CHAPTER ONE

1.1 Introduction:

As there is no menstrual cycle, the sequential changes in blood flow to the ovary seen during the reproductive years are generally not demonstrated in the normal post-menopausal patient.(O'Brien WF, 1984)

These cyclical changes, however, may be evident if the patient is on hormone replacement therapy. In fact, a pre-menopausal ovarian blood flow pattern in a post-menopausal patient should prompt the search for a history of hormone replacement therapy (although there are other differential diagnostic possibilities).

In the post-menopausal patient (as with the pre-menopausal patient), one of the most important uses of ultrasound involves the diagnosis and characterization of adnexal masses.

To diagnose the presence of an adnexal mass, gray-scale ultrasound is of substantial importance. This is especially true as the post-menopausal ovary may not be reliably palpated. In one study, 10% of masses less than 10 cm in size were missed on palpation. Transvaginal sonography is generally more sensitive than transabdominal. Some investigators feel that the absence of an adnexal mass on gray-scale examination may not rule-out the presence of a malignancy, while others presume that it does.

Interestingly, many post-menopausal women (10% - 15%) continue to demonstrate cysts within their ovaries. Ovarian cancers may manifest their presence as post-menopausal cysts. In fact, 80% of ovarian neoplasms occur in women older than 50 years of age and of these as much as 85% - 90% are of a cystic epithelial type. Also, some patients clinically diagnosed as "post-menopausal" are, in reality, peri-menopausal and continue to "cycle", though
irregularly. This latter differential diagnostic possibility can generally be confirmed clinically, biochemically or by follow-up ultrasound examination. For characterizing a discovered mass, gray-scale and Doppler (both duplex and color) ultrasound, may be of significant utility in distinguishing benign from malignant processes.

Gray-scale sonography has been used with varying success to characterize adnexal disease. Findings such as septations, papillary projections and mural nodules are more likely to be associated with malignant changes than are clear cystic masses. Also, size may be important. (Rulin and Preston; 1986) found that masses less than 5.0 cm were unlikely to be malignant. In their series, only one case of a cystic ovarian mass in 32 (3%) was malignant. In a study by (Goldstein et al, 1989) the results of sonographically detected simple cysts (defined as cysts without internal septations or solid components) of the adnexa, yielded a 0% incidence of malignancy in patients with cysts less than 5.0 cm in maximum diameter. (Hall and McCarthy, 1990) included septated cysts in their definition of "simple cyst." They found an 8% malignancy rate in their series of cysts ranging from 1.5 to 10.0 cm. The malignancy was within a 3.5 cm non-septated cyst.

Some investigators (Dickey et al; 1994) have concluded that both color and duplex Doppler are valuable tools for the diagnosis of ovarian and other malignancies. These conclusions are based upon the unique differences between normal and tumoral vascularity (neovascularity) and the apparent ability of Doppler sonography to distinguish between them. Tumor vessels are disorganized. The standard hierarchical organization of normal vessels, in which flow progresses from arteries of decreasing size through capillaries to veins of increasing size, is absent. Instead, tumoral
flow may be short-circuited through shunts. Also, tumor vessels may possess altered architecture.
Abnormal flow patterns can be demonstrated in vessels surrounding malignant masses. Finding these areas of neovascularity may not be possible on gray-scale examination only. Because color Doppler may make these vessels visible, it allows the examiner to survey the anatomy of the target structure for vascular areas of interest.
There is substantial data to suggest that the flow characteristics of some malignant diseases of the ovary are different from benign processes. In general, a low resistance pattern is unusual in the ovary of a post-menopausal patient, as are low index values, and may be associated with malignancy. False positives do occur. The absence of neovascularity and the presence of a normal index value has been shown to exclude malignancy, while the presence of neovascularity had a high association with malignant change.
When performing a Doppler ultrasound for adnexal masses, it should be performed between days 3-10 of the menstrual cycle in menstruating patients; between days 3-10 in post-menopausal patients on hormone replacement therapy; at any time in post-menopausal patients not on replacement.( AMJ Roentgenol,1995)
First a personal note. Few diseases are as evil as cancer. And among the cancers, perhaps none is as evil as ovarian cancer. It is the number one gynecologic malignancy responsible for killing women. It is usually silent until it is too late to effectively treat. Early ovarian cancer detection is both a medical and political hot potato. Medically, there is no agreement by all authorities as to how to diagnose this disease early. Politically, most "screening" procedures are considered too expensive to subsidize. As with
other diseases similarly situated, the end result is the death of some who may have been saved. Personally.

For a woman past her reproductive years, changes in the sonographic appearance of the pelvic organs still occurs, albeit, more slowly and less dramatically than in the pre-menopausal woman.

1.2. Problem of the study

Endometrial changes may occur. In some cases the endometrium, becomes too thick. It is not cancer, but endometrial thickening usually occurs after menopause, when ovulation stops and progesterone is no longer made. It also can occur during perimenopause, when ovulation may not occur regularly in some cases, it can lead to cancer of the uterus. Changes may happened with or without vaginal bleeding or symptoms.

In women without vaginal bleeding, the threshold separating normal from abnormally thickened endometrium is not known. The purpose of performing ultrasound in non-bleeding postmenopausal women may be to investigate abdominal pain or masses or to delineate the adnexae when pelvic examination is inadequate. Screening studies have been undertaken to assess whether ultrasound can detect endometrial cancer in large populations of asymptomatic women.

1.3. Objective of the study

1.3.1 General objective

To Characterize the Endometrium in a Postmenopausal Asymptomatic Patients without Bleeding. In order To formulate clinical recommendations for the assessment of endometria

1.3.2 Specific objective

To evaluate the uterus echogenicity and texture

To measure the endometrium thickness.

To evaluate the endometrium echogenicity and texture
To correlate the findings with female age and the age at menopause, early age when menstruation started, number of pregnancy.

To correlate the findings with Personal history of certain conditions, such as diabetes mellitus, polycystic ovary syndrome, gallbladder disease, or thyroid disease, Obesity, Cigarette smoking, hormonal therapy at any period of life, Family history of ovarian, colon, or uterine cancer.

1.4 Significance of the study

Vaginosonographic examination has become an examination technique that is accepted by postmenopausal women in particular and which gives the less experienced examiner an overall view of the internal genital tract in the female. Recent advantages of transvaginal color Doppler and three-dimensional ultrasound enables the more experienced examiner to visualize even the smallest vessels and investigate blood flow characteristics in the poorly perfused small pelvis in the postmenopause, what helps him to differentiate between the normal, suspicious and pathologic variations of the structures or detect and follow the effects the hormone replacement therapy on the perfusion of genital tract.

1.5 Overview of study

This study consisted of five chapters. Chapter one is an introduction which includes; problem and objective of the study. Chapter two is a literature review which includes; Anatomy, Physiology, Pathology and previous studies. Chapter three is about research methodology. In Chapter four the results are presented and Chapter five includes; discussion, conclusions and recommendations.
CHAPTER 2

2-1 Anatomy

The uterus is located in the lesser pelvis between the urinary bladder and the rectum. Although generally a midline structure, lateral deviations of the uterus are not uncommon. The broad ligaments extend from the uterus laterally to the pelvic side walls. They contain the fallopian tubes and vessels. The uterosacral ligaments serve to keep the uterus in an anterior position. They arise from the upper cervix posteriorly and extend to the fascia over the second and third sacral vertebrae. The round ligaments arise anterior to and below the fallopian tubes and cross the inguinal canal to end in the upper portion of the labia majora.

The normal adult uterus measures approximately 7.0 - 9.0 cm long, 4.5 - 6.0 cm wide and 2.5 - 3.5 cm deep (anteroposterior dimension). Its corpus-to-cervix ratio is 2:1. (AJR, AMJ Ronengenol, 1986)

During the post-menopausal years of a normal female, the uterus decreases in size and the endometrium atrophies. (Springer ,1991)

As the ovaries undergo involution, there is an associated reduction in the amount of estrogen produced. This leads to the gradual atrophy and involution of the endometrium that characterizes the uterine lining of the post-menopausal patient. In asymptomatic post-menopausal women, the mean endometrial thickness has been determined to be 3.2 +/- 0.7 mm, although other investigators have reached different conclusions. (obset gnyecol 1991)

Gray-scale examination generally reveals an inverse relationship between uterine size and the time since menopause: uterine size and volume progressively as the duration of the post-menopausal period increases. The greatest changes occur within the first ten years after the menopause and more gradually thereafter. (Jelin A ,1981)
Just how big is the post-menopausal uterus? The post-menopausal uterus has been measured at 8.0 +/- 1.2 cm long by 5.0 +/- 0.8 cm wide by 3.2 +/- 0.7 cm deep (anteroposterior dimension). A significant relationship between parity and both uterine volume and weight was not found. Others have reached different conclusions. (Kurjak and Zalud 1990)

As there is no menstrual cycle, successive changes in blood flow to the uterus are generally not demonstrated. However, some similarities between pre- and post-menopausal women may be present. (Kurjak and Zalud 1990) compared the Resistance Index values of the uterine arteries in pre- and post-menopausal women. The RI was noted to be higher in the post-menopausal patients, but apparently not statistically different. Diastolic flow was demonstrated in all subjects. (Kurjak and Zalud 1990)

If the patient is on hormone replacement therapy, the above-described findings may not be present. Among these patients, both uterine size and cyclical endometrial changes may remain. Even the corpus-to-cervix ratio approximates the pre-menopausal state. In general, estrogen therapy affects the post-menopausal endometrium similarly to estrogens in the normal cycle. The conjugated estrogens have a proliferative effect. Progestational therapy may cause the endometrium to respond with a quiescent appearance that is characteristic of the normal secretory endometrium. When used together with exogenous estrogens, synthetic progestogens reproduce the characteristic biochemical and morphological changes seen in the secretory phase of the normal menstrual cycle.

(Bourne et al; 1990) have shown altered blood flow to the uterus in patients receiving hormone replacement therapy. Using endovaginal technique, significant arterial changes were demonstrated. The PI was reduced by 50% within 6-10 weeks of initiating therapy. Also, endometrial thickness almost
doubled. Before treatment, the mean thickness was 0.37 +/- 0.08 cm. Following treatment, the values were 0.68 +/- 0.13 cm. Others have reported a possible 2-3 mm increase in endometrial thickness in patients receiving estrogen therapy. But some have obtained results different from the above. (Wolf et al, 1991) Their data suggest no statistically significant differences between the endometrial thicknesses of patients taking and patients not taking hormones.

Thus, a pre-menopausal appearing uterus or uterine blood flow pattern in a post-menopausal woman should prompt the search for a history of hormone replacement therapy (although there are other differential diagnostic possibilities). (Bourne TH and Campbell 1991)

In the post-menopausal patient, one of the most important uses of ultrasound involves the diagnosis and management of endometrial cancer. In general, endovaginal ultrasound is superior than transabdominal ultrasound for visualization of the myometrium and endometrium. (Fleischer, 1990)

Sonographic signs of endometrial cancer in the post-menopausal patient include: an obstructed fluid-filled canal, a thickened uterine cavity, an enlarged uterus and a lobular uterus with a mixed echo pattern. (Chamber eb 1986) Gray-scale ultrasound has accurately demonstrated the presence and extent of myometrial invasion. (Cacciatore et al 1998) using transabdominal technique, found that sonographic staging of endometrial cancer by ultrasound was accurate in 91% of cases and myometrial invasion was correctly identified in 80% of cases. Transvaginally, (Cruickshank et al; 1989) demonstrated good agreement between the ultrasound examination and myometrial invasion as determined microscopically. These investigators suggested that more accurate pre-operative diagnosis may allow for selective therapies, perhaps yielding better results. (Granberg et al, 1988) using
transvaginal technique, demonstrated that with a full-thickness endometrial measurement of 8 mm. or less, in patients with post-menopausal bleeding, no endometrial cancer was discovered by curettage. In general, the data would seem to indicate that a full-thickness post-menopausal endometrium of 10 mm or greater should be further evaluated, either by biopsy or D&C, to exclude either malignancy or hyperplasia. (Amj obsetgynecol 1990)

Some investigators have demonstrated the utility of Doppler ultrasound in diagnosing endometrial cancer. (Bourne et al, 1989) demonstrated increased blood flow in the uterine artery and the area of the suspected tumor in patients with malignant disease. One group appears to have substantial experience in evaluating pelvic masses and distinguishing benign from malignant disease (Kurjak A et al). It is their conclusion that abnormal blood flow can be identified in virtually all cases of endometrial carcinoma, as well as uterine sarcomas. With color Doppler, the abnormal findings include the presence of irregular, thin and randomly distributed vessels. Abnormal flow velocity waveform patterns have also been described. (Hata T et al 1989)
figure (2-1) Female Reproductive Anatomy

figure 2-2 Fallopian Tube Anatomy
Figure 2-3. (a) Human female reproductive system: lateral view (b) female reproductive system: anterior view
Figure 2-4 Anteverted and Retroverted uterus
Figure 2-5 Anteflexed and Retroflexed uterus
2-2 The Physiology of menopause

As a five-month old fetus, the human ovary has an estimated two million follicles that house and develop her eggs. From then to puberty the number of follicles drops to about three hundred thousand. Starting at puberty each month about 20 eggs begin maturing but usually only one of those eggs is actually released and made available for conception. During her reproductive years the average woman will ovulate about 500 times. The rest of her follicles gradually shrink in size causing a decrease in ovarian mass from 14 grams before menopause to about 5 grams after. Fertility peaks at age 24 and starts declining at age 35, long before irregular periods begin.

Figure 2-6 uterus Arterial Supply

2-2-1 The Normal Menstrual Cycle

In order to understand what happens to the body at menopause, it is necessary to review the basic physiology of the normal menstrual cycle. Please refer to Figure 2-6 for clarification. During the normal cycle the pituitary gland secretes two messenger hormones that communicate with the ovaries in order to stimulate ovulation. The first of these hormones is called "follicle stimulating hormone" (FSH) and it communicates estrogen needs to
the ovaries. The second messenger hormone, "luteinizing hormone" (LH), regulates the release of progesterone. (Brunner RL et al 1986)

Figure 2-2

![Normal Menstrual Cycle](image)

**Figure 2-7 Normal Menstrual cycle**

The first phase of the menstrual cycle (day 0 to approximately day 14) is known as the "follicular phase." During this time the release of FSH from the pituitary gland into the bloodstream signals the ovaries to develop several egg-containing follicles. At this time the ovaries are also signaled to secrete increasing amounts of estrogen until one of the follicles reaches the surface of the ovary. As the estrogen levels reach the required amount, the pituitary is signaled to stop production of FSH and to release LH, which travels to the ovaries and causes the follicle to burst, thus releasing its egg.

The second phase of the normal menstrual cycle is known as the "luteal phase" and covers approximately day 14 to day 28. During this phase estrogen levels drop off and progesterone levels increase. If fertilization does not occur, the levels of both estrogen and progesterone drop off toward the end of the cycle and menstruation occurs.

In the normal menstrual cycle a delicate balance between estrogen and progesterone occurs. This balance regulates many of the body's systems:
e.g., fluid balance, mood, sex drive, endometrial growth, appetite, skin, bone density and fat distribution.

Figure 2-8 Normal Endometrium

Figure 2-9 Early Proliferative Phase
Figure 2-10 Late Proliferative Phase

Figure 2-11 Secretory Phase
Figure 2-12 Menstrual Cycle

Figure 2-13 Menstrual Cycle Hormone Levels
The Perimenopausal Menstrual Cycle

During the perimenopausal phase the delicate balance between estrogen and progesterone is upset. This imbalance is what causes most of the symptoms associated with the perimenopause. During this time the ovaries do not respond to FSH stimulation by increasing estrogen production as they do in the normal cycle. As a result the pituitary continues to produce FSH in order to stimulate estrogen production. This causes a lengthening of the follicular phase of the cycle that results in longer cycles. Elevated serum FSH levels and longer cycles are two signs that a woman has entered the perimenopause. Eventually, when enough estrogen has been released to cause follicular rupture, the LH phase of the cycle occurs. During the perimenopause, ovarian estrogen production diminishes to a point at which there is not enough estrogen to cause the LH release. As a result menstruation ceases. Once a woman has gone for an entire year without menstruating she is declared menopausal. (Broun JB 1985; Br Med J 1976)
Many of the alternatives for the management of perimenopausal symptoms are based on countering the effects of the body's declining estrogen levels by stimulating estrogen receptors, the molecules that bring the hormone into its target cell. Most of the body's cells have estrogen receptors on them. As a result, the decreasing amount of ovarian estrogen that characterizes the perimenopause, affects as many as 300 different body parts. About 75% of women experience some symptoms associated with these effects (Collet et al 1954; Doring GR 1996; Kaufert PA 1986). There are at least two chemically different estrogen receptors; they are known as alpha and beta receptors. In general the alpha receptors are involved in the reproductive system while the beta receptors are active in other body tissues. To complicate things even further, many receptors are able to pair up with molecules that are chemically similar to estrogen. Some of these pairings result in estrogen-like activity in the cell while others do
not. In many cases, these pairings serve to block estrogen activity by filling up the receptors so that estrogen molecules cannot attach.

2-2-4 Uterine Changes

Uterine changes occurring during the perimenopausal years include irregular bleeding, prolonged bleeding and irregular ovulation (which can make a woman uncertain as to whether she has missed a period due to the perimenopause or because she is pregnant). When any of these symptoms occur it is recommended that the woman see her health care provider in order to rule out serious diseases associated with these symptoms such as uterine cancer, uterine fibroids and cervical cancer. (Georgiou E et al 2011)

2-3 PATHOLOGY

2-3-1 Endometrium thickness

In a postmenopausal woman with vaginal bleeding, the risk of cancer is approximately 7.3% if her endometrium is thick (> 5 mm) and < 0.07% if her endometrium is thin (< or = 5 mm). An 11-mm threshold yields a similar separation between those who are at high risk and those who are at low risk for endometrial cancer. In postmenopausal women without vaginal bleeding, the risk of cancer is approximately 6.7% if the endometrium is thick (> 11 mm) and 0.002% if the endometrium is thin (< or = 11 mm). The estimated risk of cancer was sensitive to the percentage of cancer cases that were estimated to occur in women without vaginal bleeding. For the base case we estimated that 15% of cancers occur in women without vaginal bleeding. When we changed the estimate to project that only 5% of cancers occur in women without vaginal bleeding, the projected risk of cancer with a thick measurement was only 2.2%, whereas when we estimated that 20% of endometrial cancers occur in women without bleeding, the projected risk of cancer with a thick measurement was 8.9%. As a woman's age increases, her
risk of cancer increases at each endometrial thickness measurement. For example, using the 11 mm threshold, the risk of cancer associated with a thick endometrium increases from 4.1% at age 50 years to 9.3% at age 79 years. Varying the other estimates used in the decision analysis within plausible ranges had no substantial effect on the results. (Opmeer Lc et al 2004).

Figure 2-16 ET measured in the longitudinal axis at its maximal thickness, including both endometrial layers
2-3-2 Postmenopausal Bleeding:

2-3-2-1 Causes of Postmenopausal Bleeding:

2-3-2-1-1 Polyps: These are growths, usually noncancerous, that can develop in the uterus, on the cervix, or inside the cervical canal. They might cause bleeding. (Lotfallah; Hussan et al 2004)

![Sonogram](image)

Figure 2-17 Sonogram from a patient who presented with postmenopausal bleeding.

The endometrial echo is clearly thickened.

2-3-2-1-2 Endometrial atrophy (thinning of the endometrium): The endometrium, the tissue that lines the uterus, can become very thin after menopause because of lower estrogen levels. This may cause unexpected bleeding. (Collins, Collins J 2007).
2-3-2-1-3 **Endometrial hyperplasia:** In this condition, the lining of the uterus becomes thick, and bleeding may occur as a result. Obesity may be the cause of the problem. Some people with endometrial hyperplasia may have abnormal cells that can lead to endometrial cancer (cancer of the uterine lining). (Lacey JV; Jr Chia VM 2009)
Endometrial hyperplasia

2-3-2-1-4 Endometrial cancer (uterine cancer): Bleeding after menopause can be a sign of endometrial cancer (Lanat 1989)

Figure 2-19 Endometrial hyperplasia

Figure 2-20 A 76-year-old woman with poorly differentiated endometrial adenocarcinoma. Sagittal transvaginal ultrasound image of the uterus shows a central mass.
2-3-2-1-5 Other causes: Hormone therapy, infection of the uterus or cervix, use of certain medications such as blood thinners, and other types of cancer can cause postmenopausal bleeding. (Mohen S; 2007)

2-3-3 Uterine cancer

Cancer of the uterus (uterine cancer) mostly affects women over 50. This cancer is also known as cancer of the womb, uterine cancer, endometrial cancer and cancer of the lining of the womb. Symptoms of uterine cancer may include unusual bleeding and watery, bloody discharge from the vagina. Less common symptoms include bloating and discomfort in the abdomen and pain during sex. (Laufer Mr. 1995; BMJ 1990)

The uterus (womb) is part of the female reproductive system. It is shaped like an upside-down pear and sits inside the pelvis. It is in the uterus that a fertilised egg grows into a baby.

More than 577 Victorian women are diagnosed with cancer of the uterus every year. Most of these women are over the age of 50 years. Cancer of the uterus is also known as cancer of the womb, uterine cancer, endometrial cancer and cancer of the lining of the womb. (Laufer M; 1995)

2-3-3-1 Causes of uterine cancer:

The exact cause of uterine cancer is not known. Some things seem to put women at more risk including:

- endometrial hyperplasia (an abnormal increase in the number of cells in the endometrium)
- menopause, never having children or being infertile
- being overweight
- high blood pressure and diabetes
- a family history of endometrial, breast or bowel cancer
- being on oestrogen hormone therapy without progesterone
being on tamoxifen or anastrozole for treatment of breast cancer.

Most women who have known risk factors do not get cancer of the uterus and many women who do get cancer of the uterus have none of these risk factors.

2-3-3-2 Symptoms of uterine cancer:
The most common symptoms of uterine cancer are:

2-3-3-2-1 unusual bleeding:
watery, bloody discharge from the vagina, which can sometimes be smelly. Less common symptoms include bloating and discomfort in the abdomen and pain during sex. If the cancer is very advanced, other symptoms may be present, such as tiredness, loss of weight and constipation. (Latrakis G et al; 1997).

Unusual bleeding or discharge can happen before and after menopause. It is usually not due to cancer of the uterus. However, all women with unusual bleeding or discharge should see their doctor for a check-up. (Buyuk et al 1998).

2-3-3-2-2 Types of uterine cancer:
Most cases of cancer of the uterus are cancers of the uterus lining (endometrium), though some cancers grow in the muscle layers of the uterus. (Obstet Gynecol ;1991).

The different types of uterine cancer include:

2-3-3-2-2-1 Adenocarcinoma of the endometrium – around 85 per cent of women diagnosed with cancer of the uterus have this form. An adenocarcinoma is a cancer that starts in the glandular tissue. (Smith; Bindman et al 1998)

2-3-3-2-2-2 High-risk cancers – less common types of cancer (such as adenosquamous carcinoma, papillary serous carcinoma and clear cell
cancer (carcinoma) are more likely than others to spread around the body. (Bonilla et al; 1997)

2-3-3-2-2-3 **Endometrial hyperplasia** – sometimes, women develop a thick uterus lining that can cause heavy periods. Some types of endometrial hyperplasia may later become precancerous.(kurjak Z ;1993)

2-3-3-2-3 **Diagnosis of uterine cancer:**

The tests used to diagnose cancer of the uterus include:

2-3-3-2-3-1 **physical examination** – to check the abdomen for swelling

2-3-3-2-3-2 **transvaginal ultrasound** – to look at the size of the ovaries, uterus and thickness of the endometrium.(Kurjak A; Kupesic ;2002; Van nagell;2000)

2-3-3-2-3-3 **biopsy** – removing some tissue so it can be looked at under a microscope. This can be done in several ways including having a dilatation and curettage (D&C)

2-3-3-2-3-4 **x-rays and other scans** – such as computed tomography (CT) scan or magnetic resonance imaging (MRI) (Radiol Clin North Am 1992)

![Axial contrast-enhanced CT image](image)

Figure 2-21 Axial contrast-enhanced CT image shows prominent triangular-shaped endometrium. Number 1 and circle indicate incidental measure of ovarian cyst
2-3-3-2-3-5blood tests – Macroscopi characterization and relation to histological diagnosis. (Granberge S; 1989)

2-3-4Fibroids After Menopause:

Some women who suffer from uterine fibroids – noncancerous tumors that grow in or on the muscle walls of the uterus – put off treatment because they are approaching menopause. While in many instances, fibroids have been shown to shrink after menopause, this may not always be the case. (Adanson GD; 1992)

Although often menopause causes fibroids to shrink, this may not mean that the problems and symptoms associated with fibroids will disappear complete Effects of menopause on fibroids

Since fibroids grow in part due to the amount of estrogen in a woman’s body, the changes in hormones that come with menopause can cause them to stop growing and even shrink. It may not be enough to make the tumors go away altogether.
Another thing to remember is that if a woman is taking hormone replacement therapy to help deal with menopause, the fibroids will not be affected and the woman will likely continue to experience symptoms. Every woman’s body is different though. Menopause may not cause the fibroids to shrink at all, and they may even continue growing. Some women have continued to experience painful symptoms from fibroids after menopause.

Figure 2-23 Here is a normal uterus with fallopian tubes and ovaries from an older postmenopausal woman.
The endometrial cavity is opened to reveal lush fronds of hyperplastic endometrium. Endometrial hyperplasia usually results with conditions of prolonged estrogen excess and can lead to metrorrhagia (uterine bleeding at irregular intervals), menorrhagia (excessive bleeding with menstrual periods), or menometrorrhagia.

2-4 ULTRASOUND

2-4-1 Basic physics Instrumentation of ultrasound:
Diagnostic ultrasound employs pulsed, high frequency sound waves that are reflected back from body tissues and processed by ultrasound machine to create characteristic images. Ultrasound is a form of mechanical energy which passes in wave form like sound waves and having a frequency wave the same type of wave as detected by the human ear, except the frequency is higher. Ultrasonic imaging uses frequencies in the range from 1 to 20 Mhz at powers from 0.01 to 200 mW/cm² (Bamber, et.al; 1986).
The ultrasound is generated and received by piezoelectric transducers. Ultrasound can be aimed in a specific direction and obeys the laws of geometric optics with regard to reflection, transmission and refraction. When an ultrasound wave meets an interface of differing echogenicity, the wave is reflected, refracted and absorbed. Only reflected sound waves (echoes) can be sensed by the transducer and processed to generate an Image. The transducer acts as a receiver over 99% of the time. (Goss, et al 1978)

2.4.2 Transducer:

Transducers convert electrical energy into mechanical energy to produce ultrasound and vice versa.

The part of the transducer which does this work is a piezoelectric crystal. It can be synthetic or natural. They have an inherent property of vibrating when an electric current is applied and thus produce ultrasonic waves and conversely produce an electric impulse when vibrated, thus helping the acquisition of data for the formation of an image. This effect is called "Piezoelectric effect" (Ossoinn d KC; 1979)
Figure 2-25 U/S Transducer

Quartz is a naturally occurring piezoelectric crystal. Synthetic ones are prepared from ceramics like lead zirconate and lead titanate. [Wild, et al (1952)]. The range of the velocities of ultrasound in body tissues is fortunately limited, so that time of return of an echo is a reliable indication of depth. Small variations give rise to geometrical distortions (Ossoinig and KC; 1979). Different tissues have different attenuation coefficients and this determines the quantum of reflection. This property has helped in imaging, tissue characterization and appropriate diagnosis. The greater the mismatch in acoustic impedance between two adjacent tissues the more reflective will be their boundary. (Ossoinig and KC; 1979)
2.4.3 Real time ultrasound:

B-Scan produces a single image frame. A real time ultrasound transducer produces multiple images in a very short time i.e., at least 16 or more images (frames) per second, which gives us a impression as though we are seeing the moving structures in real. This quick presentation of images is possible by oscillating the piezoelectric crystals (Ossoinig and KC; 1979)

2.4.4 Ultrasound Artifacts:

Artifacts are echoes that appear on the image that do not correspond in location or intensity to actual interfaces in the patient.

They can be of two types:

- Good Artifacts - which are helpful
- Bad Artifacts - which are disturbing

2.4.4.1 Good Artifacts

- Acoustic shadowing
- Acoustic enhancement
- Comet tail

2.4.4.2 Bad Artifacts

- Refraction
- Reverberation
- Mirror Image artifacts
- Beam width artifacts
- Movement artifacts
- Operator pressure artifacts
Figure 2-26 Transabdominal Ultrasound

Figure 2-27 Transvaginal Ultrasound

There are a variety of configuration of transvaginal probes including also the great number of overall size and shape. Examining a woman in the postmenopause the ovaries and the endometrium must be special field of interest. In general the curved linear multielement transducers afford the best density and overall field-of-view for imaging. In elderly women the stenosis
of the upper part of the vagina or adhesions of the vaginal walls can occur in addition to the atrophic changes of the epithelia. One must take special care when introducing the probe into the vagina of a woman in the senescence. Transvaginal sonography can induce unnecessary bleeding and pain. The same caution applies to women who have vaginal adhesions secondary to previous surgery for whom the vaginal examination may be painful. Using thinner transducers and lubricant gels could help to avoid the unnecessary injuries. The angled shaped transvaginal transducers (Aloka, Toshiba, General Electric) require narrower movements in the vagina when imaging the lateral structures of the female pelvis thus decreasing the discomfort. It is advisable to have an additional medical person being present during any kind of vaginal procedure.

2-4-6 TECHNIQUE

2-4-6-1 EVALUATION OF THE UTERUS:
Evaluation of contour changes, variations in echogenicity, masses and cysts. Any pathology must be measured in 2 planes. Fibroids should be labelled if they are submucosal, intramural, subserosal or pedunculated and there position within the uterus (Rt, Lt, Midline, Fundal, Body or cervical)

2-4-6-2 WIDTH OF UTERUS:
Assessment
Figure 3-2 Uterus measurement

The probe is turned slowly anticlockwise to visualise the uterus at 90 degrees to the sagittal view. The Maximum Width is measured in this transverse (coronal) plane.

2-4-6-3 ENDOMETRIAL MEASUREMENT:
Assess the endometrial status and measure the thickness: <10mm pre menopausal; <4mm post menopause or <6mm if post menopausal on HRT
Sagittal US image of the uterus obtained during the proliferative phase of the menstrual cycle demonstrates the endometrium with a multilayered appearance.

Normal premenopausal endometrium.

Sagittal US image of the uterus obtained during the secretory phase of the menstrual cycle shows a thickened, echogenic endometrium.

**Figure 3-3 Endometrial Measurement**

**2-4-6-4 ULTRASOUND OF THE PELVIS PROTOCOL:**

**2-4-6-5- ROLE OF ULTRASOUND**

To examine the uterus, ovaries, cervix, vagina and adnexae.

Classification of a mass identified on other modalities eg solid, cystic, mixed.

Post surgical complications eg abscess, oedema.

Guidance of injections, aspiration or biopsy.

**2-4-6-6 CHARACTERIZATION OF PELVIC MASSES:**

Ultrasonographically detected masses should be classified as predominantly cystic or solid. Cystic masses produce anechoic or hypoechoic images with excellent through-transmission of sound, resulting in a bright, distal surface.
(acoustic enhancement). Solid masses attenuate the sound energy and result in poor penetration. Masses containing gas also demonstrate poor sound transmission with clear proximal borders and indistinct distal boundaries. Cystic masses containing blood, tissue fragments, or other material are described as complex because they produce a cystic appearance as judged by sonic transmission, but the echo density of the intracystic material is greater than that of fluid alone. This type of echo pattern frequently is referred to as low-level echo density.

In addition to ultrasound characteristics, masses should be categorized by the suspected site of origin or location (e.g., uterine, ovarian, adnexal, cul-de-sac). If the site of origin is unclear, then a statement delineating separate, noninvolved organs frequently is helpful (e.g., an adnexal mass that does not appear to arise from either ovary).

The size of pelvic masses usually is measured along specified scanning planes to allow volume assessment if desired. In addition, the character of a cyst wall (smooth versus irregular) and intracystic anatomic appearance (e.g., septated, papillary) also assists in establishing the likelihood of a neoplastic or reactive (inflammatory or endometriotic) process, as opposed to a functional process.

**Leiomyoma:**

Uterine enlargement that is not caused by pregnancy most often is a result of uterine leiomyomata. Uterine leiomyomata represent proliferations of smooth muscle and are benign neoplasms. Leiomyomata are classified by their location as subserous, intramural, or submucous. Ultrasonographically, leiomyomata exhibit poor sound transmission because much of the sonic energy is attenuated by the solid consistency of the mass. Uterine contour irregularity is one of the most consistent findings; however, a variety of
findings may occur. Ultrasonography offers the potential of measuring uterine leiomyomata in patients in whom a conservative or nonsurgical management plan is initiated (Figs. 22 and 23). In addition, ultrasonography may detect early signs of degeneration or calcification of uterine leiomyomata. The effect of gonadotropin-releasing hormone suppression of uterine leiomyomata is easily monitored by ultrasonography. Occasionally, leiomyomata may undergo degeneration and mimic other cystic pelvic masses.

Figure 3-4 Uterine Fibroids
Figure 3-5  Anterior subserosal leiomyoma (arrow) in a postmenopausal female. The endometrial lining is atrophic and fluid is present within the endometrial.

Figure 3-6 large submucosal fibroid

Figure 3-7 A small intramural fibroid
2-4-6-7 THREE-DIMENSIONAL ULTRASOUND:

In patients with postmenopausal bleeding,( Gruboeck and coworkers;1996) showed that endometrial volume was superior to that of endometrial thickness as a diagnostic test for the detection of endometrial cancer in women not on hormone replacement. With a cutoff level of 13 mL for endometrial volume, sensitivity was 100% and the positive predictive value 92%. In addition to endometrial disease, volume estimation of cervical carcinoma has been studied using 3D ultrasonography.( Chou CY, Hsu KF, Wang ST et al ;1997)

In differentiating benign from malignant adnexal masses, (Chan and associates ;1997) found in eight women, 3D ultrasonography confirmed the preoperative diagnoses.( Wu and colleagues ;1998 )studied women with polycystic ovarian disease and compared them with routine controls. They found that stroma and volume determinations could be obtained more accurately by 3D images than by traditional ultrasonography. Not surprisingly, they found that women with polycystic ovarian disease have ovaries that are larger in size, area, and volume.( Dolz and coworkers; 1999) had similar findings.

(Kupesic and Kurjak;1998) describe the use of different ultrasonographic imaging modalities in the evaluation of septate uterus and rate of obstetrical complications.

As 3D ultrasonography becomes more available, its application and use in clinical obstetrics and gynecology will continue to be studied.
2-4-7 SCANNING IN THE POSTMENOPAUSE

The first and perhaps most important condition for transvaginal ultrasonography should be a thorough emptying of the urinary bladder. This is the condition, what makes the transvaginal ultrasonography more comfortable for the significant group of incontinent postmenopausal patients comparing to the transabdominal ultrasonography, which requires full bladder. On the other hand, some elderly patients who are unable to empty their bladder completely may need catheterization in order to the better visualization.

Once the probe covered with condom and some ultrasound coupling gel it is inserted into the introitus with slight downward pressure on the perineum while gently separating the labia majora with the fingers of the other hand. A small amount of coupling gel applied to the outside of the condom can act as lubricating interface. If the patient desires she can insert the probe herself. Inserting the probe into the midvagina the anteflexed uterus can be normally imaged in its sagittal (long-axis) plane. However, in the postmenopause with the loss of the strength of the uterine ligaments and the pelvic support, the uterus is frequently altering its position in the female pelvis. With the advanced ages it is usually situated in the midline in a straighten position and can be imaged only after inserting the probe into the fornix. Additionally, a descended, even a prolapsed uterus let the examiner to orientate only after the re-establish the about normal situation by pushing the uterus upwards manually and then holding there with the probe. There is less problem with the visualization of the retroverted, retroflexed uterus in this age. Trendelenburg position makes the examination easier, even if the cardiac status of the patient makes it impossible.
In the long axis, one can appreciate the different interfaces of the endometrium, beginning with the interface of the cervical canal. Because the echogen cervical mucus is very poor and the endometrium can be very thin and atrophic in the post menopause, visualization of the endometrium can be difficult, usually it appears in the form of a thin, echo-poor line in the midline of the sagittal plane of the uterus.

After adequate image in the long axes are obtained the probe can be moved 90° to image the uterus in a horizontal (semi-coronal, semi-axial) plane. The endometrium again have to be identified in the level of the tubal ostia.

Once the endometrium is adequately depicted in its long and short axes, the probe is withdrawn into the midvagina and images of the cervix can be obtained.

The ovaries usually located lateral to the uterus, above and medial to the hypogastric vessels, lying in the area called “Waldeyer’s fossa”. Their size, morphology and locations are altered by the age, previous diseases and surgeries and other factors in the postmenopause. During the reproductive year the follicles serve as sonographic “markers” of the ovaries. After the menopause it is hard to find them because these “markers” are not present, the ovaries themselves atrophy and there is less pelvic fluid to provide an acoustic interface. Their detection becomes more difficult with advancing of the age. Beside negative bimanual pelvic examination non-visualization of the ovaries can be accepted without serious concern about ovarian pathology. With the introduction of color coded Doppler flow imaging, by finding the color coded flow of the ovarian artery or vein one can better detect the otherwise sonographically “non detectable” ovaries. Using the other hand can be very useful for manipulating the ovaries into the “scanningsight” of the probe by pushing them slightly downward through
the lower abdominal wall into the direction of the tip of the probe. (Zimmer ez ; 1997).

Detecting some free fluid in the cul-de-sac doesn’t necessarily mean pathological finding, but in the presence of that ovarian pathology must be searched with special care. The same goes for any palpated or visualized solid structure in the cul-de-sac. The vascularization of these gynecological findings must be examined intentionally also by color and pulsed Doppler.

When a large amount of fluid is present, such as in ascites, one of the first questions is whether the pathological condition arises from an ovarian tumor or is related to a non-gynecological disorder. Fluid is clearly outline the boundaries of the structures and ovarian pathology may be revealed. If the ovaries appear normal, one may consider other reasons leading to ascites.

Constipation is not rare in postmenopausal patients. Scibalas shouldn’t be confused with pelvic masses. Excluding the vascularization of a structure by the color Doppler helps in differentiating normal to abnormal.

Solid pelvic masses also must be differentiated from the intestines. In real-time mode the motion of the bowels help to distinguish the peristalting intestines from the fixed structures. In the postmenopause the peristalsis is frequently inert, which requires proper patience from the examiner. In case of a vascularized mass color and pulsed Doppler offers again a quick possibility.

From all of the above mentioned it follows, that the sonographer have to get know the finding of bimanual pelvic examination, which should, therefore, precede the transvaginal ultrasound examination. It strengthens the recommendation that the person, who performs the transvaginal ultrasound examination in a postmenopausal patient, should perform a bimanual pelvic examination before, thus orienteering the anatomical situation and obtaining
previous information to decide, which is the adequate way for ultrasonography or is there any suspicious structure palpated. In case of uncertainty of the origin of the palpated or visualized structure transrectal examination or enema might be also necessary.

Sonographic examination of the female pelvic organs is most commonly performed using two different approaches. The first and older, is transabdominal, the second and more recent, is transvaginal. (Zimmer EZ ;1993) A third method, transperineal, is also employed, though less frequently. (Campbell S .et al ;1990). A thorough ultrasound examination of the pelvis should include both complete transabdominal and transvaginal studies, unless either limited information is needed (e.g., follicle size) or extenuating circumstances dictate otherwise (e.g., patient refusal). The techniques are complementary, not mutually exclusive. The person performing the ultrasound examination can vary certain parameters to optimize the quality of the study. These include: bladder distention, manual manipulation of the anatomy and patient positioning.

2-4-7-1 BLADDER FILLING:

Transabdominal ultrasound of the female pelvis should be performed with the bladder optimally distended. The operative phrase is "optimally distended". If too full, the patient may experience excessive discomfort, which might result in guarding. Also, the overdistended bladder may push the target structures so far from the transducer that image quality suffers. Optimal distention of the bladder can be achieved by having the patient void incrementally. If too empty, near-field artifacting and overlying bowel gas may degrade image quality. Optimal distention of the bladder can be achieved either by waiting for the bladder to fill more completely or having the patient ingest additional fluid and then waiting.
Unequivocally, there are occasions in which the empty bladder transabdominal examination may yield better results than either the transvaginal or filled bladder approaches. This is particularly true when relatively large, especially fundal, fibroids are present.

Transvaginal (or endovaginal) ultrasound is generally performed with the bladder empty. The operative phrase is "generally performed". If too full, the patient may experience excessive discomfort. Also, the distended bladder may push the target structures so far from the transducer that image quality suffers. Optimal distention of the bladder may be achieved by having the patient void, perhaps incrementally.

Unequivocally, there are occasions in which the transvaginal examination may yield better results with a filled or filling bladder than with an empty bladder. If a structure of interest is either not apparent or is suboptimally seen, patience and bladder filling may result in better visualization.

2-4-7-2 ULTRASOUND OF THE UTERUS – Normal:
<table>
<thead>
<tr>
<th>Uterus TA probe positioning for longitudinal scan.</th>
<th>Uterus sagittal US image.</th>
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<tbody>
<tr>
<td><img src="image1" alt="Uterus TA probe positioning for longitudinal scan." /></td>
<td><img src="image2" alt="Uterus sagittal US image." /></td>
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<th>Uterus TA probe positioning for Axil scan.</th>
<th>Uterus Axil US image.</th>
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<tr>
<td><img src="image3" alt="Uterus TA probe positioning for Axil scan." /></td>
<td><img src="image4" alt="Uterus Axil US image." /></td>
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**Figure 2-28**

<table>
<thead>
<tr>
<th>Transvaginal Technique Anteverted uterus.</th>
<th>Normal TV image anteverted sagittal.</th>
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</thead>
<tbody>
<tr>
<td><img src="image5" alt="Transvaginal Technique Anteverted uterus." /></td>
<td><img src="image6" alt="Normal TV image anteverted sagittal." /></td>
</tr>
</tbody>
</table>

The overall uterine length is evaluated in the long axis from the fundus to the cervix (external os). The depth (AP diameter) is measured from the anterior to the posterior wall and perpendicular to
2-5 PREVIOUS STUDY

2-5-1 Ultrasound in the Postmenopause

2-5-1-1 CHALLENGES OF THE POSTMENOPAUSE

For a female life expectancy in the developed world approaching 80 years a woman may, on average, expect to spend some 30 years—or 40% of her active life—in the postmenopausal.

It is probably true, that women have greater expectations of higher quality of life, than previous generations. Therefore, it seems likely that climacteric and postmenopausal women will continue to place increasing demands on health care resources for many years to come. The gynecological care of the fertile female population has already been well established in most countries. In the not too distant future, postmenopausal women will constitute the major proportion of the gynecological patient population. Preventive medicine and care for elderly will be an essential part of the general
gynecological practice. In parallel with the demands “climacteric and postmenopausal clinics” are being established worldwide with the active participation of gynecologists, cardiologists, rheumatologists, etc. With better nutrition, health care and living conditions more women are living long enough to develop ovarian and endometrial cancers, which are known to be more common after the menopause. Undoubtedly the care of postmenopausal population must include the early detection of ovarian and endometrial cancers, just as the access to the benefits of hormone replacement therapy. Estimating the potential individual benefits and monitoring the hormonal replacement therapy are further challenges in the medical care of the postmenopausal women. Vaginosonographic examination has become an examination technique that is accepted by postmenopausal women in particular and which gives the less experienced examiner an overall view of the internal genital tract in the female. Recent advantages of transvagal color Doppler and three-dimensional ultrasound enables the more experienced examiner to visualize even the smallest vessels and investigate blood flow characteristics in the poorly perfused small pelvis in the postmenopause, what helps him to differentiate between the normal, suspicious and pathologic variations of the structures or detect and follow the effects the hormone replacement therapy on the perfusion of genital tract.

2-5-1-2 THE POSTMENOPAUSAL UTERUS

2-5-1-2-1 Morphology:
The uterus can be imaged in three major scanning planes with TVS. There is generally a homogenous echo pattern in the postmenopause, and the uterine cavity is frequently not imaged.(Kulen Kamp et al 1995) The uterine wall is smooth and clearly outlined against its surroundings. In the myometrium,
towards its periphery and often protruding, can be found echoless vessels. In the postmenopause the arteries can calcify in this region. These calcifications appear as small, bright reflections, regularly spread in the uterine wall. They can evoke shadowing, which may impair the assessment of structures lying beyond, e.g. the endometrium (Rempen A; 1995) Unlike in all phases of the menstrual cycles, undulatory motions, i.e. uterine contractions cannot be observed in the postmenopause.

### 2-5-1-2-2 Biometry:

As in the case of all genital organs in the female, the development, the maintenance of the fertile size and shape, and the postmenopausal physiologic involution of the uterus are highly dependent on the actual serum level of the estrogen.

Measurement of the uterus in the sagittal plane can be carried out either by determination of the portio-fundus distance or in postmenopause the separate measurement of the cervix and corpus uteri can be also used. It was already mentioned in the introduction, that the corpus-cervix ratio of the postmenopausal uterus shows remarkable changes in favor of the cervix, with advanced ages it can even fall below 1 and can reach the 1:2, like in childhood.

The sagittal measurement is supplemented by the determination of the largest anterioposterior diameter of the corpus uteri, and of the largest transverse diameter of the corpus. The size of the corpus decreases markedly in the postmenopause, shrinking to average $4.5 \times 1.5 \times 2.5$, with the cervix predominantly over the corpus in the sense of the elongation of the cervix. The mean length of the postmenopausal uterus was shown to be $59 + /-.11$ mm by Andolf et al.46 The upper size limit of the postmenopausal uterus has been suggested to be 3 cm in the anterior-posterior diameter, with a cervical-
fundal length of 8 cm. The patient who is only 1–3 years postmenopausal when still has significant endogenous estrogen production by the ovaries, or who has significant endogenous estrogen production by fat from adrenal precursors will have larger uterus, than the patient who is over 10 years postmenopausal. Clinical judgment is needed in interpreting normality of uterine size in postmenopausal of uterine size in the postmenopausal patient.11

Uterine involution is a slow process. Myometrial thickness are changed as the years of the postmenopause progress.

In their study Zalud et al (1993); did not found significant changes in the myometrial thickness in women without HRT over the years after the menopause, though slow thinning can be observed. Myometrial thickness after the menopause changes less than does endometrial thickness, which may suggest that these changes are more dynamic.

2-5-1-2-3 The Uterus under Hormone Replacement Therapy

Comparing myometrial thickness between groups of postmenopausal with and without HRT, (Zalud et al;1993) did not demonstrated statistical difference, though slight difference were found in favor of women, who received HRT more than 5 years. These data are not surprising in the mirror of the findings of the same group, namely that the myometrial involution couldn’t been statistically expressed over the years throughout the postmenopause, too. The involutional process of the myometrium is very slow and other factors, than estrogen can also influence, as the sizes of the uteruses of fertile women can show great variability.

2-5-2 Color Doppler Velocimetry in the Postmenopausal:

2-5-2 -1 Uterus:
The vascular supply of the uterus is provided by a complex network of arteries originating from the uterine artery, which is a branch of the hypogastric artery. The color Doppler signal from the main uterine vessels can be seen lateral to the cervix at the level of junction between the corpus and cervix. (Kurjk et al 1992). Flow velocity waveforms from the radial arteries can be obtained within the myometrial fibers, while spiral arteries are visualized at the level of endometrial-myometrial junction.

Visualization of both uterine artery by transvaginal color Doppler can be achieved even in the advanced years of the postmenopause. In contrary, visualization rate of the myometrial and endometrial vessels are highly dependent on the length of the postmenopausal period.

The ageing process affects the uterine perfusion. In general high impedance and high velocity is characteristic for the uterine arteries, though the uterine perfusion is largely dependent on age, phase of menstrual cycle, other conditions (e.g. pregnancy, tumor) and there are complex relationships between the concentration of the ovarian hormones in the serum and uterine artery blood flow parameters. (Goswamy RK; 1988)

Additionally, there might be also a relationship between the serum gonadotropin levels and uterine perfusion. Examining normal postmenopausal patient and premenopausal patients treated with GnRH analogues Luzi et al (1993) found, that the pulsatility index of the uterine artery in spontaneous menopausal women is significantly higher than in artificial menopausal women. This phenomenon may be due to a different hormonal pattern which exists in the two groups, i.e. the gonadotropin levels increased in the former and decreased in the latter. The vascular compliance in artificially induced menopause is higher than that observed in spontaneous menopause, as shown by a higher diastolic flow and a less deep
notch. The decrease of the vascular compliance in postmenopause can be caused by progressive sclerosis of the vessel walls. Resistance to blood flow increases in both the main uterine and the radial arteries as the years of postmenopause progress, though the increase of ovarian blood flow impedance is more pronounced. The fact, that uterine artery RI does not change significantly in the first years of menopause strongly support the thesis that ageing process initially affects the uterus less than the ovary.

The diastolic flow decreases in postmenopause and the systolic peak increases. (Luzi O; Coata G 1993) The RI in the main uterine arteries continuously increases with the number of the postmenopausal ages, but unlike in the case of the ovarian artery, doesn’t reach the maximum in all women even at advanced ages. Absent diastolic flow in uterine arteries was found in 15% of women with 1–5 years duration of menopause, while clear interruption of diastolic blood flow was observed in the uterine artery of one-third of the women in the next five years of the postmenopausal period. More than half of the women has this finding with 11–15 years lasting postmenopause and finally, 80% of women whom LMB occurred more than 16 years ago demonstrated absent diastolic blood flow signal indicative of high vascular impedance.

The changes in flow velocity patterns of the radial arteries in postmenopausal patients parallel the blood flow dynamics of the uterine arteries. Visualization of clear Doppler signals from the spiral artery is possible only in less than one-third of postmenopausal women, in whom LMB occurred 1–5 years previously. The impedance is significantly increased in these vessels, too, when comparing to the premenopausal levels. In normal
postmenopausal women already 6 years after the LMB no blood flow signals can be expected from the inner third of myometrium and the area of the myometrio-endometrial junction.

Figure 2-30 Endometrial vascularization - the radial and spiral arteries. Power Doppler is used to visualize the low velocity flow in small vessels.

Figure 2-31 Reproducibility of Endometrial Vascular Patterns in Endometrial Disease as Assessed by Transvaginal Power Doppler Sonography in Women With Postmenopausal Bleeding
Figure 2-32 Normal uterine artery

Doppler Flow Velocity Waveforms

UTERINE ARTERY

PSV  
EDV  
MFV

COMMON ILIAC ARTERY

PSV  
MDV

EXTERNAL ILIAC ARTERY

PSV  
MDV

Figure 2-33
2-5-2 -2 Myomas and Malignant Potential after the Menopause

Uterine fibroids of 0.5 cm can be detected by TVS and their relationship to the endometrial cavity precisely defined (e.g. submucous, intramural, subserous). They appear with TVS as rounded, well defined, space occupying structures.(Lawit N et al; 1992) Growth of myomas is known to be estrogen dependent. The management of the myoma around the menopause is highly conservative, since after the menopause they supposed to regress in the lack of the hormonal support. Myomas with good vascularization can be seen less frequently after the menopause. They show more hypoechogenic structure, compared to the normal uterine tissue, while homogenous, hyperechogenic myoma have often undergone regressive changes and have a large amount of connective tissue. Other regressive changes such as necrosis and caseous and cystic degeneration can be recognized by the presence of hypoechogenic regions or regions without echogenicity in the myoma. Such a necrotic myoma can be confounded with an ovarian cyst or a colliquated endometrial carcinoma depending on its localization. Hyalinization and calcifications of the myoma responsible for bright reflections can be seen frequently in the postmenopause.

Transvaginal color Doppler can be used to assess leiomyoma vascularity, as well as he physiological and pathophysiological characteristics of uterine artery blood flow.(Kujak et al;1993) The vascularization of leiomyomas is supported by pre-existing myometrial vessels originating from terminal branches of uterine arteries. Since the leiomyoma grows centripetally as proliferation of smooth muscle cells and fibrous connective tissue, the color Doppler demonstrates most of the leiomyometrial blood vessels at its periphery. While their visualization rate high in the premenopausal period
(58–70%), 57–59 it decreases after the menopause with the decreased blood supply of the uterus. (Kurjak A; 1991)

The growing inclination of the myoma is in correlation with the increased blood flow in the uterine network, the latter is thought to be a result of large concentration of estrogen receptors and estrogens. (Wilson EA; 1980) Whether the regression of the myoma after the menopause is resulted directly by the fallen estrogen levels or it is only a secondary consequence of the decreased perfusion, still not known. Though Doppler studies on the perfusion of the uterine fibroids in the postmenopause are not available yet, it can be anticipated from the Doppler studies on myomas under treatment with GnRH analogues, (Mata W; 1988) that with the decreasing estrogen levels the previously decreased impedance of the uterine artery and the supplying myometrial vessels are increasing to the level of a normal postmenopausal uterus leading to the involution of the myoma. It must be emphasized, that in case of necrotic, degenerative changes in the myoma the presence of blood vessels in the central portion is usual and the impedance of them can be so low, that might be misinterpreted as malignant neovascularization. One can use TVS as a means of monitoring the size and the ability for growth of the leiomyomas around and after the menopause. If there is evidence of rapid growth of a pre-described myoma in a postmenopausal women, and in ultrasound an increase of echoless areas as a sign of necrosis, laparotomy should be performed because of a suspected malignant transformation.

Though the sarcomas are account for only 1–3% of the malignant tumors of the uterus (including the endometrium), their early diagnosis can greatly depend on an occasional but accurate ultrasound examination, since there are scarcely any symptoms of an early process. It is expected to be more
common, since the conservative treatment of uterine myomas is becoming more reliable alternative to the surgery. (Meyer WR; Meyer AR; 1990)

Nevertheless, there are, similar to macroscopic aspects, sonographic indications for the existence of sarcoma. Primary sarcomas in ultrasound examinations appear as poorly outlined masses, partly hyperechogenic, partly irregularly limited and hypoechogenic, or without echogenicity. Morphological differentiation from myoma can be facilitated by the absence of any systematic structure (onion-skin or whirlpool pattern). Differential diagnosis of inhomogenous, myometrial masses can be myomas undergoing carneous degeneration, with a pool of liquid or bleeding into the myoma.

Application of the color and pulsed Doppler may confirm or preclude the in vivo diagnosis of uterine sarcoma. The presence of irregular, thin and randomly dispersed vessels in the peripheral and/or central area of tumor, with very low impedance shunts characterizes intratumoral neovascularization and is in favor of the malignant transformation. In benign uterine lesions, even if intratumoral vascularization can be detected, the resistance to blood flow was found significantly higher. Furthermore, in the case of the uterine sarcoma, both uterine arteries shows a low impedance in comparison with that of normal, even the postmenopausal or myomatous uterus.

Unfortunately as the result of the wide range of the biological variations and the vascular characteristics of tumors an overlap exists between the blood flow patterns of benign and malignant uterine tumors. At the moment the realistic approach is to consider the above mentioned guidelines only in general, but one has to take the decreased intratumoral impedance and increased vascularity into serious consideration, especially it is accompanied with rapid growth of the tumor during the serial examination.

2-5-2 -3 Leiomyomas under Hormone Replacement Therapy:
Leiomyomas are the most common pelvic tumors in women of the reproductive age; 20–25% of women have uterine myomas. Higher concentrations of estrogens and estrogen receptors within leiomyomas than in adjacent myometrium were taken as evidence of the hormone dependence of their growth.

Though they tend to regress after menopause with the decreasing serum level of the promoter estrogen, it is questionable, whether the introduction of the HRT promotes the growing process again. The data are confronting, in some papers myoma growing, in some there is no difference in size and in some there is even decreasing in size. (Jirapinyo et al; 1998)

However, even if there are increasing in myoma size, this does not appear to cause clinical symptoms.

In practice, uterine and fibroid size can be closely monitored by ultrasound and HRT can be easily discontinued if the fibroid enlarge.

2-5-3 THE POSTMENOPAUSAL ENDOMETRIUM

2-5-3-1 Visualization and Morphology:

After the menopause, decrease of ovarian estrogen production leads to atrophy of the endometrium. In consequence the endometrium of a postmenopausal women is typically thin when examined by TVS, which corresponds to the stratum basale adjacent to the myometrium. Its sonographic feature is very similar to that of the endometrium in the early follicular phase. (Osmer et al; 1995) The endometrial band is sonographically narrow and the echogenic line of the uterine cavity often cannot be visualized. In the study of Andolf the endometrium couldn’t be localized in 7% of postmenopausal women without bleeding disorder. Granberg et al couldn’t visualize 10% of the histologically atrophic postmenopausal endometrium. (Renpon A et al; 1995) In addition, shadowing arising from
myomas or arteriosclerosis make visualization more difficult. Echotexture of the endometrium is usually more echogenic than the surrounding myometrium. The study of Andolf;(1993) 85% of the assessable endometrium were less echogenic than the myometrium in asymptomatic postmenopausal women. The poorly echogenic myometrial zone, the subendometrial halo round the endometrium is frequently absent in the postmenopause. However, the interrupted subendometrial halo was reported as a common sign of the myometrial invasion of the endometrial carcinoma.(Lin MC ;1991)

2-5-3-2 The Postmenopausal Endometrial Thickness:
Contrary to early methods of measurement of the thickness of the endometrium, now a days and according to general agreement—measurement should be carried out as follows: the uterus is viewed vaginosonographically in the longitudinal section and the total thickness of the endometrium is measured in the largest diameter (double layer). In the case of any kind of intrauterine fluid collection, the thickness of the fluid pool in the uterine cavity is subtracted from the total thickness (Osmer R et al;1995)

The postmenopausal endometrial thickness is 2–4 mm, with a considerable scatter range of 0–10 mm, and consequently there are different limiting values for the normal state in the literature. There is a correlation between the endometrial thickness and the body weight. Women with pure estrogen replacement therapy frequently have endometrial thickness exceeding that of postmenopausal women without it.(Lin MC;1991)

2-5-3-3 Suspect Postmenopausal Endometrium:
Endometrial carcinoma is the most common invasive gynecological malignancy in the United States and Europe today. The incidence of the
disease increases considerably during the fifth decade of life and the average age at diagnosis is 59 years. In the more cosmopolitan, higher income population the rate has overcome the 40 cases per 100,000 related to increased longevity, increased cholesterol in diet, and exogenous estrogen supplementation or substances with an estrogen-like effect.77,78 The five-year survival rate is related to myometrial invasion, ranging from 93.7%, when no invasion is present to 36.2% if the invasion is deep.79 When the first clinical sign, vaginal bleeding occurs, the myometrial infiltration depth is already 10 mm on average.(Williams and Wilkins ; 1988)

Of endometrial carcinomas 80–90% present with atypical bleeding demonstrating the ineffectiveness of exfoliative cervical cytology and the need for early recognition of this most frequent genital malignancy.(Osmer A. and KurjK A ; 1995) Until now curettage and histologic evaluation is the accepted method to assess the background of the atypical bleeding. However less than 10% of women with postmenopausal bleeding have endometrial cancer.(Bourner TH ; 1991) Therefore, a new non-invasive screening method must fulfill at least double requirements: it should be able to recognize the abnormal endometrial process at an earlier stage, than bleeding occurs and it should reduce the number of the “unnecessary” curettage, when postmenopausal bleeding occurs.

Osmer R ; Volks M; Rath W ; Kuhn; (1990) have reported that endosonographically measured endometrial thickness correlates closely with the presence or absence of endometrial cancer in asymptomatic postmenopausal women. An endometrium, that measures greater than 8 mm from one myometrial endometrial interface to another in postmenopausal woman without HRT is highly likely to be associated with significant endometrium pathology. Approximately 10% of asymptomatic
postmenopausal women have endometrium exceeding this cut-off level and 3.5% of them can be expected to have endometrial carcinoma. There is, however, a significant false-positive rate. Using a cut off level of 8 mm we have a very high sensitivity, but a low specificity. (Osmer R; Kuho W 1994) The only way to increase the specificity would be to adjust the cut-off value to a higher level, but in this case the sensitivity of the screening would decrease and more positive cases might be overlooked.

Another possible approach to reduce the number of false-positive findings could be the use of color Doppler.

The realistic approach is that endometrial thickness up to 5 mm can be regarded as completely normal finding in the postmenopause. Endometrial thickness 6–9 mm requires control examination in 3–6 months time, if the patient really asymptomatic. Endometrial thickness of 10 mm and above requires urgent histological examination. The most essential characteristic of a cancer screening method is not only detect the malignancy, but also improve the prognosis by early detection. (Osmers et al 1994) compared the myometrial invasion depth, the most prominent prognosis factor of asymptomatic endometrial cancers detected by TVS (cut-off value 8 mm) and symptomatic cancers (atypical bleeding). They concluded that early endometrial cancers detected by TVS will have better prognosis than symptomatic ones, since the average myometrial invasion was 4 mm and 10 mm, respectively.

Up to now, there are no sonomorphologic criteria to differentiate between benign and malignant endometrial neoplasm. Therefore we cannot detect endometrial cancer by TVS alone, but TVS is an excellent tool to define a risk group in postmenopausal women. (Osmer R ;1994)

2-5-4 Postmenopausal Intrauterine Fluid Collection
Occasionally, a small amount of intraluminal fluid may be detected in the postmenopausal uterus, the detection rate of it can reach the 16% in asymptomatic postmenopausal women. (Kurjak et al; 1995)

We can only speculate as to the pathophysiology of fluid accumulation in the uterine cavity. Senile cervical stenosis can prevent drainage of possibly minimal endometrial secretion leading to small intrauterine pools. This, however, speculative as some degree of cervical stenosis is ubiquitous in postmenopausal women, whereas intrauterine fluid is rare finding. Patients with ascites are more likely to have intrauterine fluid. The possibility of the tubal cancer is rather theoretical, than practical, although one case one reported by Carlson J 1991) Cancer of the cervix may obstruct the cervical canal and can cause intrauterine fluid accumulation. However, the main suspected reason must remain the endometrial malignancy.

Although the presence of intrauterine fluid has been considered ominous and related to malignancy by some authors, the significance of this finding is still not clear, especially in routine examinations. Management and clinical evaluation have also not determined.

In their recent series of twenty postmenopausal women with intrauterine fluid collection Pardo et al 1994) revealed 3 cases of endometrial carcinoma, though it must be emphasized, that all of these positive cases the endometrial thickness was more than 4 mm.

Carlson et al; 1994) reported also twenty cases of endometrial fluid collection in the postmenopause, of which five proved to be results of some kind of genital malignancy (two ovarian, one tubal, one endometrial, and one cervical).

However, examining the fluid pools in the uterine cavity Osmers et al; 1995 did not find association with pathological changes in narrow endometrium.
The extensive use of sonography will lead to an eventual increase in the number of postmenopausal patients diagnosed with intrauterine fluid. In every case, careful scanning is recommended—to rule out ovarian and tubal pathology. Obviously, the endometrium must be submitted serious examination by transvaginal ultrasound. Polyploid growths and irregularity of the endometrial surface are particularly well seen when surrounded by intraluminal fluid.2 Certainly, cytological evaluation of the cervix, with special regard to the cervical canal is essential. Additionally, both Pardo and Carlson;(1994) recommend immediate endometrial sampling even in the cases of thin endometrium, until accumulated data permit dismissal of endometrial sampling in that cases. According to Osmers et al indication for D&C is given only when there are pathological endometrial findings. In the lack of any other suspicious finding and beside thin endometrium Fleischer AC (.1991) also allows rescanning, but if it is present or even volume increase on repeat scans, this finding should be considered suspicious for an endometrial disorder.

Undoubtedly, color Doppler offers an additional help in getting closer to the proper management, as it is able to assess the vascularization around this questionable ultrasound finding of postmenopausal intrauterine fluid collection.

2-5-5 Color Doppler Velocimetry and the Postmenopausal

2-5-5 -1 Endometrium

The visualization rate of the postmenopausal endometrial vessels are very low. The visualization rate of endometrial vessels is in accordance with decreasing endometrial thickness with the postmenopausal years.(Kupesic S;Kurjak A 1995) As it was already mentioned, vascularization of the inner third of the myometrium and the endometrio-myometrial junction is possible.
only in about or less than one-third of those normal postmenopausal patient, who had the last menstrual bleeding not more than 5 years previously. No flow can be detected in the normal, atrophic endometrium in the postmenopause. (Kujak A et al 1993)

Although a thick endometrium may be a sign of pathological processes, no morphological features that are unique to malignant disease have been identified. (Osmer et al 1990)

Transvaginal color and pulsed Doppler has shown that the presence of intratumoral vascularization with a low impedance to blood flow can be used as an end point in screening programs for some gynecological malignancies. (Kurjak et al 1993)

Bourne et al (1990) reported the impedance to blood flow in the uterine arteries and the endometrial thickness in women with postmenopausal bleeding with or without cancer. In the women with postmenopausal bleeding who did not have endometrial cancer and in those without postmenopausal bleeding were similar. Conversely, the highest PI in the group with cancer (1.49) was below the lowest value in the group without cancer (1.95). Data from this study suggest that, in the presence of malignant tissue, the impedance to blood flow within the uterine artery is reduced significantly when compared to control groups. This observation was later confirmed by others. (Kupesic et al 1993) If color Doppler is used to interrogate the endometrium in such cases, angiogenesis can be demonstrated as areas of color superimposed on the B-mode gray scale image and the sensitivity of the technique is enhanced. (Bourne et al; 1991)

Hata et al; (1992) found a feeder artery in patients of endometrial cancer, and in seven out of nine endometrial cancers even venous blood flow in the endometrium could be detected, while no flow was detected around and
within the endometrium in noncancer patients. These findings were confirmed by pelvic angiography.

In the work of Kurjak et al (1990) visualization rate of the abnormal blood flow within the endometrium was 100% in the cases of endometrial carcinoma. Of the cases with detected endometrial carcinoma had endometrial (tumoral) thickness >10 mm, which is already a suspect sonographic sign alone. However, 10% of these endometrial carcinomas with endometrial thickness 5–10 mm would have been missed without color Doppler.

It was also suggested in the same work, that color Doppler should help in distinguishing between cancerous and hyperplastic thickened endometrium. Flow could be detected only in 92% of cases of endometrial hyperplasia. Blood flow pattern was characterized with a low RI near or <0.40, which constituted statistically significant difference compared with that of endometrial hyperplasia, if any flow detected. (Kurjak A. et al ;1990)

By using color Doppler and measurement of the endometrium thickness together whilst maintaining the sensitivity, the false-positive rate of the ultrasound-based test is reduced. If further data confirm, superimposing the color Doppler onto the endometrium at a questionable thickness (5–10 mm) and searching for vascularization in or around the endometrium might help to determine the further management of the patient and can lead a further reduction of the number of dilatation and curettage in the postmenopause. (Bourne TH. ;1990)

On the basis of the current literature and according to the recommendations of Osmer R. and Lin MC.(1991) the following approach can be regarded as
guidelines when examining the endometrium of an asymptomatic postmenopausal woman by vaginosonography.

A woman receiving sequential hormone replacement should be examined after completion of the progestational phase of the cycle (days 1–2).

All patients with hormonal replacement therapy are advised to undergo sonographic checks of the endometrium in 6 months time.

a. Endometrial thickness up to 8 mm is regarded as normal finding,

b. Endometrial thickness between 8 and 15 mm is regarded suspicious. After administration of an oral gestagen, subsequent to the withdrawal bleeding, a second sonography is performed. If the endometrium still measures more than 8 mm, D&C is recommended. With endometrial thickness of less than 8 mm, control sonography in 3 months time is recommended. Women receiving unopposed estrogen or continuous estrogen and progestogen and having endometrial thickness between 8 and 15 mm need to undergo D&C.

c. Any patient with endometrial thickness of at least 15 mm should undergo histological diagnosis on grounds of the unusual thickness, regardless of symptoms or hormone status.

The endometrium of all postmenopausal women should be assessed vaginosonographically before the onset of HRT, whereas the guidelines for assessing the postmenopausal endometrium detailed in the previous chapter should be taken into consideration.

Above recommendations are only for woman on sequential therapy. Woman on continuous therapy should be regarded and followed as postmenopausal woman without therapy.
CHAPTER THREE
MATERIALS & METHODS

3-1 MATERIALS:

3-1-1 MACHINE USED:

GE LOGIQ P5

3-1-1-1 Product Information:

3-1-1-2 Manufacturer: GE

3-1-1-3 Description:
The GE LOGIQ P5 is an ergonomic, highly mobile and easy to use high performance multipurpose imaging system. GE's Logiq P5 ultrasound has the same features and provides the same quality of a much larger system but at half the weight and half the size. It is a great ultrasound for private practices, clinics, and hospitals. Used for 3D and 4D cardio imaging, this machine is also flexible enough to be compatible with other GE probes such as the E8C Transvaginal.

3-1-1-4 Performance:

- System Architecture: TruScan, TruAccess, SmartScan, ComfortScan
- Operating Modes: B, M, M-Color, Doppler, CFM, PDI
- Listed to UL 2601-1
- Certified to CSA 22.2, 60601.1 by an SCC accredited Test Lab
- 15 inch TFT LCD, XGA Format

3-1-1-5 Capabilities:
Abdominal, OB/GYN, Neonatal, Cardiology, Small Parts, Vascular, Pediatrics, Urology

3-1-1-6 Available Transducers:
10L, 12L, 5C, 3.5, E8C, 3C
3-2-1 POPULATION:
The study population was composed of menopausal ladies presenting to the ultrasound section of Samha and Shahama clinics in Abu Dhabi UAE during the period from June 2014 to June 2015

3-2-2 Study sample
50 selected patients who were referred the interdisciplinary ultrasound department of both Samha and Shahama clinics for sonographic examination of the lower abdomen were initially included in this study

3-2-3 EXCLUDED CRITERIA:
-postmenopausal women who are under hormone therapy
-postmenopausal women who have vaginal bleeding

Methods
3-2-5 Duration the study:
This study was done during the period from June 2014 to June 2015.

3.2.6 Data collection
The data was collected by master data sheets using the variables of menopausal ladies age, diseases, echogenicity and measurement of uterus, endometrium thickness and indices include RI, PSVTAMXV.

3.3 Data analysis
Data were analyzed by using SPSS program and the results were presented in form of graphs and tables.

3.4 Ethical consideration
- No identification or individual details were published.
- No information or patient details will be disclosed or used for reasons other than the study.
CHAPTER FOUR

RESULTS

Table No : 4-1 Showing data collection statistic

<table>
<thead>
<tr>
<th>Statistics</th>
<th>Age/Y</th>
<th>Postmenopausal age/Y</th>
<th>Endometrial thickness/cm</th>
<th>WIDT H/cm</th>
<th>DEPTH/cm</th>
<th>Right PSV</th>
<th>Right TAMXV</th>
<th>Right PI</th>
<th>Left PSV</th>
<th>Left TAMXV</th>
<th>Left PI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>62.5</td>
<td>50.7</td>
<td>3.7</td>
<td>3.3</td>
<td>2.6</td>
<td>26.8</td>
<td>10.2</td>
<td>2.2</td>
<td>30.1</td>
<td>10.5</td>
<td>2.2</td>
</tr>
<tr>
<td>Media</td>
<td>64.0</td>
<td>51.0</td>
<td>3.9</td>
<td>3.4</td>
<td>2.6</td>
<td>26.8</td>
<td>10.4</td>
<td>2.2</td>
<td>30.2</td>
<td>10.6</td>
<td>2.2</td>
</tr>
<tr>
<td>S.D</td>
<td>6.23</td>
<td>1.4</td>
<td>.73</td>
<td>.28</td>
<td>.27</td>
<td>.55</td>
<td>.30</td>
<td>.02</td>
<td>.51</td>
<td>.18</td>
<td>.03</td>
</tr>
<tr>
<td>Minimum</td>
<td>49.0</td>
<td>45.0</td>
<td>2.0</td>
<td>3.0</td>
<td>2.1</td>
<td>26.0</td>
<td>9.30</td>
<td>2.1</td>
<td>29.1</td>
<td>10.0</td>
<td>2.1</td>
</tr>
<tr>
<td>Maximum</td>
<td>73.0</td>
<td>53.0</td>
<td>5.1</td>
<td>4.2</td>
<td>3.2</td>
<td>28.1</td>
<td>10.6</td>
<td>2.2</td>
<td>30.9</td>
<td>10.9</td>
<td>2.2</td>
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</table>
Table No 4-2 showing Submucosal fibroids.

<table>
<thead>
<tr>
<th></th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>No</td>
<td>48</td>
<td>98.0</td>
</tr>
<tr>
<td>Total</td>
<td>49</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Fibroids that grow into the inner cavity of the uterus (submucosal fibroids) are more likely to cause prolonged, heavy menstrual bleeding.

Figure No 4-2: submucousus

Yes 2%
No 98%
Some fibroids grow within the muscular uterine wall (intramural fibroids). If large enough, they can distort the shape of the uterus and cause prolonged, heavy periods, as well as pain and pressure.

Table No4-3: Showing Intramural fibroids.

<table>
<thead>
<tr>
<th></th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>12</td>
<td>24.5</td>
</tr>
<tr>
<td>No</td>
<td>37</td>
<td>75.5</td>
</tr>
<tr>
<td>Total</td>
<td>49</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Figure No 4-3: Intramural 2
Table No 4-4: Showing Subserosus. Fibroids that project to the outside of the uterus (subserosal fibroids) can sometimes press on the bladder.

<table>
<thead>
<tr>
<th></th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>5</td>
<td>10.2</td>
</tr>
<tr>
<td>No</td>
<td>44</td>
<td>89.8</td>
</tr>
<tr>
<td>Total</td>
<td>49</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Fibroids that project to the outside of the uterus (subserosal fibroids) can sometimes press on the bladder.

Figure No 4-4: Subserosus 3
Table No 4-5: Showing Uterus Echogenicity - Thin endometrium homogeneous – echogenic

<table>
<thead>
<tr>
<th></th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Homogenous</td>
<td>32</td>
<td>65.3</td>
</tr>
<tr>
<td>Heterogeneous</td>
<td>17</td>
<td>34.7</td>
</tr>
<tr>
<td>Total</td>
<td>49</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Figure No 4-5: Uterus Echogenicity - Thin endometrium homogeneous – echogenic
Table No 4-6: showing correlation between PSV in both R&l uterine arteries

<table>
<thead>
<tr>
<th>Correlations</th>
<th>Right PSV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left PSV</td>
<td>Pearson Correlation</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
</tr>
<tr>
<td></td>
<td>N</td>
</tr>
</tbody>
</table>

**. Correlation is significant at the 0.01 level (2-tailed).

Table No 4-7: showing correlation between TAMXV in both R&l uterine arteries

<table>
<thead>
<tr>
<th>Correlations</th>
<th>Right TAMXV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left TAMXV</td>
<td>Pearson Correlation</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
</tr>
<tr>
<td></td>
<td>N</td>
</tr>
</tbody>
</table>

**. Correlation is significant at the 0.01 level (2-tailed).
Table 4-8: Showing correlation between PI in both R&l uterine arteries

<table>
<thead>
<tr>
<th>Left PI</th>
<th>Right PI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearson Correlation</td>
<td>.847**</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.000</td>
</tr>
<tr>
<td>N</td>
<td>49</td>
</tr>
</tbody>
</table>

**. Correlation is significant at the 0.01 level (2-tailed).
Table No4-9: Showing Correlation Coefficient/ Uterus Echogenicity and uterine arteries blood flow -

<table>
<thead>
<tr>
<th>Spearman's rho</th>
<th>Correlations</th>
<th>Uterus Echogenicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right PSV</td>
<td>Correlation Coefficient</td>
<td>-.748**</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td>Right TAMX V</td>
<td>Correlation Coefficient</td>
<td>-.336*</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.018</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td>Right PI</td>
<td>Correlation Coefficient</td>
<td>-.475**</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.001</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td>Left PSV</td>
<td>Correlation Coefficient</td>
<td>-.579**</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td>Left TAMX V</td>
<td>Correlation Coefficient</td>
<td>-.500**</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td>Left PI</td>
<td>Correlation Coefficient</td>
<td>-.479**</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
</tbody>
</table>

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).
### Table No 4-10: Showing Correlation Coefficient/ submucousus and uterine arteries blood flow -

<table>
<thead>
<tr>
<th>Spearman's rho</th>
<th>Correlations</th>
<th>submucousus 1</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>Correlation Coefficient</strong></td>
<td><strong>.062</strong></td>
</tr>
<tr>
<td>Right PSV</td>
<td>Sig. (2-tailed)</td>
<td><strong>.674</strong></td>
</tr>
<tr>
<td></td>
<td><strong>N</strong></td>
<td>49</td>
</tr>
<tr>
<td>Right TAMXV</td>
<td><strong>Correlation Coefficient</strong></td>
<td><strong>-.044</strong></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td><strong>.765</strong></td>
</tr>
<tr>
<td></td>
<td><strong>N</strong></td>
<td>49</td>
</tr>
<tr>
<td>Right PI</td>
<td><strong>Correlation Coefficient</strong></td>
<td><strong>-.111</strong></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td><strong>.450</strong></td>
</tr>
<tr>
<td></td>
<td><strong>N</strong></td>
<td>49</td>
</tr>
<tr>
<td>Left PSV</td>
<td><strong>Correlation Coefficient</strong></td>
<td><strong>.005</strong></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td><strong>.972</strong></td>
</tr>
<tr>
<td></td>
<td><strong>N</strong></td>
<td>49</td>
</tr>
<tr>
<td>Left TAMXV</td>
<td><strong>Correlation Coefficient</strong></td>
<td><strong>-.071</strong></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td><strong>.628</strong></td>
</tr>
<tr>
<td></td>
<td><strong>N</strong></td>
<td>49</td>
</tr>
<tr>
<td>Left PI</td>
<td><strong>Correlation Coefficient</strong></td>
<td><strong>-.059</strong></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td><strong>.687</strong></td>
</tr>
<tr>
<td></td>
<td><strong>N</strong></td>
<td>49</td>
</tr>
</tbody>
</table>

**. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).
Table No4-11: Showing Correlation Coefficient/ intramural uterine arteries blood flow -

<table>
<thead>
<tr>
<th>Spearman's rho</th>
<th>Correlations</th>
<th>intramural 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Right</strong> PSV</td>
<td>Correlation Coefficient</td>
<td>.514**</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td><strong>Right</strong> TAMXV</td>
<td>Correlation Coefficient</td>
<td>.099</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.500</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td><strong>Right</strong> PI</td>
<td>Correlation Coefficient</td>
<td>.218</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.132</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td><strong>Left</strong> PSV</td>
<td>Correlation Coefficient</td>
<td>.269</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.062</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td><strong>Left</strong> TAMXV</td>
<td>Correlation Coefficient</td>
<td>.233</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.107</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td><strong>Left</strong> PI</td>
<td>Correlation Coefficient</td>
<td>.200</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.168</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
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</table>

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).
Table No 4-12: Showing Correlation Coefficient/ subserosus uterine arteries blood flow

<table>
<thead>
<tr>
<th>Spearman's rho</th>
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</tr>
</thead>
<tbody>
<tr>
<td><strong>Right</strong></td>
<td></td>
</tr>
<tr>
<td>PSV</td>
<td>Correlation Coefficient</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.005</td>
</tr>
<tr>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td><strong>Right</strong></td>
<td></td>
</tr>
<tr>
<td>TAMXV</td>
<td>Correlation Coefficient</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.000</td>
</tr>
<tr>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td><strong>Right</strong></td>
<td></td>
</tr>
<tr>
<td>PI</td>
<td>Correlation Coefficient</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.000</td>
</tr>
<tr>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td><strong>Left</strong></td>
<td></td>
</tr>
<tr>
<td>PSV</td>
<td>Correlation Coefficient</td>
</tr>
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<td>Sig. (2-tailed)</td>
<td>.014</td>
</tr>
<tr>
<td>N</td>
<td>49</td>
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<tr>
<td><strong>Left</strong></td>
<td></td>
</tr>
<tr>
<td>TAMXV</td>
<td>Correlation Coefficient</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.000</td>
</tr>
<tr>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td><strong>Left</strong></td>
<td></td>
</tr>
<tr>
<td>PI</td>
<td>Correlation Coefficient</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.001</td>
</tr>
<tr>
<td>N</td>
<td>49</td>
</tr>
</tbody>
</table>

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).
Table No 4-13: Showing Correlation Coefficient/Endometrial thickness and Uterus Echogenicity

<table>
<thead>
<tr>
<th>Correlations</th>
<th>Endometrial thickness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spearman's rho</td>
<td>Uterus Echogenicity</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**. Correlation is significant at the 0.01 level (2-tailed).

Table No 4-14: Showing Correlation Coefficient between Endometral thickness and age/Postmenopausal age

<table>
<thead>
<tr>
<th>Correlations</th>
<th>Endometrial thickness</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Age</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Postmenopausal age</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).
Table No 4-15: Showing Correlation Coefficient between Endometral thickness and uterus measument

<table>
<thead>
<tr>
<th>Correlations</th>
<th>Endometrial thickness</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>WIDTH</strong></td>
<td></td>
</tr>
<tr>
<td>Pearson Correlation</td>
<td>-.306*</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.033</td>
</tr>
<tr>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td><strong>DEPTH</strong></td>
<td></td>
</tr>
<tr>
<td>Pearson Correlation</td>
<td>-.567**</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.000</td>
</tr>
<tr>
<td>N</td>
<td>49</td>
</tr>
</tbody>
</table>

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).
### Table No 4-16: Showing Correlation Coefficient between Endometral thickness and uterus blood flow indecis

<table>
<thead>
<tr>
<th></th>
<th>Correlation</th>
<th>Endometrial thickness</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Right PSV</strong></td>
<td>Pearson Correlation</td>
<td>-.883**</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td><strong>Right TAMXV</strong></td>
<td>Pearson Correlation</td>
<td>-.480**</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td><strong>Right PI</strong></td>
<td>Pearson Correlation</td>
<td>-.557**</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td><strong>Left PSV</strong></td>
<td>Pearson Correlation</td>
<td>-.467**</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.001</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td><strong>Left TAMXV</strong></td>
<td>Pearson Correlation</td>
<td>-.438**</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.002</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
<tr>
<td><strong>Left PI</strong></td>
<td>Pearson Correlation</td>
<td>-.572**</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>49</td>
</tr>
</tbody>
</table>

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).
CHAPTER FIVE
DISCUSSION

5-1 DISCUSSION

The main objective of this study was to evaluate sonographically the correlation between the endometrial thickness, uterus measurements, uterus echogenicity, uterine arteries indices and any detectable fibroids.

As shown in table 4-6, 4-7, 4-8, correlations is significant at the 0.01 level between the left and right uterine arteries indices. In table 4-9, correlations between uterine echogenicity and blood flow indices is significant at the 0.01/0.05 level.

Correlations between different types of myomas (detected during study) and uterine arteries blood flow indices very clear as shown in tables 4-10, 4-11, 4-12. Correlations between submucinous / intramural fibroids and blood flow shown different significant as in subserous significant due to their location according to the uterus.

In tables 4-13, 4-15, 4-16. Correlation between endometrial thickness and uterus echogenicity, uterus measurement and uterus indices, is significant at the 0.05/0.01. Table 4-13 shown correlation is significant at 0.01/0.05 level between endometrial thickness and age/postmenopausal age and there is clear effect of the thickness proportionally to postmenopausal age while there is no effect of the thickness regarding the main age.

Goswarm RK (1988) found that the aging process affects the uterine perfusion. In general, high impedance and high velocity characteristic for the uterine arteries, though the uterine perfusion is largely dependent on age, phase of menstrual cycle.

Resistance to blood flow increases in both the main uterine and the radial arteries as the years of postmenopause progress, though the increase of
ovarian blood flow. The fact, that uterine artery RI does not change significantly in the first years of menopause strongly support the thesis that ageing process initially affects the uterus less than the ovary.

The diastolic flow decreases in postmenopause and the systolic peak increases. (Luzi O; Coata G 1993) The RI in the main uterine arteries continuously increases with the number of the postmenopausal ages, but unlike in the case of the ovarian artery, doesn’t reach the maximum in all women even at advanced ages. Absent diastolic flow in uterine arteries was found in 15% of women with 1–5 years duration of menopause, while clear interruption of diastolic blood flow was observed in the uterine artery of one-third of the women in the next five years of the postmenopausal period. More than half of the women has this finding with 11–15 years lasting postmenopause and finally, 80% of women whom LMB occurred more than 16 years ago demonstrated absent diastolic blood flow signal indicative of high vascular impedance. (kurjak et al., 1994)
5-2 Conclusions:
In summary, similar sensitivities for detecting endometrial carcinoma are reported for transvaginal sonography when an endometrial thickness of greater than 5 mm is considered abnormal and for endometrial biopsy when “sufficient” tissue is obtained. Despite the above outcome, controversy remains regarding the relative roles of these uterine imaging modalities. Future research needs to be directed toward providing effectiveness and cost-effectiveness. So, as the exclusion of endometrial cancer is very important and due to respect to woman’s health and quality-of-life endpoints, there is an urgent need in the future for better quality primary accuracy studies using ideal reference standards and good-quality criteria to guide decision making.

5-3 Recommendations
Transvaginal ultrasound should not be used as screening for endometrial cancer.
Endometrial sampling in a postmenopausal woman without bleeding should not be routinely performed.
Indications for tissue sampling of the endometrium in bleeding postmenopausal women with an endometrial thickness of greater than 4 to 5 mm should not be extrapolated to asymptomatic women.
A woman who has endometrial thickening and other positive findings on ultrasound, such as increased vascularity, inhomogeneity of endometrium, particulate fluid, or thickened endometrium over 11 mm, should be referred to a gynaecologist for further investigations.
Decisions about further investigations should be made on a case-by-case basis in asymptomatic women with increased endometrial thickening and risk factors for endometrial cancer such as obesity, hypertension, and late
menopause.

In asymptomatic women on tamoxifen, a routine ultrasound for endometrial thickening should not be performed.

Not all postmenopausal women who have asymptomatic endometrial polyps require surgery. Women found to have asymptomatic polyps on ultrasound should be triaged for intervention according to size of the polyp, age, and other risk factors.
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Ultrasound in the postmenopause 1057


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Appendix B

B-1: Postmenopausal patient with abnormal thickened endometrium & large fibroid
B-2: Postmenopausal patient with normal endometrial thickness
B-3: Postmenopausal patient with abnormal endometrial thickness & multiple fibroids
B-4: postmenopausal patient with normal endometrial thickness
B-5: Normal young patient with normal (Periovulatory phase) endometrium
B-6: Normal young patient with normal (Proliferative phase) endometrium
B-7: Normal young patient with normal (secretory phase) endometrium
B-8: Normal young patient with normal (secretory phase) endometrium & IUCD also
B-9: Normal young patient with normal (Proliferative phase) endometrium
B-10: Normal young patient with normal (secretory phase) endometrium