DYSMENORRHOEA

ESSAY

Submitted in Partial Fulfillment of
Master Degree in
Obstetrics and Gynaecology

By
Mohamad Emam Rashed
M.B.,B.Ch., Ain Shams University

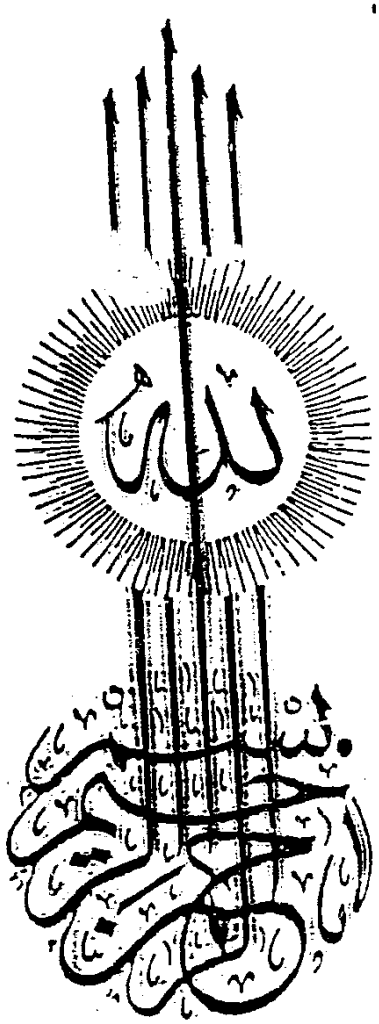
Under Supervision of

Prof. Dr. Ali Farid Mohamad
Professor of Obstetrics and Gynaecology
Faculty of medicine
Ain Shams University

Dr. Essam Mohamad Khater
Assistant Professor of Obstetrics and Gynaecology
Faculty of Medicine
Ain Shams University

Faculty of Medicine
Ain Shams University
1993

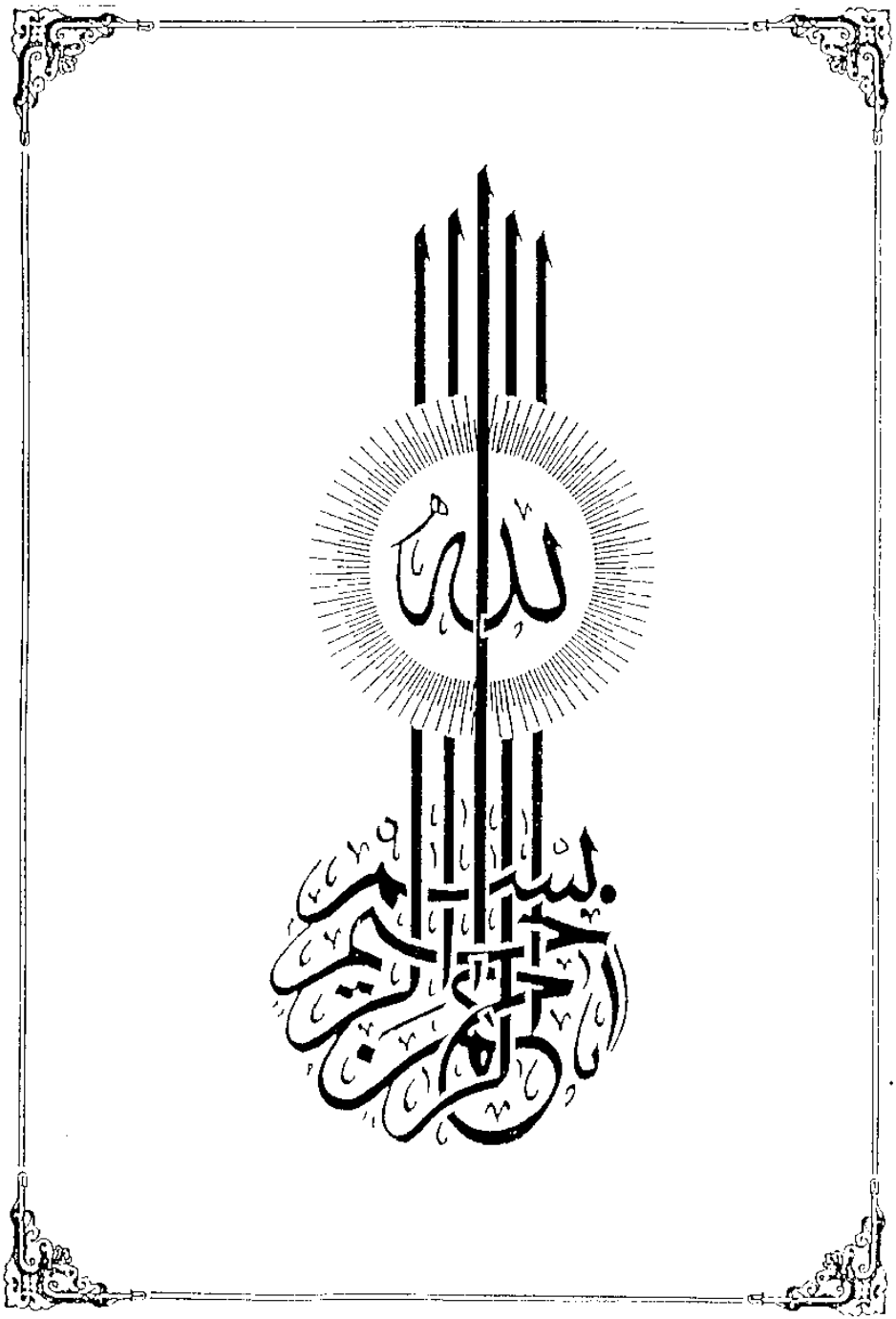
Central Library - Ain Shams University



علم الإنسان ما لم يعلم

صدق الله العظيم

Central Library - Ain Shams University



Central Library - Ain Shams University

ACKNOWLEDGEMENT

I would like to express my sincere gratitude to Prof. Dr. **ALI FARID MOHAMAD**, *Professor of Obstetrics and Gynaecology, Faculty of Medicine, Ain Shams University, for his whole hearted support, close supervision and experienced advice.*

I am also deeply indebted to Dr. **ESSAM MOHAMAD KHATER**, *Assistant Professor of Obstetrics and Gynaecology, Faculty of Medicine, Ain Shams University, for his continuous encouragement and unforgettable effort.*

..ɴɴɴɴ..

Central Library - Ain Shams University

INDEX

Central Library - Ain Shams University

DYSMENORRHOEA

INTRODUCTION :

Dysmenorrhoea comes from the Greek word meaning difficulty monthly flow but it is now usually taken to mean painful menstruation (Marry Ann, 1985).

Dysmenorrhoea is a well knwon gynecologic problem since old ages. It was mentioned in the writings of Ibn-Sina. He considered dysmenorrhoea as a problem in his book Al-Cannon .

Definition :

Dysmenorrhoea is painful menstruation (Marry Ann, 1985). It can be classified into primary or secondary dysmenorrhoea.

In primary dysmenorrhoea there are painful menstrual cramps but no visible pelvic pathology that causes the painful menstrual cramps.

Secondary dysmenorrhoea occuring as the result of disease or pain which has its onset after regular menstruation been established for some years and usually pelvic examination reveals the presence of pathology,

Central Library - Ain Shams University

e.g. uterine abnormality, chronic pelvic infection, endometriosis, uterine fibroids, cervical stenosis or adenomyosis.

Primary dysmenorrhoea is a disorder of young women occuring more frequently during the teenage years and early twenties (Dawood, 1990).

Secondary dysmenorrhoea affects an older age group and causes pain with different characteristics (Moos, 1968 and Chan, 1972).

Pain in primary dysmenorrhoea is characterized by crampy lower abdominal pain, worst at the onset and often radiating to back and legs, pelvic pain, nausea and vomiting, and other variable disturbances occuring in association with menses (Norman et al., 1952).

Pain in secondary dysmenorrhoea taken form of premenstrual pain situated on either the back or the lower abdomen 3-5 days before the onset of menstruation and it is always relieved by the menstrual flow (Tindal, 1987).

Central Library - Ain Shams University

AETIOLOGY AND PATHOGENESIS OF PRIMARY DYSMENORRHOEA

Several theories regarding the aetiology of spasmodic dysmenorrhoea were postulated over the years (Dawood, 1983) :

1. Cervical obstruction :

It is the earlist and most persistant theory to explain the cramp like pain particularly if the uterus is acutely anteflexed. In fact, cervical obstruction is not present but the pain may be due to inability of the uterus to extrude a large endometrial fragment or a large clot of blood. Such mechanical obstruction might stimulate vigorous uterine contraction causing pain (Sloan, 1972).

In 1982, Budkle claimed that the effect of cervical stenosis is not relevant in multiparous woman as a cause of primary dysmenorrhoea although it is possible for it to be aquired as a result of operation on the cervix such as cone biopsy or amputation of the cervix in repair operation.

In 1984, Dawood concluded that there is no evidence suggesting the role of cervical stenosis in primary

Central Library - Ain Shams University

dysmenorrhoea.

2. Myometrial contractility :

The role of uterine hyperactivity in the causation of dysmenorrhoea was first suggested by Novac and Reynolds in 1932.

Dysmenorrhoea may be due to irregular dysrhythmic contraction or simply elevated uterine tone (Filler and Hall, 1970).

Also, Akerlund et al. (1976) showed that in primary dysmenorrhoea there is one or more abnormality in uterine activity. So, there is increase either in resting tone, in active pressure, in number of uterine contractions or uncoordinated or dysrhythmic uterine activity. When more than one of these abnormalities occur simultaneously, they potentiate each others by the appearance of abnormal uterine activity. There is reduction of the uterine blood flow leading to ischaemia which could be one of the mechanisms of pain. With ischaemia, there is a failure to remove waste product and accumulation of metabolities, mainly lactic acid and H^+ ions. The presence of waste product or failure to maintain electrolyte imbalance is probably the cause of pain and necrosis due to ischaemia.

Central Library - Ain Shams University

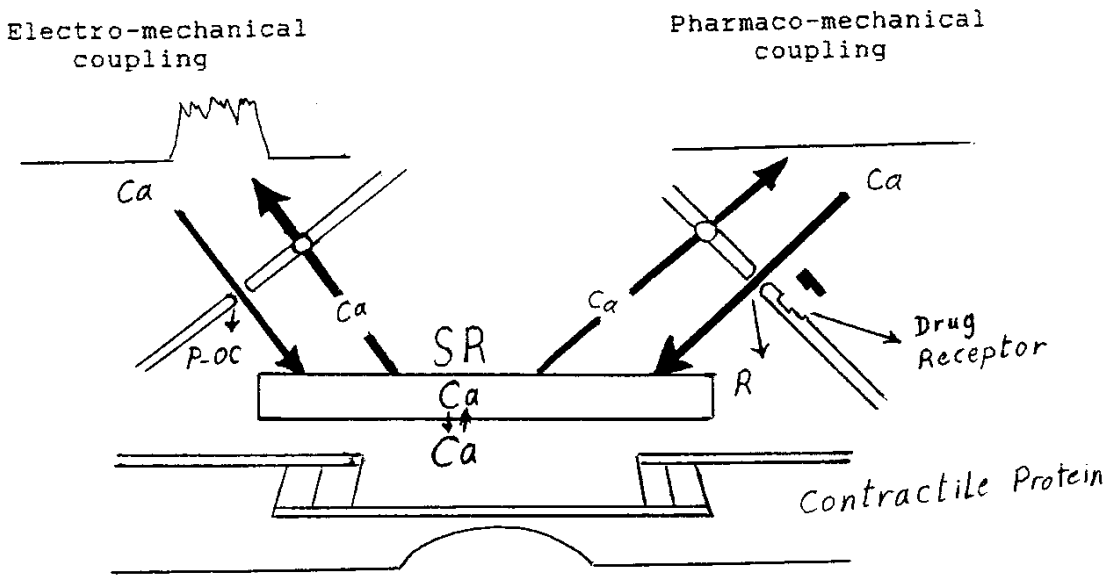
Mechanical activation of the myometrium is probably initiated by action potentials generated or evoked by exogenous factors such as hormones or drugs. The action potentials seems to be activated mainly by influx of calcium ions. It is believed that the flow of calcium into myometrium occurs through specific channels in its membrane. One of these channels has been called the "potential operated channel" (P-OC). The other type of membrane channels is called the "Receptor operated channel" (R-OC), but is less effective or specific for calcium ions than the P-OC. The possible influence on the mechanical activity of the uterus through (P-OC) channel with electrical activity (electro-mechanical coupling) or without the electrical activity through (R-OC) (Pharmaco-mechanical coupling) is shown in Fig. (1) (Forman and Erick, 1983).

3. Blood flow :

Akerlund demonstrated that during exacerbations of the pain, blood flow was decreased and was accompanied by pain (Akerlund et al., 1976).

Marry Ann (1985), by using highly sensitive methods for measuring uterine contractility, it is possible to see uterine artery pulsations. In subjects with dysmenorrhoea, the pulsation disappears whereas it remains through the peak in control subjects.

Central Library - Ain Shams University



SR = Saccoplasmic reticulum.

Fig. (1) : Mechanism of uterine contraction.

(Quoted from Forman and Erick, 1983)

Central Library - Ain Shams University

4. Prostaglandins and primary dysmenorrhoea :

The idea that prostaglandins may be involved in dysmenorrhoea was first suggested by Pickles during the 1960 (Pickles et al., 1965). He extracted a smooth muscle stimulant from menstrual fluid which was identified as a mixture of prostaglandins.

Lumsden et al. (1983) in their study of 28 women aged less than 25 years with regular cycles, sixteen of the women had dysmenorrhoea while the other 12 women had painless menstruation. They collected the menstrual fluid in the first and second day. They found that the amount of PGs in menstrual fluid of dysmenorrhoic women were higher in the first day than in the second and it was high in dysmenorrhoic than in the symptomless women. So, they concluded that severe dysmenorrhoea is associated with increase concentration of PGF2 and this increase is more pronounced in the first than in second day of the menstrual period.

Prostaglandine E2 and F2α are present in human menstrual fluid and in endometrial curettings obtained during the proliferative and secretory phase of menstrual cycle, prostaglandine-like activity in peripheral blood obtained during menstruation has also been reported (Pickles, 1967).

Central Library - Ain Shams University

Attempts have been made to correlate the levels of prostaglandins in the endometerium and in peripheral circulation with the onset of menstruation.

Downie, Poyser and Wunderlick (1974) showed that endometrial PGE2 and PGF2α increase progressively in the luteal phase of the cylce. The ratio of PGF2α : PGE2 increased from 0.6 in the proliferative phase to 1.5-1.7 around the time of ovulation. The raised amounts of prostaglandins in the presence of low levels of progesterone immediate in the premenstrual period may account for the greater degree of uterine activity that time.

Lundstzem, Wiqvist and Green (1976) have shown that the concentration of PGF2α metabolites in plasma from dysmenorrhoic was higher than in plasma from normal women.

Dawood, (1990) said that several lines of evidence strongly point to increase uterine prostaglandin production and release as a cause of primary dysmenorrhoea. The clinical symptoms of primary dysmenorrhoea are strikingly similar to the side effects observed when prostaglandins are administered either from induction of labour or from abortion. These include uterine contractions, nausea, vomiting and diarrhea.

Central Library - Ain Shams University

Direct evidence based on measurement of prostaglandins inthe endometrium, jet washing of the uterus and menstrual fluid have shown that their levels of prostaglandins are significantly elevated over those of non dysmenorrhoic women.

Finally, many clinical trials haveshown that the non steroidal anti-inflammatory drugs that are also prostaglandin synthetase inhibitors will alleviate the symptoms with several of the non steroidal anti-inflammatory drugs. The clinical relief is achieved through a reduction in menstrual fluid prostaglandins secondary to the drug action.

5. Steroid hormones :

Normally, in the preovulatory phase under the influence of oestrogen the uterine contraction increases in frequency until it reaches a peak at the time of ovulation. In the post ovulatory phase of the cycle when progesterone is present, the contraction is less frequent and more regular (Bengstsson and Theobald, 1966).

In 1971, Roberts et al., stated that as it is known that oestrogen leads to contraction while progesterone causes relaxation of the uterus, therefore, the imbalance between the two hormones might lead to primary

Central Library - Ain Shams University

dysmenorrhoea. Also, Roberts et al. (1971) stated that excessive decrease in progesterone level will lead to dysmenorrhoea as happens normally at the end of the luteal phase if pregnancy does not take place, corpus luteum will regress and so progesterone level will drop resulting in labilization of lysosomes in the endometrial cells. This will lead to release of lysosomal enzyme, phospholipase A2 which will hydrolyze the phospholipid present in the cell membrane to generate arachidonic acid, the main precursor of prostaglandins. Prostaglandins will lead to contraction of the smooth muscle of the uterus causing pain. If the amount of prostaglandin is excessive pain would be exagerated.

The control of contractions is dependent on the rate of prostaglandin synthesis and, in particular, on the rate limiting enzyme phospholipase A2 which controls the formation of prostaglandin from arachidonic acid. Oestrogens have been shown to labilize and progesto — gens to stabilize the intracellular lysosomes which produce phospholipase A2 increase in the oestrogen/ progesterone ratio or withdrawal of progesterone may in this way stimulate or enhace pro staglandin synthesis and uterine contractions (Dewhurst, 1981).

Central Library - Ain Shams University