PROBLEMS IN PROVING BREAST CANCER CAUSATION

by

JEFFREY C. ANDERSON

LAW OFFICES OF JEFFREY C. ANDERSON
9601 McAllister Freeway, Suite 1250
San Antonio, Texas 78216
(210) 340-8880
(210) 340-8885 Fax
jca@texaslawfirm.com
www.texaslawfirm.com

ST. MARY'S - TEXAS TECH SCHOOLS OF LAW
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TABLE OF CONTENTS

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I. INTRODUCTION

Because of some ill-advised publicity campaigns for screening programs by our colleagues in the medical community, there appears to be a significant gap between public expectations and the medical community's ability to cure breast cancer. Causes of action based upon missed breast tumors account for the highest percentage of medical malpractice cases in the United States. Thanks to the medical community's persistent claims that early detection of breast cancer can result in a cure, patients have been taking the medical community at its word and have been insisting upon a clinical success rate that is not yet possible.

Trial lawyers all too frequently accept the dogma that if cancer can be diagnosed early enough, it can be cured; therefore, the failure of a physician to make the correct diagnosis resulted in the cancer becoming invasive and metastatic. If the interval between the point at which a tumor should have been diagnosed and the time that it is actually found was the sole or even primary determining factor in a patient's survivability, proving the element of causation in breast cancer cases would not be tremendously difficult, and I would not be writing this paper. Too many trial lawyers jump into medical negligence cases involving the misdiagnosis of breast cancer only to be buried by a boxcar load of conflicting statistics regarding doubling times, lead time bias, genetic factors, survival rates and treatment effectiveness. The factors frequently associated with the prognosis of malignant breast tumors include tumor size, nodal involvement, metastasis, histologic type, estrogen and progesterone receptor proteins, genetics and treatment protocols. These factors frequently combine in such a manner that predicting survivability accurately becomes impossible. There are conflicting statistics on all of these elements in the medical literature. And therein lies the problem: How do you prove that but for a delay in the

diagnosis and surgical treatment of a malignant breast tumor, the patient probably would have been cured?

II. WHAT CONSTITUTES A CURE?

Before we can address the problem of proving the element of causation in breast cancer cases some focus should be directed in regard to the concept of "cure." In breast cancer cases, the definition of "cure" may depend upon the context in which it is being used, and who is using it. In the medical community, the most common concept of cure among physicians is that of the *clinical cure*. The *clinical cure* for an individual refers to the complete elimination of the disease. When this definition is applied to a group, a *clinical cure* is considered to have occurred when the long-term follow up of the causes of death reveals that the risk of dying from breast cancer is the same for all women of the same age in the general population.[1] Since there is a tremendous amount of unreliable information recorded on death certificates, the concept of a *clinical cure* is not legally relevant in medical negligence cases, under Rule 401 and Rule 702 of the Texas Rules of Evidence; and *Merrill Dow Pharm., Inc. v. Havner*, 953 S.W.2d 706, 712 (Tex. 1997).

The concept of cure when discussed with individual patients is that of a *personal cure*, which refers to an individual patient living symptom free from breast cancer and dying from another cause. The concept of a *personal cure* is also not relevant or acceptable in legal cases.[2]

The commonly accepted concept of cure utilized in legal proceedings is that of the *statistical cure*. Statistical sampling is the generally accepted method of assessing an outcome of cancer treatment in legal cases. Where a group of treated patients can be considered to be

[1] *Breast Cancer: Controversies in Management*, Futura Publishing Company, Inc., (1994), @ page 75.
[2] *Id.*

statistically cured is when their subsequent death rate from all causes is similar to that of the normal demographic population.[3]

III. CANCER CONTROL WINDOW

In attempting to prove that a patient's loss of a "statistical cure" resulted from a delay in diagnosis, success will depend on the duration of the *cancer control window.*[4] The *cancer control window* is the interval between the time a cancer reaches the size that it first becomes diagnosable if appropriate radiographic tests and clinical observations are performed and the time when the cancer becomes invasive and is no longer regional and presumably curable. A *positive cancer control window* occurs when the cancer was detectable before it metastasized and therefore the delay in identifying the cancer resulted in the loss of a surgical cure. When a *negative cancer control window* occurs the cancer was already metastatic and therefore largely incurable before it developed to a threshold size that would be discoverable by a physician exercising ordinary care.[5] If the evidence establishes a *negative cancer control window*, causation cannot be established.

IV. FACTORS AFFECTING THE CANCER CONTROL WINDOW

1. **Tumor Size**. There are many features of breast tumors that relate to the disease's prognosis, and therefore the legal concept of causation. One of those factors is that of tumor size. Generally speaking, the smaller the tumor, the better the prognosis. For all breast cancers, both *in situ* and invasive carcinomas, no tumor less than five millimeters (5 mm) was palpable on manual examination.[6] While a five millimeter (5 mm) tumor (the size of a B-B) can be easily missed by even the most experienced clinician, a reasonable and prudent physician should be

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[3] *Breast Cancer: Controversies in Management*, Futura Publishing Company, Inc., (1994), @ page 75.
[4] *Id.*
[5] D. Reintgen, *The Anatomy of Missed Breast Cancers*, Surgical Oncology, 1993; 2 @ page 66.
[6] *Id* @ page 67.

able to palpate and identify invasive carcinomas with a tumor size of eleven millimeters (11 mm) or greater. For all forms of breast cancer, no physician should ever fail to identify a ductal carcinoma sixteen millimeters (16 mm or the size of a large marble) in diameter. In a recent study only 58% of all breast cancers in the series were palpable with 19% of the *in situ* cancers and 67% of the invasive cancers palpable. When the breast lesion was sixteen millimeters (16 mm) or larger virtually all breast tumors could be identified by palpation.[7] Mammography can identify some tumors smaller than five millimeters (5 mm).

Tumor size is an important factor in breast cancer staging. Tumors of twenty millimeters (20 mm) or less are considered to be classified as T1 tumors, which in the absence of nodal involvement or distant metastasis, have a five-year survival rate of greater than 85% and 70% at ten years.[8]

The larger the tumor the more likely that disease will be found in the lymph nodes; and generally, the larger the tumor the more nodes will be involved.

2. **Nodal Involvement.** Once a carcinoma moves from *in situ*, its first stop is usually the axillary lymph nodes. This is the most common route of regional spread. The relationship between positive axillary lymph nodes and the risk of breast cancer recurrence at five years is significant. When no axillary lymph nodes are found to contain cancerous cells, the risk for breast cancer recurrence is **less than 10%.** When more than ten positive axillary lymph nodes are found with disease, the risk of breast cancer occurrence is **greater than 50%.**

Recent developments in breast cancer investigation involves *lymphatic mapping and sentinel node biopsy.*[9] Lymphatic mapping is an alternative to the blind sampling of multiple axillary lymph nodes, which may actually miss the positive axillary lymph nodes. Full axillary

[7] D. Reintgen, *The Anatomy of Missed Breast Cancers*, Surgical Oncology, 1993; 2 @ page 68.
[8] Current: Medical Diagnosis and Treatment 2000, 39th Edition, Lange Medical Books (2000), @ page 719.
[9] Brunner & Suddarth's, "Textbook of Medical-Surgical Nursing," 9th Edition (2000), @ page 1276.

dissection frequently causes lymphedema, which is a chronic swelling of the extremity due to an interrupted lymphatic circulation, resulting from the surgical dissection. Sentinel node mapping involves a procedure using a radiopaque dye and nuclear medicine procedures to identify and analyze the first draining lymph node from the breast within the axillary region. Rather than removing all the nodes, the surgeon removes only the first identifiable node and sends it for immediate histological examination. If no cancer cells appear in the sentinel node, the physician does not remove any of the other nodes, and therefore avoids unnecessary surgery with the accompanying risk of lymphedema. Sentinel node mapping has substantially reduced the need for unnecessary, extensive surgery. At the present time, however, if the sentinel node is found to contain tumor cells, all remaining nodes must be removed and investigated. Other sites of lymphatic spread may also include the internal mammary and supraclavicular nodes. Generally speaking, the greater the extent of the spread within the lymphatic system, in other words the more nodes that are involved with disease, the lower a patient's prognosis for a five-year survival.

3. **Distant Metastasis**. Once tumor cells spread beyond the regional lymph nodes, a patient's prognosis for survival becomes virtually impossible. In the staging system set up by the American Joint Committee on Cancer once distant metastasis occurs, a patient's overall five-year survival rate decreases to less than 10% and their ten-year survival to less than 2%. If metastasis is found in the ipsilateral or supraclavicular lymph nodes or in any other organ system, a patient's cancer is not only incurable but it is almost certain that the disease will recur within five years. Distant metastasis can occur in any organ system, but the most common sites are the bone (71%), lungs (69%), liver (65%), pleura (51%), adrenals (49%), skin (30%) and brain (20%).[10]

[10] Brunner & Suddarth's, "Textbook of Medical-Surgical Nursing," supra @ 1272.

4. **Estrogen and Progesterone Receptor Proteins**. Estrogen and progesterone receptor assay is a test used to determine whether the breast lesion is being nourished by estrogen or progesterone. The presence of these hormones provides information useful in making a prognosis and determining a course of treatment for the patient. The presence of both receptor proteins is associated with an improved prognosis. Conversely, the absence of these proteins is associated with a poorer prognosis.[11]

5. **Cell Differentiation**. Breast tumors comprised of tumor cells with a high degree of differentiation are easier for the body's defenses to recognize and attack. Therefore, tumors with a high degree of differentiation are associated with a better prognosis than those with poorly differentiated cells found in antiplastic tumors.

6. **Assessment of the S-Phase Fraction and DNA**. The assessment of a tumor's prolific rate (S-Phase Fraction) and DNA content (ploidy) by laboratory investigation provide some clinical evidence relative to the tumor's prognosis. How significant these factors may actually be is still the subject of significant medical research. Generally, however, tumors classified as diploid (normal DNA content) are associated with a better prognosis for survivability than are tumors classified as aneuploid (abnormal DNA content). The extent to which DNA content may actually impact a patient's survivability in the absence of other factors such as tumor size and regional spread metastasis is not clear at this time.[12]

7. **Heredity**. By 1996, medical researchers determined that the existence of inherited genetic mutations resulted in an increased risk of breast cancer. A woman who inherited a mutation in the BRCA-1 or BRCA-2 gene, may have a breast cancer risk 85% higher than the general population over her life expectancy, in addition to an increased risk of contracting other

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[11] Brunner & Suddarth's, "Textbook of Medical-Surgical Nursing," supra @ 1273.
[12] *Id.* @ 1274.

cancers. BRCA-1 is a gene on chromosome 17 that, when damaged or mutated, is also associated with placing a woman at greater risk for ovarian cancer, as compared with women who do not have this mutation. BRCA-2 is also a gene on chromosome 17 that, is also associated with placing a woman at greater risk for breast cancer than a woman without this mutation, but somewhat less than a woman with a BRCA-1 mutation.[13]

V. CLASSIFICATION OF BREAST CANCERS

Statistical cancer survivability also depends to a great extent on whether the cancer is invasive or noninvasive and whether it is ductile, lobular, medullar, mucinous, or tubular.

A. **Carcinoma *In Situ* (Noninvasive).** With the widespread use of mammography, in situ carcinoma is being diagnosed more frequently. The incidence of in situ carcinomas has increased substantially over the last two decades. *In situ* carcinoma now accounts for 25% of the diagnosed breast cancers, up from less than 10% two decades ago. The disease is characterized by a large number of malignant cells located within the ductals or lobules, without invasion into the surrounding tissue. It is for this reason that it is considered a noninvasive form of cancer and is classified as Stage 0 breast cancer. There are two types of *in situ* carcinoma: ductal and lobular.

1. **Ductal Carcinoma *In Situ* (DCIS).** DCIS is more common than lobular carcinoma *in situ*. It has the capability of progressing to an invasive form so the most traditional treatment is a total or simple mastectomy resulting in a statistical cure rate of 99%. With localized lesions, lumpectomy followed by radiation is an appropriate protocol. Most cases now are managed with breast conservation therapy involving limited surgery and radiation.[14]

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[13] Kerry Harwood, R.N., "Straight Talk About Breast Cancer," *Nursing*, October 1996, @ page 42.
[14] Brunner & Suddarth's, "Textbook of Medical-Surgical Nursing," supra @ 1260.

2. **Lobular Carcinoma In Situ (LCIS).** LCIS is characterized by proliferation of cells within the breast lobules. In many instances this is an incidental finding discovered on pathological evaluation of a breast biopsy for a breast change noted during a physical examination or on screening mammography. LCIS is commonly associated with multicentric disease, and is rarely associated with an invasive form of cancer. Current thinking, however, is that LCIS is **a marker** of an increased risk for the development of an invasive cancer rather than an actual malignancy. This concept has changed the approach for dealing with LCIS. Long-term surveillance is now an appropriate option, since LCIS may not be a true carcinoma itself.[15]

B. **Invasive Carcinoma.** As the phrase implies, this disease is characterized by the spread of malignant cancer cells from the breast to other structures.

1. **Infiltrating Ductal Carcinoma.** Infiltrating ductal carcinoma is the most common histologic type and accounts for 75% of all breast cancers. These tumors are notable because of their hardness on palpation. These tumors readily metastasize through the lymph system, and therefore prognosis is poorer than for other types of cancer.[16]

2. **Infiltration Lobular Carcinoma.** This form of carcinoma accounts for less than 10% of breast cancers. It typically occurs as an ill-defined thickening in the breast and does not present with a hardness on palpation. Clinical identification is difficult. Infiltration lobular carcinomas are most often multicentric; that is, several areas of thickening may occur in one or both breasts. Infiltrating lobular carcinoma is usually spread to the bone, lung, liver, or brain.[17]

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[15] Brunner & Suddarth's, "Textbook of Medical-Surgical Nursing," supra @ 1260.
[16] *Id.* @ 1269.
[17] *Id.*

3. **Medullary Carcinoma**. Medullary carcinomas constitute less than 6% of the breast cancers. These tumors are often very large making their clinical diagnosis easier, and their prognosis is relatively favorable as compared with infiltrating ductal or lobular carcinomas.[18]

4. **Mucinous Carcinoma**. Mucinous carcinoma accounts for less than 3% of breast cancers. It is slow growing and therefore has a more favorable prognosis than many of the other infiltrating types.[19]

5. **Tubular Ductal Carcinoma**. Less than 1% of all cancers are found to be the tubular ductal cancer. Axillary metastasis are uncommon and prognosis is excellent.[20]

VI. TUMOR STAGING

In order to arrive at a uniform system for classifying malignant tumors, including those found in the breast, for the purpose of arriving at a prognosis for survivability, the American Joint Committee on Cancer and the International Union Against Cancer developed the TNM Staging System.[21] The TNM Staging System was originally created to provide a uniform staging system to enhance the accuracy of communications between investigators and clinicians. A copy of this TNM classification may be found as an attachment to this article. In arriving at the staging system, the committee recognized that there were other factors that impacted a patient's survivability that would not be included in analysis of tumor size, nodal involvement or distant metastasis, but felt that for the purposes of arriving at a reasonable prognosis for statistical survivability, the three primary factors that would need to be considered, tumor, nodes and metastasis would be incorporated into this classification.[22] In attempting to prove a client's loss of survivability during the cancer control window, the evidentiary starting point must include the

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[18] Brunner & Suddarth's, "Textbook of Medical-Surgical Nursing," supra @ 1269.
[19] *Id.*
[20] *Id.*
[21] AJCC Cancer Staging Manual, 5th Edition, Lippincott-Raven, 1997.
[22] Current: Medical Diagnosis and Treatment 2000, 39th Edition, Lange Medical Books (2000), @ page 709.

TNM classification. The questions that must be answered: What was the TNM classification when the tumor was at a threshold size that a diagnosis should have been made compared to the TNM classification when the tumor was actually diagnosed and biopsied? From this point, other factors such as doubling time, estrogen and progesterone receptor assays, S-Phase Fraction, and DNA content will need to be determined.

VII. RISK FACTORS FOR BREAST CANCER

Even when a physician is presented with clinical or mammography evidence of a tumor and misdiagnoses the lesion as benign, evidence may show that even though the threshold size of the lesion permitted its timely diagnosis, the patient nevertheless had no risk factors for breast cancer and therefore, the index of suspicion for malignant breast disease, was very low. This delay in diagnosis may occur because the lesion's characteristics did not manifest themselves until the tumor was of a size that made metastasis almost a virtual certainty.

Although benign lesions and fibrosistic breast changes certainly increase the difficulty that physicians face in identifying malignant from benign lesions, there are certain identifiable risk factors which should increase a physician's index of suspicion that his patient may be at a higher risk for breast cancer and therefore, increased vigilance must be utilized in investigating breast abnormalities. While there are no absolute causes for breast cancer, medical literature has identified clusters of risk factors associated with the development of breast malignancies. The following risk factors have generally been referred to in the medical literature including the 9th Edition of Brunner & Suddarth's, "Textbook on Medical-Surgical Nursing" published this year:

A. **Probable Risk Factors**

1. **BRCA-1 or BRCA-2 Genetic Mutation**. Women with this chromosome 17 genetic mutation have a greater than 85% risk of developing breast cancer in their lifetime and approximately 50% chance of developing breast cancer before age 50.

2. **Hormone Replacement Therapy**. Women who are taking estrogen supplements for menopause for lengthy period of times, are at some increased risk for the development of breast malignancies. While the addition of estrogen and progesterone as a hormone supplement may decrease the occurrence of endometrial cancer, it does not appear from the literature to decrease the risk of breast cancer.

3. **Family History of Breast Cancer**. If a woman's mother was diagnosed with breast cancer before the age of 60 or she had a sister or daughter with breast cancer, her chance of developing a breast malignancy increases substantially.

4. **Personal History of Breast Cancer**. If a woman has already had breast cancer, the risk of developing breast cancer again increases by approximately 1% per year. This means if the woman was at increased risk for breast cancer to begin with, such as having a chromosome 17 BRCA-1 genetic mutation, she is virtually certain to develop breast cancer again.

5. **Early Menarche**. If a woman menses began before age of 12, she has an increased risk for breast cancer.

6. **Late Menopause**. Women who do not experience menopause until after 55 years of age are at an increased risk for developing a breast malignancy.

7. **Pregnancy After Thirty**. Women who do not give birth to their first child until after thirty years of age, seem to have nearly twice the risk of developing breast cancer than women who gave birth to their first child before twenty years of age.

8. **Benign Prolific Breast Disease**. Women with a history of benign tumors or with significant epithelial changes are at a greater risk for developing breast malignancy than those women with no such tumors. Atypical hyperplasia or LCIS may also be an indication of a precancerous condition increasing the woman's risk of developing a breast malignancy at a later date.

9. **Radiation Exposure**. A woman who has been exposed to ionizing radiation between puberty and thirty years of age has twice the risk of developing malignant breast cells, than an individual that is not exposed to radiation or exposed at a later time in life.

In addition to the risk factors for which there appears to be some agreement within the medical literature, there are also some factors that are considered controversial in regard to their association with an increased risk for breast cancer:

B. **Possible Risk Factors**

1. **Obesity**. Obesity was once thought to increase a woman's risk of developing breast cancer, primarily in post-menopausal women. The theory was that estrogen was stored in the body's adipose tissue and because dietary fat increases pituitary prolactin, there was an increased estrogen production and therefore an increased risk of developing breast tumors. Obese women diagnosed with breast cancer have a higher mortality rate which was originally thought to be the result of these hormonal influences; however, recent studies have indicated this may be nothing more than lead-time bias resulting from the difficulty in diagnosing or identifying tumors in patients with large breasts.

2. **High-Fat Diet**. A high-fat diet was once thought to increase the risk of breast cancer. Epidemiologic studies of American and Japanese women seem to show a five-fold difference between the rate of breast cancer between the two groups, with the American women having the

greater incidence of breast malignancy. Japanese women who migrated to the United States were shown to have breast cancer rates similar to their U. S. citizen counterparts. Recent cohort studies, however, show only a weak or inclusive relationship between a high-fat diet and breast cancer. While a high-fat diet may be implicated in colon cancer and heart disease, its relationship to breast cancer is still controversial.

3. **Alcohol Intake**. Alcohol intake also has not been conclusively shown to increase a woman's risk of breast cancer. Although studies show a slight increase in women who consume one drink a day and a greater increase among woman averaging three drinks daily, in countries where wine is consumed daily, such as France or Italy, the increase does not appear to be statistically significant. There is some research that suggests young women who drink alcohol may be more vulnerable to breast malignancies at old age, but this study is inconclusive and alcohol intake remains controversial in regards to its association with an increased risk for developing breast cancer.

VIII. BACKWARD EXTRAPOLATION OF TUMOR SIZE

Attempting to determine the preclinical size of a malignant breast tumor when there is a suspected delay in its diagnosis requires either a backward extrapolation from the clinical observation or the use of statistical tumor models. Both of these techniques can be used, but have significant limitations. Usually, tumors which are of sufficient size to be identified and measured clinically, are likely to have a slower growth rate than the microscopic preclinical lesions. Some studies on the relative growth rates of human breast tumors indicate that these tumors have a longer *doubling time* than other human tumors.[23] A tumors *doubling time* is defined as the time it takes for a tumor to double in size, and is based upon three dimensionable measurements. The *doubling time* for breast cancer in its earliest identifiable clinical phases has

[23] *Breast Cancer: Controversies in Management*, Futura Publishing Company, Inc., (1994), @ page 74.

been determined in some studies based upon measurements of lesions present, but not initially appreciated as cancer on serial mammograms.[24] The observed *doubling time* in some studies average between 115 and 325 days for clinical breast tumors, but the range of *doubling times* in individual patients varied from between 23 days to more than 940 days.[25] The average *doubling time* for breast tumors seems to range from between 220 days and 252 days.[26] These figures are statistical and therefore obviously hypothetical.

In attempting to extrapolate backward from clinical observations to determine the size of a preclinical tumor, it is usually assumed that the preclinical growth of breast cancer is both logarithmic and continuous. This presumption is probably not accurate. Tumors that can be identified through palpation that are 10 mm in diameter are thought to contain approximately 109 cells. If you assume that the origin of the tumor is a single-cell mutation, then statistically it would take more than 30 doublings for a single malignant cell to produce 109 cells, and that would require an additional assumption that no cell loss occurred during this interval. Assuming this hypothetical is accurate, and if one accepts that the preclinical *doubling time* occurs approximately every 100 days, then in theory, the preclinical phase of breast cancer should exceed **ten years**. In other words, the preclinical tumor was growing at a logarithmic rate doubling every 100 days for ten years before it first reached a clinically identifiable size. If this assumption is accepted as accurate, then it would be impossible to prove that during a tumor's ten year existence no cancer cells were spread and no micrometastasis occurred. Of course, this hypothetical growth is subject to significant challenge in the medical research community and significantly underestimates the difference in growth rates which occur in preclinical lesions. It is generally thought that a preclinical lesion may grow logarithmically whereas a clinically

[24] *Breast Cancer: Controversies in Management*, Futura Publishing Company, Inc., (1994), @ page 74.
[25] *Id.*
[26] *Id.* and *Cancer of the Breasts*, 4th Edition, W. D. Sanders Company (1955), @ page 329.

detectable lesion increases in size at a much slower rate during the plateau phase of growth.[27] It is also probable that during the preclinical growth of a breast tumor, cancer cells are lost or destroyed and the growth rate is significantly less than logarithmic and is probably discontinuous. The preclinical phase of growth could be much longer, or much shorter. It has also been observed that both the clinical and preclinical phase of growth of a breast tumor should be affected by the same environmental stimuli that effect the growth and development of all human cells. A patient's age, nutrition, wellness, and other environmental factors presumably have the same influence on the growth and development of cancer cells as those factors have on other human cell development.

The use of *doubling times*, alone, in predicting preclinical tumor size is inaccurate and unreliable, and subject to challenge under *Daubert, Robinson* and *Kumho Tire Co.* Other factors must also be relied upon if one is to accurately predict whether a preclinical identification of a tumor could have been made before regional and distant metastisis occurs.

IX. CONCLUSION

It is apparent from numerous research and medical studies on breast cancer that some tumors evade diagnosis by either physical examination or mammography at a defined rate in the population. Under the best of circumstances, trained physicians with good diagnostic skills who are able to arrive at a good correlation between mammographic abnormalities and a clinical physical exam, may still misidentify breast tumors smaller than 1.6 cm in size. Some studies have identified mammographies defined rate of misinterpretation as 1.8%.[28] When the number of interval breast cancers and false negative examinations are added to the study, the error rate

[27] *Breast Cancer: Controversies in Management*, Future Publishing Company, Inc., (1994) @ page 74.
[28] D. Reintgen, *The Anatomy of Missed Breast Cancers*, Surgical Oncology, 1993; 2: @ pages 65-75.

increases to 7%.[29] In either case, statistics indicate that well-trained, competent physicians who are attentive to their patients will probably be able to identify even very small tumors. The prudent physician, following the appropriate and recommended standards of care, may occasionally fail to identify a malignant tumor, since a small percentage are truly undetectable on physical examination and mammography, or the patient may truly develop the cancer during the interval between screening examinations. With the prudent, well-trained physician, these failures should be extremely rare. In some cases, even with timely diagnosis, some breast cancers may also have a poor outcome with nodal metastasis and resulting diminished survival rate based primarily on a biological aggressiveness of the tumor itself. This occurrence is also rare, and its effects minimized by the early, timely diagnosis obtained by a competent, prudent physician. While there are many factors that determine a patient's ultimate survival from breast cancer, the one factor uniquely iatrogenic in nature is the failure to provide a patient with a timely and accurate diagnosis of a malignant breast tumor. The failure to make an accurate, early diagnosis of a breast malignancy is still the most important factor in establishing the element of causation in breast cancer cases.

[29] D. Reintgen, *The Anatomy of Missed Breast Cancers*, Surgical Oncology, 1993; 2: @ pages 74.

November 2-3, 2000

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