

Clinical Phytotherapy for Women's Health

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Materia Medica

Primary and Secondary dysmenorrhoea

Pelvic inflammatory disease

Primary and Secondary amenorrhoea

Prolactinemia

Androgen Excess

Ovarian cysts

Polycystic ovarian disease

Menorrhagia

Fibroids

Endometriosis

Pre-menstrual syndrome

Phyto-oestrogens

Differential diagnosis of gynaecological symptoms

MATERIA MEDICA FOR THE FEMALE REPRODUCTIVE SYSTEM

Uterine Tonics:

Leonurus cardiaca (Motherwort)
 Rubus idaeus (Red raspberry)
 Mitchella repens (Partridge Berry)
 Verbena officinalis (Vervain)
 Lamium album (White dead nettle)
 Hydrastis canadensis (Golden seal)
 Angelica sinensis (Dong quai)
 Caulophyllum thalictroides (Blue cohosh)

Hormonal balancers:

Vitex agnus-castus (Chaste berry)
 Chamaelirium luteum (False unicorn root)
 Verbena off. (Vervain)
 Dioscorea villosa (Wild yam)
 Cimicifuga racemosa (Black cohosh)
 Paeonia lactiflora (White peony)
 Smilax spp. (Sarsaparilla)

Anti-spasmodics:

Viburnum prunifolium (Black haw)
 Chamomilla recutita (German chamomile)
 Dioscorea villosa (Wild yam)
 Lamium album (White dead nettle)
 Lobelia inflata (Lobelia)
 Piper methysticum (Kava)
 Aletris farinosa (Unicorn root)

Oestrogenics:

Trifolium pratense (Red clover)
 Salvia officinalis (Sage)
 Humulus lupulus (Hops)
 Cimicifuga racemosa (Black cohosh)
 Glycyrrhiza glabra (Licorice)

Circulatory stimulants:

Zanthoxylum americanum (Prickly ash)
 Zingiber officinalis (Ginger)
 Achillea millefolium (Yarrow)
 Angelica sinensis (Dong quai)

Ovarian tonics:

Anemone pulsatilla (Pasque flower)
 Chamaelirium luteum (False unicorn)
 Paeonia lactiflora (White peony)

Antiseptics:

Baptisia tinctoria (Wild indigo)
 Thymus vulgaris (Thyme)
 Origanum marjorana (Marjoram)

Decongestants:

Collinsonia canadensis (Stone root)
 Lamium album (White dead nettle)
 Achillea millefolium (Yarrow)
 Zingiber off. (Ginger)
 castor oil packs

Anodynes:

Anemone pulsatilla (Pasque flower)
 Chamomilla recutita (Chamomile)
 Piscidia erythrina (Jamaican dogwood)
 Eschscholzia californica (California poppy)
 Corydalis ambigua (Yan hu suo)

Galactagogues:

Galega officinalis (Goat's rue)
 Foeniculum officinalis (Fennel)
 verbena off. (Blue vervain)

Anti-galactagogues:

Salvia officinalis (Sage)

Emmenagogues:

Artemisia absinthium (Wormwood)
 Artemisia vulgaris (Mugwort)
 Ruta graveolens (Rue)
 Mentha pulegium (Pennyroyal)
 Tanacetum vulgare (Tansy)
 Caulophyllum thalictroides (Blue cohosh)

Anti-fungals:

Calendula officinalis (Marigold)
 Thuja occidentalis (Arbor vitae)
 Commiphora molmol (Myrrh)
 Tea tree oil

Astringents:

Alchemilla vulgaris (Ladies mantle)
 Capsella bursa pastoris (Shepherd's purse)
 Geranium robertianum (Herb Robert)
 Rubus idaeus (Red raspberry)

Prostaglandin inhibitors / regulators:

Tanacetum parthenium (Feverfew)
 Zingiber off. (Ginger)
 Curcuma longa (Turmeric)
 Angelica sinensis (Dong quai)
 Verbena off. (Blue vervain)

DYSMENORRHOEA

Primary or Spasmodic or Functional

Uncoordinated contractions of the uterus, coupled with ischaemia of the uterus or cervix cause spasmodic pain. There is often an associated progesterone imbalance and prostaglandin dysregulation. This type of dysmenorrhoea is most common in young women and is rare after childbirth. The cramping pain begins with or just before the onset of bleeding. There may also be general weakness, irregularity of bowels (typically pre-menstrual constipation and diarrhoea with onset of menses), shivering or sweating, pains in the legs, nausea, faintness etc. There is no organic pathology.

Natural remedies would include:

For daily use over several months:

Bitters and liver stimulants
 Uterine tonics
 Hormonal balancers
 essential fatty acids
 calcium and magnesium
 high fibre diet
 probiotics

For symptom relief:

Anti-spasmodics
 Analgesics
 Pelvic circulatory stimulants
 Warming herbs
 Emmenagogues
 Prostaglandin regulators
 Warm baths or a hot water bottle
 Lavender / Clary sage / Chamomile essential oils
 Warming foods and hot drinks

Gentle exercise is often helpful, especially walking, swimming, dancing or stretching/yoga. Alternating hot & cold sitz baths (always ending with cold) may help, especially if you add a strong infusion of Chamomile or Lavender to the water.

Secondary or Congestive

The usual age of onset is in the 30's, or after childbirth. This type is often associated with other gynaecological conditions eg. uterine inflammations, fibroids, endometriosis, PID and especially with PMS. The major symptoms are feelings of heaviness, weakness, pains in the legs and disturbed bowel function. The discomfort usually peaks just prior to menstruation, and is often relieved by the onset of bleeding.

Natural remedies would include:

For daily use over several months:

Bitters and liver stimulants
 Uterine tonics
 Hormonal balancers
 Pelvic decongestants
 Uterine astringents
 essential fatty acids
 calcium and magnesium
 high fibre diet
 probiotics

For symptom relief:

Anti-spasmodics
 Analgesics
 Pelvic circulatory stimulants
 Warming herbs
 Emmenagogues
 Prostaglandin regulators
 Warm baths or a hot water bottle
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 Warming foods and hot drinks

Gentle exercise is often helpful, especially walking, swimming, dancing or stretching/yoga. Alternating hot & cold sitz baths (always ending with cold) may help, especially if you add a strong infusion of Chamomile or Lavender to the water.

PELVIC INFLAMMATORY DISEASE (PID)

This is a general term for inflammation of the pelvic organs in women. It may be chronic, sub-acute or acute, with signs & symptoms getting progressively worse. It is caused by bacteria ascending from the vagina, which is more common at times when the cervical canal is dilated eg. after childbirth, miscarriage, abortion, insertion of a I.U.D. or after a D & C.

PID is given different names according to the part affected: endometritis, salpingitis, oophoritis, peritonitis. Abscesses and adhesions (bands of scar tissue) may form in fallopian tubes or ovaries, or in the pelvic cavity. If an abscess ruptures, or if adhesions are so bad as to cause tubal occlusions, then infertility can result.

Symptoms of PID may be mild, moderate or severe, and may develop gradually or may come on suddenly.

Acute & sub-acute PID signs & symptoms may include:

- dull or acute lower back pain
- pain during or after intercourse
- cramps
- fever with or without rigors
- abnormal vaginal discharge
- fatigue & weakness
- pain on urination or defecation
- local lymphadenopathy
- nausea or vomiting
- congestive dysmenorrhoea
- heavy frequent menstruation
- mittelschmerz.

Symptoms of chronic PID. are similar but they may come and go over a period of months or even years. Infertility is commonly a result of chronic PID.

Allopathic treatment involves broad-spectrum anti-biotics, sometimes for several weeks. Bed rest, a high fluid intake, and local application of heat are recommended during the acute phase. Intercourse should be avoided for the duration of the illness. Where there are abscesses it may be necessary to drain them because they are often inaccessible to anti-biotics.

Natural remedies would include:

- | | |
|--------------------|------------------------|
| Pelvic antiseptics | Uterine tonics |
| Analgesics | Circulatory stimulants |
| Immune modulators | Lymphatic cleansers |
| Blood cleansers | |

Hot poultices of anti-inflammatory and anodyne herbs can also be useful. Castor oil or clay packs over the abdomen are particularly good to deal with congestion, pain and inflammation.

Vitamin C should be supplemented to bowel tolerance and Iron supplementation should be avoided.

AMENORRHOEA

This refers to the absence of menstruation.

Primary

This means that menstruation has never begun, and there is usually also an absence of normal sexual development (pubic & axillary hair, breast growth). If the girl is less than 14 years old she may be just a 'late developer', but if older than 14 then there is the possibility that she is lacking in some of the developmental

hormones, and should be assessed by an endocrinologist.

Possible causes of primary amenorrhoea

- extreme or prolonged stress
- excessive exercise
- very low body weight (body mass index < 20)
- hypothalamic, pituitary or ovarian abnormalities
- androgen excess
- chromosomal abnormalities
- uterine or cervical abnormalities - usually congenital (eg absence of the uterus, stenosis of the cervix)

Stress

Phase 1: alarm response → sympathetic stimulation of adrenal medulla → epinephrine → fight or flight response

Phase 2: resistance response → sympathetic stimulation of hypothalamus → CRH → anterior pituitary → STH to liver for release of glycogen, ACTH to adrenal cortex for release of glucocorticoids and mineralocorticoids, TSH to thyroid gland for release of thyroid hormone

Phase 3: exhaustion response → adrenal exhaustion and nutrient depletion

In prolonged stress where phase 2 persists the hypothalamic secretion of GnRH is disrupted and pituitary - ovarian stimulation is disturbed. Can lead to ovulatory failure, luteal disruption, excessive or inhibited menstruation.

Excessive exercise and / or low body weight

Body mass index calculated by weight in kg by height in metres squared. BMI < 20 = underweight. BMI > 26 = overweight. BMI > 30 = obesity.

17% total body weight must be fat in order to menstruate and 22% to have regular periods. Conversion of androgens to oestrone by aromatase enzymes occurs in adipose tissue and muscle.

Exercise promotes oestrogen clearance. Too much exercise can cause low oestrogen and menstrual disruption., as well as dropping the body mass index too low.

Hypothalamic abnormalities

Stress disrupts GnRH as described above. Lesions of the hypothalamus may cause reduced GnRH.

Pituitary abnormalities

Benign or malignant lesions may cause hyperprolactinemia → amenorrhoea. Impaired pituitary oxygenation secondary to a blood clot may disrupt gonadotropic hormone production.

Ovarian abnormalities (failure to ovulate)

Polycystic ovarian disease (PCOD). Cushing's syndrome. Congenital adrenal hyperplasia. Androgen secreting tumours. Hypothyroidism → lowered SHBG → excessive availability of oestrogen. Hyperthyroidism → increased conversion of androgens to oestrogen.

Secondary

This means that normal menstruation has ceased. **This is always assumed to be due to pregnancy until proven otherwise.** It is not uncommon to miss one or two menstrual periods so a diagnosis of Secondary

Amenorrhoea is usually not made until there has been no period for 6 months.

Possible causes of secondary amenorrhoea

- extreme or prolonged stress
- excessive exercise
- very low body weight (body mass index < 20)
- disturbance of the hypothalamic/ pituitary/ ovarian axis
- thyroid dysfunction
- androgen excess
- ovarian cysts or tumours
- adrenal hyperplasia
- post-contraceptive pill
- certain other prescription medications
- acquired cervical stenosis

The underlying pathology must be identified before commencing treatment for secondary amenorrhoea.

Disturbance of the hypothalamic/ pituitary/ ovarian axis

Same possible causes as for primary amenorrhoea plus:

Phenothiazide drugs (eg Largactil) increase prolactin hormone → menstrual irregularities or amenorrhoea. Occasionally anti-hypertensives can cause amenorrhoea. Post-contraceptive pill. Breast-feeding. Premature ovarian failure (idiopathic).

Hyper-prolactinemia

May or may not cause galactorrhoea (inappropriate milk production). Usually causes scanty menses (oligorrhoea) or amenorrhoea. May be caused by:

- prolactin-secreting pituitary tumours
- hypothyroidism
- epilepsy → circulatory disturbance to pituitary gland
- spinal cord tumours
- excessive or prolonged stress → transient prolactinemia
- excessive breast stimulation
- excessive exercise → reduced oestrogen → disturbance of negative feedback loop (body thinks it is pregnant)
- phenothiazines and other dopamine depleting drugs (dopamine is also known as prolactin-inhibition factor)
- anti-ulcer drugs
- high-oestrogen contraceptive pill
- some anti-hypertensive drugs
- opiates, cocaine and alcohol (especially beer which is oestrogenic)

Usually oestradiol and most androgens are low but adrenal androgens are elevated. Alpha-5-reductase is usually low so androgens not converted to potent form therefore little masculinizing effect. SHBG reduced.

Allopathic treatment is usually *bromocriptine* but on-going treatment is required unless underlying cause is treated. Prolactin is highest when oestrogen is highest (just before ovulation and in luteal phase of cycle). Also elevated between 3 - 5 am and 2 - 4 pm, after eating fats or protein, after exercise, after breast stimulation and during stressful events.

Natural remedies would include:

Zinc and B6 which are required for synthesis of dopamine
 Phosphatidyl serine increases dopamine production
 S-adenyl-methionine increases dopamine production
 Vitex agnus-castus

Androgen excess

Androgens (DHEA, androstenedione, androstenediol & testosterone) made from 17-alpha-OH-pregnenolone. Androgens can be converted by aromatase enzymes into oestrone in hair follicles, skin, brain, bone, muscles, fatty tissue and bone marrow. Testosterone is the most abundant androgen and the most potent. 25% comes from ovaries, 25% from adrenal glands and 50% from aromatase conversion.

Excess androgens may be due to:

- ovarian disease eg. PCOD or androgen-secreting tumours
- adrenal gland disorders eg. congenital or acquired hyperplasia, androgen-secreting tumours
- post-menopausal state
- obesity
- prescription drugs eg. Dilantin, progestogens, corticosteroids, corticotrophics, anabolic steroids
- reduced SHBG (increased availability of free androgens in circulation)

Excess androgens causes masculinizing effects:

- hirsutism
- androgenic alopecia
- acne
- menstrual irregularity, oligomenorrhoea, amenorrhoea
- voice deepening and breast atrophy in advanced cases
- increased sensitivity to androgens in the pilo-sebaceous unit may cause hirsutism, acne and male pattern baldness in the absence of elevated androgens.

Tests for excess androgens include:

- elevated serum testosterone (best test of ovarian androgen production)
 - reduced SHBG
 - elevated DHEA (best test for adrenal hyperplasia or androgen-secreting adrenal hormones)
- (note that acquired adrenal hyperplasia also associated with fluid retention, immune disturbance and hypertension)

Treatment of excess androgens depends on underlying cause. Cyproterone acetate often used as a competitive inhibitor of androgens by blocking receptor sites. Adrenal hyperplasia is treated with a corticosteroid which reduces adrenal production. Treatment with oestrogen will elevate SHBG. Some birth control pills have types of progesterone that is itself androgenising while others may be helpful by raising oestrogen that counters the androgenic effects.

Natural remedies include:

- Phyto-oestrogens and meat avoidance which can raise the levels of SHBG
- Smilax spp. and Turnera are mild competitive inhibitors of androgens
- Glycyrrhiza glabra and Paeonia lactiflora increase the rate of conversion of testosterone to oestrogen in the tissues
- Reducing obesity, high fibre also raise SHBG

OVARIAN CYSTS

Ovarian cysts may be solid, semi-solid or fluid filled. They can be symptomatic or asymptomatic, benign or malignant. They may be self-limiting (resorbed), spontaneously rupture or require surgery. Often considered by the natural therapist to be indicative of a toxic overload. One in every three ovarian cysts in women over 45 years is malignant.

Symptoms may include:

- Mittelschmerz
- Mid-cycle bleeding
- Congestive dysmenorrhoea
- Hormonal disturbance and PMS
- Infertility
- Dyspareunia
- Severe pain, haemorrhage and shock with rupture

Benign cysts

Serous and mucinous

- May become very large.
- Often on a stalk or pedicle.
- Torsion of pedicle often causes severe pain and may cause cyst to rupture.
- Serous cysts filled with thin, clear fluid
- Mucinous cysts filled with thick, sticky mucus.
- Adhesions and peritoneal irritation more common with mucinous cysts
- Both types may occasionally become malignant

Fibromas

- Fibrous and solid, akin to a uterine fibroid

Dermoid cysts (teratomas)

- Improper differentiation of foetal cells that become the ovum → hair, teeth, bones and skin develop in the cyst.
- Often on a stalk and prone to torsion
- Rarely rupture but severe peritoneal irritation and pain will occur if they do
- Frequently become malignant

Brenner cysts

- Small and benign, occasionally become malignant
- More common post-menopausal and often misdiagnosed because patient not expecting gynaecological problems

Chocolate cysts

- Occur in endometriosis
- Filled with old, coagulated blood
- Do not rupture nor resorb
- May prevent ovulation
- May cause severe ovulation pain

Functioning cysts

- Benign but progressive
- Hormone-secreting and disruptive to menstrual cycle.
- Usually secrete oestrogens, occasionally androgens

Functional / physiological cysts

Deviation of normal ovarian functions rather than abnormal cell growth. Non-malignant.

- Follicular cysts are quite common. They occur when a follicle fails to extrude the mature egg or when multiple developing follicles fail to disintegrate after ovulation.
- Small and often asymptomatic
- Rarely rupture, occasionally stalk will twist
- Usually resorbed

- Luteal cysts occur when disintegration of the corpus luteum is delayed
- Small and usually resorbed after onset of menses
- Occasionally become large, blood filled and likely to rupture causing severe pain
- May cause dull aching pain that ceases with onset of menses as the corpus luteum disintegrates
- May interfere with (suppress) progesterone production and cause menstrual disturbance and PMS

Natural treatment of ovarian cysts includes:

- Avoidance of exogenous oestrogens in diet
- Cleansing and detoxification programs
- Avoidance of animal fats and supplementation with essential fatty acids and GLA
- Avoidance of coffee and other liver stressors
- High fibre diet
- Ovarian tonics especially Chamaelirium luteum, Paeonia lactiflora and Anemone pulsatilla
- Pelvic decongestants and pelvic circulatory stimulants
- Astringents
- Castor oil packs

Polycystic ovarian disease (PCOD)

Characterised by the presence of multiple ovarian cysts and hormonal irregularities. One or both ovaries may be affected, suggesting the primary cause is ovarian malfunction rather than disruption of the hypothalamic - pituitary - ovarian axis.

Signs and Symptoms include:

- Menstrual irregularities especially oligomenorrhoea, secondary amenorrhoea and erratic cycles
- Androgen excess → hirsutism, deepening of the voice, male pattern baldness, breast atrophy
- Commonly associated with elevated LH and normal or low FSH.
- Elevated acyclic oestrogen due to peripheral conversion (aromatisation) of androgens to oestrogens (chiefly androstenedione to oestrone).
- Reduced SHBG → increased availability of androgens
- Ovarian pain
- Ovulation failure and infertility
- Obesity and insulin resistance
- Increased risk of endometrial cancer due to failure of uterine lining to shed regularly

PCOD is easily confused and commonly mis-diagnosed as Cushing's syndrome, adrenal hyperplasia,

hormone-secreting ovarian cysts, hormone-secreting tumours of ovary or adrenals, hyperprolactinemia or hypothyroidism.

Postulated causes include:

- Familial tendency (genetic predisposition)
- Ovarian malfunction → reduced oestradiol (ovarian oestrogen) and excessive ovarian androgens. These androgens inhibit normal follicular development and ovulation → accumulation of undischarged follicles that become cystic.
- Excessive weight predisposes to aromatisation of androgens into oestrogens → elevation of oestrone → reduced FSH → inadequate stimulation of developing follicle and follicle maturation failure → tendency for undischarged follicles to accumulate in ovary and become cystic.
- Excessive adrenal androgen production → elevated serum androgens and increased aromatisation to oestrone → elevated LH (and depressed FSH) → increased ovarian androgen production.
- Low FSH impairs ability of follicle cells to convert ovarian androgens to oestrogens → inadequate stimulation of developing follicle and follicle maturation failure → tendency for undischarged follicles to accumulate in ovary and become cystic.

Natural treatments attempt:

- Reduction of masculinizing effects of androgens
- Stimulation of ovulation
- Promotion of endometrial shedding

Natural treatments include:

- Avoidance of exogenous oestrogens in diet
- Cleansing and detoxification programs and avoidance of coffee and other liver stressors
- Avoidance of animal fats and supplementation with essential fatty acids and GLA
- High fibre diet
- Treat blood sugar disturbances
- Ovarian tonics and normalisers especially Chamaelirium luteum, Paeonia lactiflora and Anemone pulsatilla
- Pelvic decongestants and pelvic circulatory stimulants and castor oil packs
- Emmenagogues
- Astringents
- Phyto-oestrogens
- Avoid Vitex because it may increase LH
- Humulus, Cimicifuga and Lycopus will reduce circulating androgens
- Smilax may act as a competitive inhibitor for androgens

MENORRHAGIA

This means a very heavy or prolonged menstrual flow. It is not an illness in itself but is a symptom of several possible underlying pathologies. Elevated prostacyclin 2 (in the PG2 series) reduces blood clotting and dilates blood vessels. It has been shown to be elevated in functional menorrhagia in the absence of other overt disease processes. A relative oestrogen excess and high animal fats in the diet may increase prostacyclin 2.

Pelvic congestion:

This may be due to poor pelvic circulation, inflammation or tumours. Fibroids and endometriosis are common causes. The actual cause needs to be identified and treated. Herbs that may help include: Uterine tonics, astringents, pelvic decongestants. Colonic irrigation and exercise may also be beneficial as well as bitter remedies to cleanse and tone the liver and thus remove congestion. Constipation should be remedied. Castor oil packs and pelvic exercises may be helpful.

Capillary fragility or impaired clotting

This may be due to Vitamin C or bioflavonoid deficiency, hyaluronidase imbalances, free-radical damage or blood clotting problems including anaemia and liver disease.

Herbs that may help to reduce capillary fragility include:

Fagopyrum esculentum (Buckwheat)	Tilia europea (Linden)
Achillea millefolium (Yarrow)	Crateagus oxyacantha (Hawthorn)
Vaccinium myrtillus (Blueberry)	Aesculus hippocastanum (Horse Chestnut)
Centella asiatica (Gotu kola)	Equisetum arvense (Horsetail)

Pelvic Inflammatory Disease

Hormonal imbalance associated with PMS

Hypertension

Diabetes or renal disease

Intra-uterine contraceptive device

Tubal ligation

Herbs to use symptomatically for menorrhagia would include:

Alchemilla arvensis	Capsicum minimum
Capsella bursa-pastoris	Urtica dioica
Geum urbanum	Geranium robertianum
Cinnamomum zeylanicum	
Agrimonia eupatoria	

Phyto-oestrogens can block oestrogen receptor sites and reduce endometrial proliferation which reduces bleeding. Do not Angelica sinensis in menorrhagia as it can worsen bleeding.

Supplements should include:

Iron (vegetable source)

Beta carotene or vitamin A. Women with menorrhagia have reduced vitamin A levels. Vitamin A is required for proper function of *beta-3-dehydrogenase* which is necessary to the formation of oestradiol.

Vitamin K to enhance blood clotting. Normally manufactured by bowel flora and present in dark green leafy vegetables. Adding chlorophyll and alfalfa supplements may be helpful. Probiotics promote good bacterial balance.

Bioflavonoids reduce capillary fragility. Essential fatty acids and gamma-linolenic acid

A high fibre diet promotes oestrogen clearance. Avoid animal fats that promote the PG2 series.

FIBROIDS

These are a type of benign tumour, occurring in the uterus, and occasionally on the cervix. Their development appears to be related to oestrogen because they are more common in women of menstruating age. In women who are taking the contraceptive pill, where oestrogen levels are unusually high, the rate of growth of fibroids is accelerated. Hormonal disturbances with elevated oestrogen and reduced progesterone promote fibroid growth. Pregnancy and breast feeding reduces fibroid growth. Fibroids usually cease to grow, and may even shrink after menopause. Obesity and high coffee consumption promotes fibroid growth. Tobacco smoking reduces fibroid growth, presumably due to impaired micro-circulation. African-American women are up to three times more likely to develop fibroids than Caucasian or Asian women. It is not unusual for fibroids to be asymptomatic. The severity of symptoms will depend on where the fibroid is located and how large it grows.

The majority of fibroids grow within the thick uterine muscle, and are called intramural or interstitial. If they expand towards the outer surface of the uterus they are called subserous or subperitoneal. If they grow towards the uterine cavity they are called submucosal.

Symptoms may include:

- Menorrhagia (heavy periods)
- Metrorrhagia (inter-menstrual bleeding)
- Chronic pain in lower back, supra-pubic area or upper legs
- Dyspareunia (pain on intercourse)
- Congestive / Secondary dysmenorrhoea

Either submucous or subserous fibroids can become pedunculated (ie. develop a 'stalk'). Occasionally this may undergo torsion which compromises blood supply and causes acute pain and ultimately necrosis.

Other possible complications of fibroids include:

- Adhesions leading to chronic pain.
- Infertility due to distortions of uterine shape & lining.
- Varicose veins or haemorrhoids due to compression of pelvic veins.
- Urinary retention due to compression of the bladder or of urethra.

The only allopathic treatment available is surgery. This may mean a 'D&C' or myomectomy (fibroid removal) but more usually involves hysterectomy.

Holistic treatment of fibroids

With herbs it is difficult to cause fibroids to regress, but it may be possible to retard or inhibit their growth.

A cleansing and detoxification diet will be helpful in removing toxins from the body and opening channels of elimination.

A maintenance diet should be based around low oestrogen. This means avoiding all farmed meats, eggs and all dairy products. All dietary fats should be kept to a minimum, polyunsaturated vegetable oils being preferable to any animal fats. Salt, sugar, coffee and alcohol should be avoided because of their stressful effect on the liver. Any liver congestion will lead to raised levels of circulating oestrogen and thus aggravate fibroid growth. Seaweeds should be included in the diet to boost thyroid function and enhance normal oestrogen production. Essentially the diet should emphasize fresh fruits and vegetables, unrefined carbohydrates and legumes/pulses.

Supplements would include:

- High potency B complex (at least 100 mg. of each B vitamin daily). A deficiency of B vitamins may lead to higher than usual oestrogen levels by interfering with the livers ability to inactivate it. Conversely, high levels of oestrogen can lead to low B vitamin levels.
- Vitamin E (600 iu. daily). This helps to normalize oestrogen levels and regulates bleeding.
- Vitamin C and Bioflavonoids to bowel tolerance. These strengthen capillaries and minimise bleeding.
- Iron (20 mg. daily). This will help to prevent anaemia.

Herbal remedies would include:

- Uterine tonics
- Circulatory stimulants
- Pelvic decongestants
- Pelvic astringents
- Anodynes

Phyto-oestrogens may be used to competitively inhibit oestrogen receptor sites.
Castor oil or clay packs will be useful for pelvic decongestion

ENDOMETRIOSIS

This refers to the growth of endometrial tissue outside of it's usual location. It is believed to affect as many as 10% of adult women, being slightly more common in the late 20's to mid 30's. The aetiology is unclear but there are 3 postulated causes:

- 1) Retrograde flow of menstrual tissue out of the fallopian tubes into the pelvic cavity. Note that all menstruating women have some menstrual blood in the pelvic cavity. Cervical stenosis or vaginal blockage increases incidence of endometriosis. Women with endometriosis have higher than usual levels of prostaglandins in the peritoneal fluid which may cause increased contractions of the uterus and dilation of the fallopian tubes which would facilitate retrograde blood flow.
- 2) Spread of endometrial tissue via blood or lymph.
- 3) All reproductive organs develop from the same foetal tissue. Some of this may fail to differentiate properly, giving rise to ectopic endometrium.

- A relative oestrogen excess and / or unusual sensitivity to oestrogen may cause or aggravate the problem.
- A history of early menarche, short cycles and heavy periods increases the risk of developing endometriosis.
- Pregnancies reduce the risk.
- There is a strong family correlation.
- Regular exercise decreases the risk, probably by increasing oestrogen clearance, but heavy exercise during menstruation increases the risk, probably by aggravating retrograde flow.
- Use of the IUD increases the likelihood of developing endometriosis, probably by promoting retrograde flow.
- 5 - 7 grams of caffeine / month (equivalent to two coffees or four teas per day) increases development and symptomatology of endometriosis.
- Intercourse during the menses has been suggested as an aggravating factor, probably by increasing retrograde flow.

Endometrial tissue anywhere in the body is sensitive to the cyclical hormonal fluctuations; swelling up and

becoming more glandular with oestrogen and progesterone, then degenerating with the drop in hormones around day 26-28. Where there is ectopic endometrium the degenerating tissue cannot escape from the body and this gives rise to a variety of symptoms.

Adenomyosis

This refers to the growth of endometrium within the muscle wall of the uterus. It is more likely over the age of 35 and in the multi-parous. It is associated with fibroids in 50% of cases.

Symptoms include:

- Severe dysmenorrhoea - spasms and cramping
- Menorrhagia
- Increased frequency of menstruation
- Enlarged hard uterus

Extra-uterine

This refers to the growth of endometrium outside of the uterus. It is much more common than the previous type. It is more common in women who have not had children. Endometrial growth may occur anywhere in the pelvic cavity: ovaries, broad ligaments, around the bladder, sigmoid colon, rectum and occasionally on the intestines. Rarely it may occur far removed from the pelvis eg. axillae or lungs.

Ectopic endometrium occurs on the ovaries in about 60% of cases. It often becomes encapsulated by the ovary to form a cyst. Over time as blood trapped in the cyst thickens and turns dark brown the so called 'chocolate cysts' appear.

Symptoms of extra-uterine endometriosis include:

- Menstrual irregularities especially menorrhagia and increased frequency of menstruation as well as spotting and metrorrhagia and erratic cycles..
- Progressively worsening dysmenorrhoea - heavy and dragging
- Dyspareunia.
- Chronic lower abdominal pain.
- Pain on defecation.
- Infertility due to adhesions.
- Tender, enlarged ovaries.
- Anaemia.
- PMS

Elevated macrophage counts are found in the peritoneal fluid in the early stages of endometriosis and depressed levels as the disease progresses. They may contribute to associated infertility by engulfing and destroying sperm or the ovum. They may also increase the rate of adhesion formation. Macrophages promote PGE2 which increases uterine spasms and dilates blood vessels which can worsen the condition. Women with endometriosis have reduced levels of Natural Killer T cells in the peritoneal fluid and this reduces the ability of the body to attack and remove unwanted materials.

In response to the irritation and inflammation caused by blood in the pelvic cavity, widespread adhesions may occur. These may seriously distort the usual positioning of the pelvic organs. Occasionally endometriosis involves the bladder and urethra. This may cause dysuria, urinary retention or frequency of urination, but only very rarely is there blood in the urine.

Allopathic treatment usually involves hormonal manipulation in the first instance, or surgery if that fails.

Micro-surgery via laparoscopy may be used to remove adhesions and cysts. Hysterectomies are common.

Holistic Treatment Of Endometriosis

Birth control pills or other hormonal treatments should be discouraged as they make it difficult to assess the success of herbal and holistic treatments and anyway, they do not constitute a cure but simply a masking of the symptoms. The diet should emphasize fresh fruits and vegetables, unrefined carbohydrates, legumes and plenty of water. A cleansing and detoxification diet may be helpful at the onset of treatment and juice fasting or raw foods can be helpful in the 4 or 5 days preceding a period. All animal foods should be minimized, especially animal fats. Exercise is very important to help with stress control and also to ensure that there is adequate circulation in the pelvis. Walking, bicycling, yoga, swimming and a rebounder are all recommended. Strenuous exercise is not recommended.

Herbal remedies

- Uterine tonics
- Ovarian tonics
- Pelvic decongestants
- Astringents
- Anodynes
- Phyto-oestrogens
- Prostaglandin regulators

As with any congestive pelvic condition, the use of castor oil or clay packs is highly recommended.

PRE - MENSTRUAL SYNDROME

This condition affects, to some degree at least, up to 75% of all women of menstruating age. It usually occurs from 2 - 14 days prior to menstruation, and is thought to be primarily a problem of inappropriate hormone secretion or inappropriate bodily response to hormones.

There is a wide spectrum of symptoms in PMS but some common denominators include:

- Unusually high oestrogen and low progesterone or low oestrogen and high progesterone 5 - 10 days before the period.
- Elevated Prolactin levels.
- Elevated FSH levels 6 - 9 days before the period.
- Elevated Aldosterone levels 2 - 8 days before the period.
- Hypothyroidism.

Note that symptoms only occur in those cycles in which ovulation happens. This explains why some months the women have no symptoms and other months are severe.

PMS is classified into 4 sub groups, each with specific symptoms, hormonal pictures and metabolic abnormalities.

PRE - MENSTRUAL SYNDROME CLASSIFICATION

<i>SUBGROUP</i>	<i>SYMPTOMS</i>	<i>MECHANISMS</i>	<i>PREVALENCE (%)</i>
PMS A	Anxiety Irritability Mood swings Nervous tension	High oestrogen Low progesterone (leads to elevated epinephrine, nor-epinephrine & serotonin and reduced dopamine in the brain)	65 - 75%
PMS C	Food cravings Headache Fatigue Dizziness Palpitations	Low prostaglandin PGE1 leads to excessive insuline release and hypoglycemia	25 - 35%
PMS D	Depression Crying Forgetfulness Confusion Insomnia	Low oestrogen High progesterone (leads to elevated aldosterone and too rapid breakdown of excitatory neurotransmitters)	23 - 37%
PMS H	Fluid retention Weight gain Breast tenderness Abdominal distention Swollen hands & feet	Stress and elevated oestrogen lead to increased angiotensin II which causes fluid retention. Aggravated by reduced dopamine which causes elevated prolactin & aldosterone.	65 - 72%

Some authorities also mention PMS type P (for pain) associated with prostaglandin imbalance consequent upon elevated oestrogen and excessive intake of animal fats which promote the PG2 series.

PMS A

In this type of PMS there is an excess of oestrogen relative to progesterone. Oestrogen stimulates the brain by altering the ratio and levels of certain neurotransmitters. Specifically, oestrogen raises levels of adrenalin (→ anxiety), nor-adrenalin (→ hostility and irritability) and serotonin (→ nervous tension, drowsiness, palpitations) and decreases levels of dopamine (→ reduced feelings of mental relaxation, impaired mental clarity, increased prolactin). Oestrogen also affects mood by blocking the action of vitamin B6 and decreasing the body's ability to maintain normal blood sugar levels. There is also a possibility that high oestrogen and low progesterone may impair the functioning of the endorphins which promote mental relaxation. The situation is aggravated by poor nutrition in general (especially an excess of sugar) and by stress. Epinephrine and nor-epinephrine are competitive inhibitors of progesterone receptors which further compounds the problem.

PMS C

The symptoms of this type of PMS are similar to those of hypo-glycaemia, and following general guide lines to control hypoglycaemia will be beneficial. Glucose Tolerance tests performed on PMS C sufferers in the 10 days preceding the period indicate an excessive secretion of insulin in response to blood sugar levels, the

insulin : blood sugar ratio being normal at other times of the month. The exact mechanism by which this comes about is not clearly understood but a deficiency of PGE1 is known to inhibit glucose-induced insulin production and thus may be operative in PMS C. Sodium chloride (salt) enhances insulin response to sugar ingestion and low pancreatic magnesium levels leads to increased insulin production. Thus when treating this form of PMS it is advisable to avoid table salt and supplement with 300 - 600 mg. of magnesium daily. A supplement of Evening Primrose Oil (500 mg. twice a day) will help to regulate the prostaglandin picture. PMS C may be aggravated by elevated oestrogen which inhibits the breakdown of epinephrine, nor-epinephrine and serotonin while increasing the breakdown of dopamine.

PMS D

This type of PMS appears to be due to an excess of progesterone relative to oestrogen, the progesterone acting on the brain as a depressant. It may also be aggravated by the low levels of oestrogen which promotes breakdown of mood-enhancing neurotransmitters. It is more common in peri-menopausal women and those with low oestrogen due to excessive thinness or excessive dietary fibre. PMS D is aggravated by deficiencies of B6 and magnesium, and by stress.

An unusual feature of this type of PMS is that there tends to be an excess of lead in the plasma and deposited in the hair. This is thought to be due to the relative deficiency of magnesium which thus favours the uptake of lead. Detoxification and cleansing may thus be an appropriate part of the treatment of PMS D.

L-Tyrosine supplementation (500 mg. twice daily) may be useful as an anti-depressant, along with magnesium.

PMS H

The symptoms of PMS H are essentially those of water retention, brought about by stress, low magnesium and high oestrogen which disrupt the normal ACTH/Aldosterone axis. A vicious circle can occur in which the high aldosterone level increases the renal output of magnesium which further raises the aldosterone level. Stress and elevated oestrogen lead to increased angiotensin II which causes fluid retention. And elevated oestrogen also raises aldosterone. This may be aggravated by reduced dopamine which causes elevated prolactin & aldosterone.

Because pyridoxine requires magnesium for conversion into its active form, a deficiency of vitamin B6 is commonly associated with PMS H.

Remember that any one person may have symptoms from each of the sub-groups - there is a lot of overlap.

HOLISTIC TREATMENT OF PMS

Diet

- All refined carbohydrates should be limited (sugars, honey, white flour products) and all concentrated carbohydrates should be eaten in moderation (dried fruits, maple syrup, fruit juice etc).
- Vegetable protein intake should be increased (legumes and unrefined carbohydrates).
- All dairy products should be decreased or avoided. They are full of artificial hormones which will disrupt your own hormone levels, and the high fat content will disrupt your prostaglandin balance.

- All animal fats should be decreased, while polyunsaturated vegetable oils should be increased to boost intake of essential fatty acids.
- Increase intake of green leafy vegetables to boost fibre intake and promote hormone clearance, but restrict intake of the brassicas (cabbage, cauliflower, broccoli, brussel sprouts) because they can aggravate hypothyroidism.
- Use only hormone-free, organic meat and chicken, and increase your intake of fish.
- Restrict salt, alcohol, coffee, tea, chocolate and cocoa.

Lifestyle

- Exercise regularly to help with stress reduction and weight control. Do not over exercise and lose too much weight.
- Expose your eyes and body to sunlight on a regular basis to help regulate pineal gland function.

Supplements

- Vitamin B 6 200 - 400 mg (less in first three weeks of cycles, more in last week of cycle)
- Vitamin B complex 100 mg
- Magnesium chelate 300 - 600 mg (less in first three weeks of cycles, more in last week of cycle)
- Calcium citrate equal to magnesium

Herbal Remedies

In the botanical treatment of PMS the 2 main areas to concentrate on are hormonal balancing and supporting the liver function. Adaptogens may be very helpful as well. Phyto-oestrogens may be particularly helpful.

Hormonal balancers include:

Vitex agnus-castus (Chaste berry)
 Chamaelirium luteum (False unicorn root)
 Verbena off. (Vervain)
 Dioscorea villosa (Wild yam)
 Cimicifuga racemosa (Black cohosh)
 Paeonia lactiflora (White peony)

Vitex agnus-castus is the herb of first choice to balance the hormones and regulate the menstrual cycle. **Glycyrrhiza glabra** may also be useful to support the adrenal glands and thus indirectly regulate hormone production as well as helping to balance the blood sugar.

To support the liver in its role of deactivating hormones, **Taraxacum off.** or other bitters would be appropriate.

Symptomatic herbal treatment may be helpful:

- Diuretics for PMS H.
- Relaxing or sedative nervines for PMS A
- tonic nervines and anti-depressants for PMS D
- Oestrogens in PMS D.
- Progestogenics in PMS A.
- Anodynes for headache or muscle aching.
- Peripheral circulatory stimulants for confusion or forgetfulness.

PHYTO-OESTROGENS

In terms of total oestrogen exposure over a woman's life time, we are all suffering from hyper-oestrogenism today. Modern women have between 350 and 400 periods while our ancestors probably had 30 - 40 !

Oestrogen stimulates cell proliferation (increase in cell number) as well as increase in oestrogen receptors therefore over-exposure may pre-dispose to cancerous changes in sensitive tissues (hence increasing levels of breast cancer).

Three types of oestrogen:

- Oestradiol - most active form, produced by ovaries and converted into oestrone before circulating to other tissues. Oestradiol converts into *2-hydroxyoestrone* which has a mild proliferative effect and *16-hydroxyoestrone* which is much more active and is noted to be elevated in the breast tissue of women with breast cancer. These two metabolites cannot be made simultaneously. Production of *16-hydroxyoestrone* is favoured in the presence of xeno-oestrogens.
- Oestrone - produced by conversion from ovarian oestradiol and from peripheral aromatisation of androgens.
- Oestriol - weakest form. Formed in the kidney and mostly excreted in urine.

Oestrogens in blood stream are conjugated in the liver for excretion via bile. *Beta-glucuronidase* enzyme from intestinal bacteria can convert conjugated excreted oestrogen back into an active form that can be reabsorbed. Meat eaters have higher levels of the bacteria that produce this enzyme and slow transit time (more common in meat eaters) will aggravate the problem.

Xeno-oestrogens include many pesticide residues, agricultural chemicals, hormones used to treat animals or fish, polychlorinated biphenols (PCBs), plastics and paints. Soft plastics are worse than hard plastics, especially if they are heated or come into contact with fatty foods.

Phyto-oestrogens

Plant molecules that have structural similarity to endogenous oestrogens and can bind with oestrogen receptors in the body. Up to 400 times smaller than endogenous oestrogens and much weaker. Can be *isoflavones*, *coumestans*, *lignans*, *triterpenoid* & *steroidal saponins* and *resorcylic acid lactones*. Highest in germinating (sprouting) seeds and in plant when it is going to seed. Also higher when plant is stressed by drought, pests or disease. May be a protective device of plants to lower fertility of grazing animals and thus reduce plant deaths.

Phyto-oestrogens are anti-oestrogenic in pre-menopausal women because they are competitive antagonists of oestrogen receptor sites (ie they fill the site and prevent endogenous oestrogen working there, but the effect of the plant oestrogen is much weaker). In peri and post-menopausal women they are oestrogen synergists because even the slight stimulus from the plant molecule is greater than nothing, and the phyto-oestrogen can summate with residual endogenous oestrogen.

Oestrogenic isoflavonoids include *genistein*, *diadzein*, *biochanin A* and *formononetin*. Found especially in

soya alfalfa and red clover. Formononetin is converted to diadzein in the plants and then to *equol* by intestinal flora. Equol is the strongest of the phyto-oestrogens but still about 1000 times weaker than oestradiol. Biochanin A is converted to genistein which is about 100,000 times weaker than oestradiol. Biochanin A also carcinogenic activity in cells.

Oestrogenic lignans are found in seeds and grains. Very high in flax seed (but not extracted into oil). Must be converted by bowel flora into active form know as *enterolactone* and *enterodiol*.

Oestrogenic isoflavonoids and lignans are notable cancer-preventatives by virtue of reducing the effects of endogenous oestrogens. This is especially true in pre-menopausal women and obese post-menopausal women where peripheral aromatisation of androgens to oestrogen is high. Studies into the treatment of women with oestrogen dependent cancers by administration of phyto-oestrogens are scarce and reliable information is lacking. Caution is recommended.

Oestrogenic isoflavonoids and lignans stimulate hepatic production of SHBG which serves to reduce availability of androgens to undergo aromatisation and of oestrogens. Phyto-oestrogens also inhibit aromatase enzyme which further reduces endogenous oestrogens.

Coumestrol inhibits bone resorption and promotes bone remineralization in laboratory experiments and may be useful in preventing and treating osteoporosis.

Saponins have structural similarity to cholesterol and hence to hormones manufactured from cholesterol including oestrogens, progesterone, androgens and glucocorticoids (cortisol).

Triterpenoid saponins with steroid-like structure and oestrogenic effects are found in *Cimicifuga racemosa*, *Panax ginseng* and *Glycyrrhiza glabra*. They cause an indirect oestrogenic effect by modification of hypothalamic - pituitary - ovarian axis. *Cimicifuga* reduces LH and is helpful in treating PCOD. Glycyrrhetic acid (metabolized from glycyrrhizin by bowel flora) reduces synthesis of testosterone from androstenedione. Whole herb extracts of *Glycyrrhiza* reduces prolactin and is useful in treating hyperprolactinemia.

Triterpenoid saponins can also act as anti-inflammatories by a modulation of the hypothalamic - pituitary - adrenal axis.

Steroidal saponins with a hormone modulating effect are found in *Chamaelirium luteum*, *Dioscorea villosa*, *Trillium erectum*, *Trigonella foenum-graecum* and *Aletris farinosa*. Must be hydrolysed into the aglycone or sapogenin by bowel flora before they are biologically active. Binding capacity is very weak therefore limited competitive inhibition with endogenous oestrogens. May act through modulation of hypothalamic - pituitary - ovarian axis and potentiation of ovarian hormones. *Tribulus terrestris* (Puncture vine) has high levels of *disogenin* that increases FSH and oestradiol in pre-menopausal women and reduces hot flashes in post-menoapusal women without increasing oestradiol, indicating an effect in the hypothalamus.

DIFFERENTIAL ANALYSIS OF SOME FEMALE CONDITIONS

MENSTRUAL DISTURBANCE

Dysfunctional:

Ovarian or pituitary hormone imbalance

Secondary:

Fibroids
Cervical polyps
Endometriosis
PID
IUD
Thyroid dysfunction
Uterine, cervical or ovarian CA.

Inter-menstrual bleeding:

Ovulation (usually with mittelschmerz)
Cervical erosion, polyps or Ca.
Uterine polyps or Ca.

Menorrhagia:

Pelvic congestion
Capillary fragility
PID
Hormonal imbalances
Hypertension
Endometriosis
Diabetes or kidney disease

ACUTE LOWER ABDOMINAL PAIN

Non pregnant:

Fibroids
Salpingitis
Ovarian tumours - torsion, rupture
Endometriosis
Appendicitis

Early pregnancy:

Abortion
Degeneration of fibroid
Impaction of retroverted uterus
Ectopic pregnancy
Corpus luteum haemorrhage
Stretching of round ligaments
Appendicitis

Late pregnancy:

Labour
Placental abruption
Ruptured uterus
Rectus muscle haematoma
Appendicitis

CHRONIC LOWER ABDOMINAL PAINS

Endometriosis
Uterine tumours including fibroids
Uterine displacements
Chronic salpingitis
Ovarian tumours
Peritoneal adhesions

BACKACHE

Dysmenorrhoea
Malposition of uterus
Pelvic infection
Endometriosis
Pelvic tumours
Pregnancy or after effects.

VAGINAL DISCHARGE

White, non-infected:

Pregnancy
Cervical erosion or polyp
Fibroid
Endometrial polyp
IUD
General ill health

Purulent:

Cervical erosion
Cervicitis
Cervical polyp
Trichomoniasis
Neglected pessary or tampon
Atrophic vaginitis
Endometritis

Cottage cheese:

Candida albicans

Watery:

Uterine Ca
Urine via fistula
Leaking amniotic fluid

Blood stained:

Cervical erosion, polyp, Ca
Endometritis/vaginitis
Incomplete abortion
Retained foreign body.

PAINFUL INTERCOURSE (Dyspareunia)

Lower genital tract:

Anatomic abnormality
Rigid hymen
Local infection/inflammation
Post operative scarring
Post - episiotomy
Post radiotherapy
Vaginismus (spasm)

Upper genital tract:

PID
Endometriosis
Cervicitis
Retroverted uterus
Ovarian cyst
Fibroid
CA of cervix or uterus