patient admitted to 413' via w/c - IV infusing**", and was **signed** by **nurse S. Ferguson** at **18:45 hours** on May 23rd, of 2000. There are **no further entries;** the **remainder** of the **entire page** is a **complete blank**.

A-16 documents a **blood pressure** of **163/117** at **0330 hours**, while **N-3** of the **Nurses Notes** documents a **blood pressure** of **136/85 at the very same time**.

The same record at **A-16** 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 documents a blood pressure of 78/70 at 0235 hours, while N-5 documents a blood pressure of 98/70 at the very same time, suggestive of copious error, usually made while attempting to copy from someone elses illegible handwriting.

The 24 hour fluid balance record seen at A-15 appears not only to have been rewritten, but also to be a "forgery", based on the handwritten entries seen between 1745 hours and 1900 hours, bearing the initials of three nurses in the very same handwritting. Notably, the handwriting appears to be that of nurse McCrank, RN.

N-10 of the Nurses' Notes document the patient's level of care as "routine" with a single entry in the upper right hand corner, 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 also 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.

Further, there is nothing on record to suggest that any Supportive Care & Symptom Control Regimens were ever implemented. NO Complete Physical Exam to include a Rectal Examination (colon-oscopy), and NO meaningful Nurses Diagnosis was made as per INTERNATIONAL CLASSIFICATION FOR NURSING PRACTICE.

N-4 of the record documents that Dr. Jordan was called in at 0225 hours and the time of his arrival at 0305 hours.

A-1 of the record documents Dr. Jordan's "I was called in later that night because the patient had become obtunded" (unresponsive), while N-2 documents "attempts to pull away to painful stimuli" (suggestive of a deep sleep-like state.) as late as 0400 hours, some 55 minutes later. For the record, raised ICP (intra-cranial pressure) is an expected complication of meningitis and is the major cause of "obtundation" and coma. However, in

coma, a person is highly unlikely be roused to painful stimulation.

NO protocols were ever followed or implemented in this case. 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. For examle, the record at OP-53 is totally devoid of annotation with respect to the patient's "bowel routine" for toileting, marked by a complete absence of nursing care plan as further evidenced at A-21 of the medical record.

The element of duty is straightforward and relatively easy to prove because once doctors and nurses undertake care for their patients they have a clear duty to provide care for that patient in a competent and reasonable manner. That has NOT been the case here.

With the exception of OP-53, 54 and 55, the remaining Outpatient Records, particularly between November of 1999 and mid May of 2000 were withheld altogether. According to the Kirkland & District Hospital Health Records Charge Person (Muriel Olson), "that's all of them".

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 "call received at 0620 hours", a significant difference suggesting that not only did the primary care physician lie, but also that this patient was deliberately made to deteriorate.

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 as being "55 years", suggestive of possible record swapping, which cannot be ruled out. Arlene

Berry was only **41** at the time of her death. **How does age 41 become confused with age 55 in a hospital setting where accurate record keeping is paramount to good pt care?**

A-1 and **A-2** documents the patient's **HISTORY OF PRESENT ILLNESS** beginning with **"This is a young 42-year-old-female"** — she was only **41** at the time of her death. The same record documents **"She had a left pneumonectomy back in October of 1999"** — the **pneumonectomy** was actually done in Timmins on **January 13th of 2000**. **How does October 1999 become confused with January 2000 ?** The **signature** is that of the **attending physician**, namely **"Dr. Jordan"**.

It should be clear by now that the record of Arlene Berry for May 23 and 24 of 2000 **mirrors** the degree of quality provided in her healthcare.

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"** (No Code) 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 DNR declaration of death** and in fact **waited for the patient's death**.

Do not resuscitate (DNR) means **no chest compressions**, **no defibrillation**, **no assisted ventilation**, **no endotracheal intubation**, and **no cardiotonic medications**. The same record documents a Code 3.3 "Withholding Treatment". **There was NO "Do Not Resuscitate" order on the patient's health record nor had there ever been a designated agent who declined continued resuscitation on behalf of the patient. The decision to terminate Arlene Berry was made solely by Dr. Edward Henry Jordan and his accomplices.**

Within only a matter of 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 held in Sudbury for several days prior to being returned to Kirkland Lake.

One might ask, was the minimally acceptable observation period to ensure that neurologic functions had ceased irreversibly following the patient's transfer to sudbury applied ? Or was the patient simply rushed to her death over a period of only a meager few hours ?

Many patients diagnosed as brain dead do not have hemodynamic collapse, they have physical findings such as bowel sounds, spontaneous breathing and are reported to have autonomic reflexes (tachycardia and hypertension) at the time of organ retrieval, suggesting a horrific death.

Autonomic dysfunction is also a well recognized manifestation of Guillain-Barre syndrome with unusual autonomic dysfunction at its onset, consisting of constipation and hypertension, followed by adynamic ileus, endocrine abnormalities and flaccid paraparesis with areflexia. Progressive motor weakness may be accompanied by sensory and autonomic dysfunction, requiring rapid respiratory or hemodynamic stabilization.

GBS frequently follows a flu-like illness, or viral syndrome by a week or two, as in this case. The most frequent impairment results from immunologic reaction against nerve roots, peripheral nerves, and cranial nerves. The typical clinical manifestation is motor weakness beginning in the legs and ascending to the arms, with symptoms evolving over a few days. Approximately 14% of patients will present with symptoms beginning in the cranial nerves or arms, descending to the legs.

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.

Flu-like illness is also a common complication of radiation therapy which

results from **radiation injury of the CNS.** However, in addition to **flu-like** symptoms, **untoward symptoms** including **low-grade fever, headache, dizziness, nausea and vomiting** are also **high on the order of infection**.

Autonomic dysfunction in **Guillain-Barré Syndrome**, is **manifested as tachycardia** and mild **hypertension** in the **acute stage** and **cardiac arrhythmias** associated with **autonomic dysfunction** are a **recognized manifestation in GBS**.

Sinus tachycardia, (>90/min), is **seen in over 35% of patients** with Guillain-Barré Syndrome, and over 30% suffer from **hypertension** (Parry, 1993). Symptoms of GBS **get worse very quickly**. The clinical features of GBS can range from **asymptomatic to life threatening**. It **may only take a few hours** to reach the **most severe** symptoms. The **consequences** of a **missed diagnosis** and **delayed treatment** can be **catastrophic**.

Fulminant Guillain-Barré syndrome can rapidly progress to a pseudo-coma state resembling brain death but with self-awareness preserved.

Bickerstaff's brainstem encephalitis (BBE), **Miller-Fisher syndrome** (MFS) and **Guillain-Barré syndrome** (GBS) are **similar clinically**; BBE and MFS have been postulated to be **variants of GBS**. **BBE** is characterized by **acute onset** of **ophthalmoplegia, ataxia, disturbance of consciousness, hyperreflexia or Babinski sign** (Bickerstaff, 1957; Al-Din et al.,1982).

The characteristic clinical feature of **GBS is rapidly progressive muscle weakness**, often ascending from lower to upper limbs and more marked proximally than distally. Distal paraesthesia and limb pains often precede the weakness. **Facial** or bulbar **weakness commonly develops**, and **respiratory weakness requiring ventilatory support** occurs in 20% of cases. In most patients **muscle weakness** progresses for **1-3 weeks**, but **rapid deterioration** with **respiratory failure** can **develop within hours**. The most **striking findings** on examination are **"diffuse weakness"** with **widespread loss of reflexes**. An

unusual variant of GBS described by Miller Fisher comprises the triad of ophthalmoplegia, ataxia and areflexia.

Miller Fisher syndrome (MFS) is characterized by the acute onset of external ophthalmoplegia, ataxia of cerebellar type, and the loss of tendon reflexes. It is considered a variant of Guillain-Barré syndrome (GBS), because some patients who present with MFS progress to GBS. In contrast, patients who show drowsiness, brisk reflexes, extensor plantar responses and hemisensory disturbance are usually considered to have Bickerstaff's brainstem encephalitis (BBE) rather than MFS. The fact that BBE and MFS share a common autoantibody suggests that they are closely related. Bickerstaff and Cloake speculated that the etiology of BBE is similar to that of GBS because they found areflexia and cerebrospinal fluid albuminocytologic dissociation; however, overlapping MFS, GBS and BBE form continueous spectrum.

In its typical form, GBS causes rapidly progressive diffuse weakness or paralysis of the four limbs, and areflexia. The sudden and unexpected onset of paralysis is devastating for patients and represents one of the serious emergencies in neurology.

Knowledge of GBS and its variants is important to prevent inappropriate declaration of brain death or withdrawal of life support in the face of potentially reversible causes.

Most sets of criteria for brain death (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 are done to identify brain death or even if they are accurately done.

CNS depressant drug effects were undoubtedly still present at the time of the brain death declaration. Further, prochlorperazine (Stemetil) has

neuromuscular blocking effects; neuromuscular blockade is a reversible causes of coma that can mimic brain death.

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 (BD) 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. It is the "deprevation of oxygen" that results in brain death.

To practice euthenasia by withdrawing life support to a critically ill patient constitutes 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.

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. In my opinion, the care-givers who attended to this patient acted with wanton and reckless disregard for human life right from the very beginning of her hospital admission and subsequent transfer out to Sudbury Regional Hospital.

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.

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 further, with intent to defraud the estate of the deceased (her children) of any lawful claim they may have had against the doctors, nurses, and hospitals for their part in substandard care, in both civil and criminal wrongdoing. From the record, it is also clear that they chose by their own doings to escalate their plot into a "criminal conspiracy and criminal coverup".

No autopsy was performed because the patient died "while under the care of a physician". A family request for a formal inquest into this death was also denied. In terms of the brain death declaration, 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.

Are you aware, 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, or lack of diagnostic thoroughness, incompetence, or gross medical negligence. In my opinion its about time that doctors and hospitals be held criminally accountable for their actions.

A wrong diagnosis can be devastating. Far too many physicians suffer from "tunnel vision" based on preconceived notions which corrupt differential diagnoses. A missed or wrong diagnosis can lead to delays in treatment, wrong treatments or no treatments at all. Mistaking symptoms of another condition for cancer when no cancer is present can have catastrophic consequences.

At the time of Arlene Berry's death the KDH had been undergoing costly renovations in the face of hospital cutbacks. For the record, public funds are for patients, not profits, and certainly NOT for superficial renovations to bolster corporate ego's.

Arlene Berry's remains were returned to Kirkland Lake several days after family had been notified of her death, only hours after her transfer out to Sudbury Regional Hospital.

On seeing the deceased following her return to Kirkland Lake, her eyes were "sunken in appearance", with signs of dehydration, such as dryness and pallor, with swelling and distortion of the face, eyes, and lips, as was the case, marked by a 3-4 cm rash-like redness (resembling a sunburn), and what appeared to be an erythematous halo surrounded by circular blistering wrinkles in the skin on her right cheek in the area just below the right eye, consistent with a delayed hypersensitivity reaction, or fixed drug-eruption, evidenced by all who attended Arlene Berry's viewing at the Monette Funeral Chapel in Kirkland Lake on the days just prior to her burial.

Death by dehydration is accompanied by convulsions, retraction of the eyes into their orbits, drying out of the mouth and skin, among other things. before death results several days later in a cruel and violent death.

Fixed drug eruption (FDE) is a common cutaneous reaction which may be seen in reaction to several medications. For example, drug interactions are reported among people who take Gravol and Morphine together. Swelling of the lips is highly suggestive of acute anaphylaxis.

The onset of a fixed drug eruption can be sudden, developing within 30 minutes to 8 to 16 hours after ingestion of the medication. In patients who continue to take the offending drug, the number of eruption sites may gradually increase. Fixed drug eruptions can occur anywhere on the skin surface after drug withdrawal, that is consistent with "reaction with readministration".

Notably, Arlene Berry had been scheduled for an x-ray follow-up in Sudbury on Tuesday May 30th of 2000 at 2:30 PM. Sadly, due to negligence, mismanagement and criminal wrongdoing, she did not live long enough to meet this appointment. Ontario's healthcare system had failed her.

A Time For Change

As few as **5 percent** and only **up to 10 percent** of **iatrogenic illness is ever reported.** **Less than 1% is even looked into**. The **unpalatable truth** is if you are an **iatrogenic** patient due to **clinical negligence** there is a **culture of denial and cover-up**. The **coroners office** is **without exception**. They **prefer power, lies** and **criminal cover-up over morality**. In terms of a **medical homicide**, the **true nature of death** in this case was **falsely ascribed** or simply **chalked up to natural causes** in a way that **flaunts** the **law, regardless of the truth** which **attempts to appease** everyone.

It is common knowledge that **stealth doctors hasten death** by **withholding life support** or using **high doses of morphine** in **cancer patient**s. A **combination of other drugs may also be used** to **obscure the clinical course**. **When combined, certain drugs, medications, substances or toxins may also react causing seizures**. 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**.

As **autopsy** rates continue to **decline**, the **opportunity** to **measure mis-diagnosis** or **foul play** in this way is **purposely reduced** leaving the door wide open to **medical homicide**. Not a day goes by without a "**medical murder**" somewhere. Even **Ontario's Coroner system** is **riddled** with **flaws** deemed to be **systemic** in nature, which **allows coroners** to return a **false finding contrary to the provisions of the Criminal Code of Canada**, and if the truth be known, **doctors and hospitals** are the **leading cause of death globally** and **Canada is no exception**.

Whenever you have a **flawed system** whereby **only doctors can investigate doctors**, **nothing gets done**. Its the very same with **complaints** against the police, including **any of the various authorities**. **Self regulation has** and **will always be a failure**. In Canada, **especially in Ontario**, the **government cannot even be trusted to regulate itself**. In Ontario, **hospital deaths** are **rarely if ever investigated**. In fact, **Ontario** has the most **protected medical murder operation** in **Canada** and **justice delayed is justice denied**.

That we are living in a disposable society, without values, there is not a shadow of a doubt. Such are crimes as in this case ranging from medical murder to criminal negligence causing death to corporate (hospital) criminal cover-up, criminal conspiracy and government acquiescence, obstruction of justice and collusion utilizing half truths, bald falsehoods and all the cloak and dagger techniques known to spies to obfuscate the truth; paradoxically, in Ontario, we have a rise to prominence of a powerful minority of misguided zealots opposed to truth and justice who find it easier to turn a blind-eye than confront the truth.

In terms of conspiracy and cover-up, silence doesn't only imply concealment, it also infers guilt

Pressing action for criminal accountability is urgently needed in preventing medical homicide from occurring in the future. The present climate of impunity in Ontario courts has only encouraged bad doctors to flout the law itself.

The Federal Court of Canada may offer some relief, since federal authority for criminal law and procedure ensures fair and consistent treatment of criminal behaviour across the country.

There's a trend in the UK towards prosecuting doctors for manslaughter. Some are even ending up in jail. So I think that the message I'm giving you is that if Canadian doctors and nurses do something really serious or show a blatant disregard for a patient's welfare, then such person(s) can and should also be threatened, charged and prosecuted with criminal charges.

Whenever you grant immunity from fault you breed irresponsibility.

No policy change or audit will ever bring back this young mom. However, by making the doctors and nurses criminally accountable, this investigator hopes to help reduce the likelihood of a similar recurrence.

In Memory of Arlene Berry 1958-2000

Malcolm Everett