MINISTRY OF HEALTH

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COGNITIVE FUNCTIONS IN THE POST-HYSTERECTOMY AND OVARIECTOMY ASPECTS: TREATMENT DYNAMICS

(Monograph)

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Scientific Research Results Presented in the Monograph

This monograph explores the clinical features of neurological disorders and their impact on cognitive functions in women with anxiety-depressive syndrome (ADS) following hysterectomy and oophorectomy. Within the scope of the study, not only the clinical-neurological manifestations but also the cognitive and emotional disturbances, along with their clinical and differential-diagnostic aspects in patients with ADS, were analyzed. The authors substantiate the necessity of applying multicomponent therapeutic approaches in such cases, emphasizing the relevance of treatment strategies aimed at preventing cognitive and emotional impairments, as well as reducing insomnia and fatigue.

The scientific findings obtained provide a solid foundation for the development of new therapeutic approaches in the treatment of this condition.

This monograph is intended for neurologists, neuropsychologists, family physicians, as well as students, master’s degree seekers, and clinical residents of medical universities.

ISBN:

LIST OF ABBREVIATIONS

- **BP** – Blood Pressure
- **ANS** – Autonomic Nervous System
- **VDS** – Vegetative Dystonia Syndrome
- **HE** – Hysterectomy
- **H-OE** – Hysterectomy and Oophorectomy
- **DE** – Dyscirculatory Encephalopathy
- **HRT** – Hormone Replacement Therapy
- **PA** – Personal Anxiety
- **OE** – Oophorectomy
- **PHOS** – Post-Hysterectomy and Oophorectomy Syndrome
- **RA** – Reactive Anxiety
- **CVS** – Cardiovascular System
- **ADS** – Anxiety-Depressive Syndrome
- **CCI** – Cerebral Circulatory Insufficiency
- **CNS** – Central Nervous System
- **RR** – Respiratory Rate
- **HR** – Heart Rate
- **EEG** – Electroencephalogram

CONTENTS

ABBREVIATIONS LIST……………………………………………………………………… 5
INTRODUCTION……………………………………………………………………………7

I. HISTORY OF NEUROLOGICAL DISORDERS IN WOMEN AFTER HYSTERECTOMY AND OOPHORECTOMY……………………………………………10

II. MODERN CONCEPTS ON THE MECHANISMS OF DEVELOPMENT OF ANXIETY-DEPRESSIVE SYNDROME IN WOMEN AFTER HYSTERECTOMY AND OOPHORECTOMY…………………………………………………………………..18

III. CLINICAL-NEUROLOGICAL AND NEUROPSYCHOLOGICAL ASPECTS OBSERVED IN THE PROBLEM……………………………………………………………36

IV. ANALYSIS OF VEGETATIVE, COGNITIVE, AND NEUROPSYCHOLOGICAL CHANGES IN PATIENTS: ISSUES OF TREATMENT STRATEGY AND PREVENTION……………………………………………………………………………..54

CONCLUSION…………………………………………………………………………….86
SUMMARY………………………………………………………………………………….93
PRACTICAL RECOMMENDATIONS…………………………………………………….94
LIST OF REFERENCES………………………………………………………………….95
APPENDIX 1……………………………………………………………………………..103

INTRODUCTION

Women's health preservation is one of the priority directions of modern medicine, as it deserves particular attention as the foundation of socio-economic stability and the health of future generations. In particular, reproductive health represents a crucial criterion reflecting the balanced interaction of biological, psychological, and social factors in the female body, and it occupies a central position in contemporary gynecology. The growing incidence of pathologies affecting the reproductive system, along with the application of radical treatment methods (surgical interventions), further underscores the urgency of this issue. In recent years, the frequency of radical surgical interventions on the pelvic organs—specifically hysterectomy (removal of the uterus) and oophorectomy (removal of the ovaries)—has been rapidly increasing. These operations not only terminate a woman's reproductive function but also negatively affect other organs and systems, including the cardiovascular, central nervous, endocrine, and autonomic nervous systems. In the postoperative period, a considerable number of women develop a complex of functional disorders known as post-hysterectomy and post-castration syndromes.

According to a number of studies, these syndromes affect between 27% and 70% of women (Nazarova N.M. et al., 2022; Timofeeva L.I., 2023; Magamedova F.D., 2021; Challberg et al., 2016). They are characterized by a complex of neuropsychiatric, autonomic-vascular, and metabolic-endocrine disturbances arising as a consequence of hysterectomy and oophorectomy. In particular, in women of active reproductive age, removal of these organs disrupts homeostasis, provokes profound neuroendocrine imbalance, and delivers a progressive and systemic blow to women's health (Biryukova et al., 2021).

These disturbances, in turn, lead to insufficiency of compensatory mechanisms within the autonomic nervous system. This is manifested in pathological processes in the cardiovascular system—especially cerebral circulation—along with anxiety-depressive syndromes and cognitive impairments (Shuster et al., 2008). Symptoms such as depression, anxiety, sleep disorders, memory decline, and reduced motivation directly affect women's personal, social, and professional lives.

To date, the clinical features of anxiety-depressive disorders and cognitive impairments associated with hysterectomy and oophorectomy, their close relationship with autonomic nervous system dysfunction, and the underlying pathophysiological mechanisms have not been sufficiently systematized in the scientific literature. This gap contributes to diagnostic errors, delayed identification, and inadequate treatment in clinical practice (Semenova L.A., 2018; Sadykova A.R., 2020; Platonova T.M., 2023; Kotsopoulos J. et al., 2015; Chen I. et al., 2023; Ndumele C., Michos E.D., 2024).

Furthermore, complications arising during this period, such as osteoporosis, arterial hypertension, arteriosclerosis, metabolic syndrome, and other conditions, significantly worsen women's health, increase disability, and even elevate the risk of premature mortality (Maichuk E.I. et al., 2014; Pivina L.V. et al., 2020; Biryukova I.A. et al., 2021; Schwartz et al., 2023; Jiang et al., 2020; Dørum et al., 2016; Challberg et al., 2016; Etchegary et al., 2015). Neurological and psychological syndromes arising after these surgical interventions are often underrecognized or misattributed to other disorders. Therefore, early detection, assessment, and prevention of these conditions in women represent an urgent and mandatory task of modern medicine.

This monograph addresses these pressing issues by analyzing the clinical course of post-hysterectomy and post-castration syndromes, dysfunctions of the autonomic nervous system, interactions with the cardiovascular system, as well as the mechanisms underlying psycho-emotional and cognitive impairments. Moreover, the potential for developing diagnostic and therapeutic strategies based on individualized approaches is considered, with a particular focus on the effectiveness of psychological rehabilitation, hormone replacement therapy, and other complex treatment methods.

Additionally, through this research, new clinical recommendations are proposed by clarifying the interplay of neuropsychic, autonomic, and endocrine factors associated with hysterectomy and oophorectomy, with the aim of improving approaches directed at enhancing women's quality of life.

I. HISTORY OF NEUROLOGICAL DISORDERS IN WOMEN AFTER HYSTERECTOMY AND OOPHORECTOMY

At present, a number of pathological conditions that arise after surgical interventions on various organs have been identified. Neurological disturbances manifest as postoperative complications, developing as functional impairments due to partial or complete organ removal (A.Z. Mandelshtam, S.N. Davidova, 2021; L.V. Kiloson et al., 2016). Neurological problems following hysterectomy and oophorectomy primarily originate from the autonomic nervous system, which is considered their main underlying cause. Such disturbances develop in different reproductive organs and require timely diagnosis and treatment.

An analysis of the available literature demonstrates that systemic disorders originating from the autonomic nervous system are frequently observed. These disturbances transmit signals to the hypothalamic–pituitary region and may influence neurosecretory processes in higher centers of the central nervous system. This phenomenon has been sufficiently studied in the works of S.N. Davidova and colleagues. In addition, impairments in circulation, lymphatic drainage, and neural pathways have been identified in such cases, with evidence that these complications often persist after surgical interventions. Partial or complete removal of the reproductive organs through surgery disrupts the feedback mechanisms within the endocrine system, leading to alterations in hypothalamic and pituitary function (R.A. Hatcher et al., 2004).

Therefore, the disturbances observed are based on several factors, which can be described as follows:

1. During surgical removal of the uterus, fallopian tubes, or ovaries, anatomical connections (circulatory, lymphatic, and neural systems) are altered, thereby revealing the interdependence of organs.
2. Removal of the uterus, fallopian tubes, and ovaries negatively impacts endocrine function and, through disrupted feedback mechanisms, impairs higher activities of both the autonomic and central nervous systems.

3. Partial or complete removal of these organs adversely affects the function of the hypothalamic–pituitary system.

Hysterectomy in obstetric practice is associated with serious complications and often requires urgent surgical intervention. It is one of the most commonly performed procedures among women of reproductive age and ranks as one of the most frequent operations in the United States (A.M. Wolker, 2023; R.E. Deiker, 2017, et al.). Hysterectomy is frequently recommended for pregnancy termination and sterilization purposes.

Advantages of abortive hysterectomy. Abortive hysterectomy is performed to eliminate menstrual dysfunction, guarantee sterilization, and prevent the risk of future uterine pathology. In 1976–1977, the University of Chicago conducted abortive hysterectomies in 50 women of reproductive age. This procedure simultaneously aimed to terminate pregnancy and halt menstrual and reproductive functions. However, this issue remains controversial and requires further research. Radical operations lead to disturbances in homeostasis and profound neuroendocrine disorders, which in most cases necessitate therapeutic interventions. Following uterine removal, a number of systemic disturbances associated with hysterectomy syndrome arise, particularly autonomic–vascular disorders. These disturbances develop in response to changes within the hypothalamic–pituitary–ovarian–uterine system.

Hysterectomy syndrome encompasses a range of disturbances, including consequences associated with the loss of menstrual and reproductive functions. The close relationship between the uterus and other reproductive organs confirms the role of this system in maintaining overall homeostasis.

Several authors (R.A. Elebesova, 2019; N.I. Yegorova, 2020) have described degenerative–atrophic changes in the uterus and ovaries. Experimental studies in animals (S.A. Antipin, 2021) have confirmed these findings. At the same time, other researchers have obtained different results. For example, in 1986 A.Z. Sowza performed ovarian biopsies prior to hysterectomy and again one year later. Repeat biopsies of the uterus during laparoscopy revealed no signs of atrophy. A number of authors (I.S. Kogan-Malienko, 2016; S.R. Davidov, 2013) have classified the uterus among the internal secretion glands, noting that the luteolytic hormone prostaglandin is produced there. The cessation of uterine endocrine function has been suggested as one of the causes of developing autonomic–neurotic disorders. However, it has also been demonstrated that significant amounts of prostaglandins are produced in parts of the uterus and in the ovaries (T.Ya. Pshenichnikova, 2015). The hypothesis of the uterus as an endocrine organ therefore requires further long-term studies and clinical observations.

N.D. Fanchenko and G.A. Anashkin (2013) opposed the concept of the uterus as an internal secretion organ and did not recognize it as an independent endocrine gland. Clinical observations of women with congenital uterine aplasia demonstrated that ovarian activity continues in a normal cyclical rhythm, and even in the absence of the uterus, the metabolism of ovarian steroid hormones by the liver does not differ from normal. Receptors in the cervix and their connections with the autonomic nervous system, spinal cord, and key brain nuclei have been identified. For example, hormonal reflexes of genital origin may arise through hypothalamic influence on the cervix. Therefore, removal of the uterus, by eliminating interactions with cervical receptors, may negatively affect central nervous system function. Some neurohormonal disorders observed after hysterectomy may be explained by this pathogenic mechanism.

Post-castration syndrome (PCS) is a symptom complex of neuro-psychic, autonomic–vascular, and metabolic–endocrine disturbances that develops after removal of the uterus and ovaries. PCS occurs frequently in women of reproductive age following hysterectomy with oophorectomy. At present, there is no unified understanding regarding the severity, clinical characteristics, and dependence of PCS on the extent of surgery. Furthermore, clear diagnostic criteria and objective measures to evaluate treatment effectiveness are lacking. Specialized studies have demonstrated that PCS occurs more often and in a more severe form in women under the age of 45, while after the age of 45 the risk of developing PCS increases threefold.

The pathogenesis of PCS is explained by a decline in estrogen levels, which, following removal of the ovaries, leads to increased secretion of gonadotropic hormones. This condition is associated with hypoestrogenemia reactions in the endocrine glands. Hormonal studies show that in operated patients (regardless of the presence of PCS), follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels are elevated, while prolactin levels are reduced. The increase in LH and FSH secretion is attributed to the absence of inhibin production by the ovaries and disruption of the feedback mechanism between estrogens and the pituitary gland. Other data suggest that the rise in FSH and LH levels after castration is associated with increased activity of the anterior pituitary (S. Nicozia, 2017). Elevated concentrations of gonadotropins in the blood are thus identified as a consequence of ovarian removal.

However, according to several authors, there is no clear correlation between hormone levels and the severity of PCS. In studies conducted by Y. Barrett-Connor (2020), no relationship was found between FSH and LH levels and PCS severity. Similarly, M. Alper et al. (2015) reported that estrogen levels are not directly related to PCS severity.

Adaptation of the female body after oophorectomy is ensured through integrated restructuring of the hypothalamic–pituitary–adrenal axis. In patients with post-castration syndrome (PCS), cortisol levels increase while testosterone levels decrease. Adrenocorticotropic function of the pituitary and its secondary markers may contribute to hyperactivation of this major regulatory system. Episodes of “hot flushes” associated with PCS may result from dual adrenal mechanisms, central and peripheral neurotransmitter activity, and cortical processes of the brain, particularly those linked to fear responses and higher brain centers.

According to several hypotheses, hot flushes arise due to an imbalance in neurotransmitter levels, including norepinephrine, histamine, β-endorphins, dopamine, and others. There is no direct relationship between hot flushes and estrogen levels; moreover, the decline in estrogen occurs well before castration. Research indicates that elevations in tropic hormones are associated with changes in luteinizing hormone (LH), thyroid-stimulating hormone (TSH), and adrenocorticotropic hormone (ACTH). Hormone surges during hot flush episodes are thought to result from central regulatory disturbances involving modulatory influences of gonadotropin-releasing hormone (GnRH, luliberin), corticotropin-releasing hormone (CRH, corticoliberin), and thyrotropin-releasing hormone (TRH, thyroliberin). These neurohormones directly affect thermoregulatory centers.

At present, there is no consensus regarding the state of the hypothalamic–pituitary–thyroid axis in PCS. Some authors have emphasized that estrogens activate thyroid hormones, and that thyroid hormones in turn influence steroid metabolism. Ueno N. et al. (2010) demonstrated an association between PCS severity and increased TSH levels. In some studies, elevated TSH was explained by altered catecholamine synthesis and metabolism, with a link to estrogen deficiency. L.V. Kiloson et al. (2016) reported correlations between PCS severity and catecholamine levels in different hypothalamic regions. Other researchers have identified relationships between hot flushes and plasma adrenaline and noradrenaline levels. Such episodes are associated with altered catecholamine synthesis, regulated indirectly through estrogen deficiency. Thyroid hormones enhance tissue sensitivity to catecholamines by altering the number and responsiveness of α- and β-adrenoreceptors.

Recent investigations indicate that PCS significantly affects cardiovascular function by increasing the risk of ischemic heart disease, arterial hypertension, and atherosclerosis—diseases that remain the leading causes of morbidity and mortality in highly developed countries. Currently, the association between oophorectomy and cardiovascular risk is well established. The increased prevalence of atherosclerosis-related ischemic heart disease has been directly linked to cessation of ovarian function. Transient arterial hypertension is among the common clinical manifestations of PCS. According to G.V. Krasnopolsky (2013), the severity of PCS correlates with transient hypertension. Hypertensive disease (chronic high blood pressure) is largely associated with structural and functional disturbances in central brain centers, particularly the hypothalamus. This condition is accompanied by heightened activity of the sympathetic nervous system and adrenal glands.

In labile hypertension, characterized by fluctuating blood pressure levels, adrenal system activity is markedly increased, driven largely by stress hormones, especially adrenaline. In this state, adrenal and mediator systems are activated, enhancing synthesis and metabolism of catecholamines such as adrenaline and norepinephrine. All these processes play a crucial role in the pathogenesis of hypertension. In addition, mediators such as serotonin and histamine are key regulators of vascular tone, thereby ensuring cardiovascular stability.

Post-castration syndrome (PCS) leads to dysfunction of the hypothalamic–pituitary–ovarian axis, with consequent disruption of neurohormonal regulation following ovarian removal. Electroencephalographic (EEG) findings vary depending on PCS severity. In mild forms of PCS…

In patients with mild PCS, EEG recordings show a slight decrease in the amplitude of the α-rhythm and the appearance of θ-waves. In patients with moderate to severe PCS, EEG demonstrates a more pronounced reduction of α-rhythm and an increase in β-waves, indicating disturbances in brain activity and functional impairment of the central nervous system. These changes reflect both the severity of the syndrome and the brain's response to these processes.

Furthermore, following castration, i.e., surgical removal of the ovaries, gonadotropin levels and EEG parameters may undergo restoration. These changes are regarded as compensatory adaptation processes in the absence of ovarian hormonal activity. Administration of estrogens after oophorectomy helps maintain the feedback mechanisms of neurohormonal regulation. This, in turn, is a crucial factor for postoperative adaptive responses and the restoration of homeostasis.

Castration syndrome develops primarily as a result of ovarian endocrine dysfunction, leading to functional disturbances across all levels of the neurohormonal system. This condition activates protective–adaptive mechanisms at both central and peripheral levels, aimed at adjusting the organism to postoperative stress and restoring homeostasis. At the same time, these mechanisms not only serve protective functions but also play a vital role in adaptation to stress and stabilization of the organism's condition in the postoperative period.

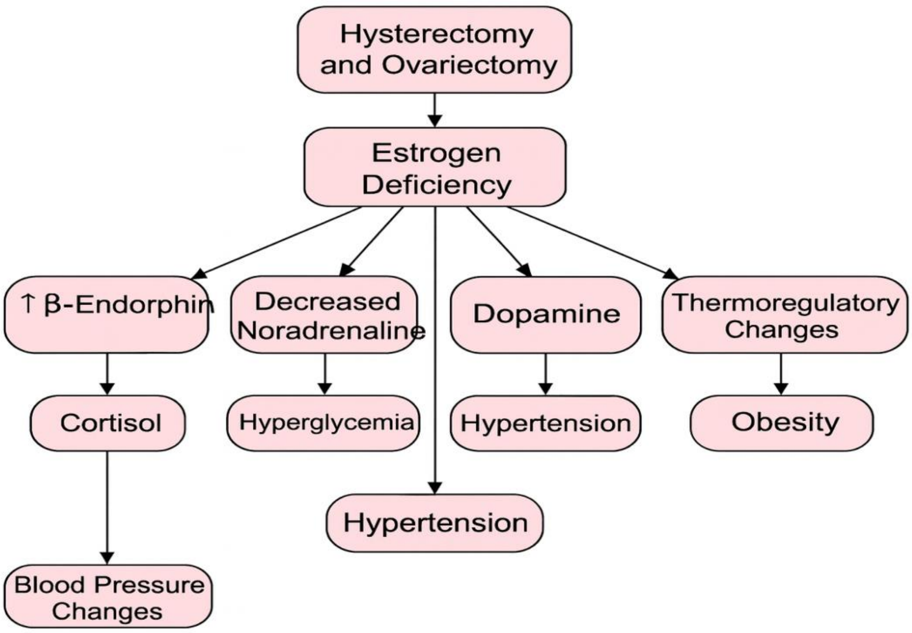
II. CONTEMPORARY PERSPECTIVES ON THE MECHANISMS OF DEVELOPMENT OF ANXIETY–DEPRESSIVE SYNDROME IN WOMEN AFTER HYSTERECTOMY AND OOPHORECTOMY

Ovarian resection results in hypo- and agonadism. This condition develops against the background of the functional "shutdown" of ovarian activity, which in turn is accompanied by alterations in hypothalamic and limbic system function. These disturbances are manifested through changes in hormone synthesis, secretion, and neurotransmitter activity.

The neuroendocrine mechanism involves decreased dopaminergic activity in the hypothalamus and limbic structures, alongside increased noradrenergic tone.

These mechanisms underlie the syndrome that develops after oophorectomy. Clinically, dysfunction of the hypothalamus manifests as hot flushes, hypertension, and obesity. Emotional disturbances, anxiety, depression, and cognitive impairments reflect neuroendocrine dysfunction characteristic of the postmenopausal state.

Taking these observed changes into account, we structured them in the form of a table and analyzed the pathological processes based on this tabular representation.



Figure№ 2.1. Neuroendocrine dysfunction of the limbic system in the post-hysterectomy and post-ovariectomy state.

- In this figure, the condition of the female body after surgical removal of major reproductive organs, such as hysterectomy and oophorectomy, is illustrated. These procedures lead to profound hormonal and neuroendocrine changes, most notably associated with estrogen deficiency, which may result in various physiological and psychological problems. This section provides a comprehensive analysis of the key aspects of such dysfunctions, their impact on the organism, and the related pathological conditions.

- **Estrogen deficiency and its effects.** Following hysterectomy and oophorectomy, estrogen levels decline significantly. This deficiency directly induces a series of hormonal and neuroendocrine alterations:
- **Reduced β-endorphin activity.** Estrogen modulates β-endorphin secretion, and its decline lowers pain tolerance and negatively influences emotional state.
- **Alterations in norepinephrine and dopamine activity.** Reduced estrogen disrupts the regulation of these neurotransmitters, which play essential roles in blood pressure control and mood regulation, thereby contributing to neurological and psychological disturbances.
- **Thermoregulatory changes and their consequences.** Estrogen deficiency also adversely affects thermoregulation in the body, leading to several clinical manifestations:
- **Hyperhidrosis.** Impaired thermoregulation may cause excessive sweating, frequently observed during menopause and in post-surgical states.
- **Hypertension.** By influencing norepinephrine and dopamine levels, reduced estrogen may elevate blood pressure, which poses a serious risk for the development of cardiovascular diseases.
- **Obesity.** Alterations in metabolic control mechanisms may occur under estrogen deficiency, resulting in increased body weight due to hypertrophy and reduced energy expenditure.
- **Additional hormonal and physiological changes.** Estrogen deficiency impacts not only thermoregulation but also the functioning of other hormones and contributes to the progression of various associated disorders.
  - **Cortisol.** As the primary stress hormone, fluctuations in cortisol levels influence stress responses, as well as blood pressure regulation and glucose metabolism.

- **Serotonin.** Estrogen is closely linked to serotonin, which plays a vital role in mental health. A deficiency in serotonin may lead to psychological disturbances such as depression and sadness.
- **Insulin.** A reduction in estrogen affects insulin sensitivity, thereby increasing the risk of developing diabetes mellitus.

Pathological conditions associated with estrogen deficiency Several disorders may arise as a result of estrogen deficiency:

- **Depression.** Estrogen has a direct impact on mood regulation, and its decline intensifies depression, stress, and overall psychological instability.
- **Osteoporosis.** Estrogen is essential for maintaining bone strength. Its deficiency weakens bones and significantly increases the risk of osteoporosis.

Conclusion. Neuroendocrine dysfunctions caused by estrogen deficiency following hysterectomy and oophorectomy exert profound effects on the body. These hormonal changes disrupt blood pressure regulation, metabolism, mental health, and thermoregulation, ultimately contributing to the development of various diseases. Early detection and management of these dysfunctions are of critical importance for protecting women’s health.

Table №2.1.

Primary and secondary effects of neuroendocrine dysfunction: hormonal and physiological changes

№	Category of effect	Effect	Outcome
1	Primary	Estrogen deficiency	Affects mood, cognitive function, and physiological processes
2	Primary	Altered thermoregulation	Leads to excessive sweating and affects blood pressure
3	Primary	Metabolic disturbance	Reduced energy expenditure and weight gain

4	Primary	Vascular dysfunction	Vascular impairment and increased cardiac workload
5	Secondary	Hypertension	Elevated blood pressure
6	Secondary	Obesity	Excessive weight accumulation
7	Secondary	Loss of bone density	Osteoporosis
8	Secondary	Cardiovascular complications	Development of heart diseases

Primary changes resulting from neuroendocrine dysfunction are direct effects on the body that adversely impact essential hormonal and physiological processes.

1. **Estrogen deficiency.** Following hysterectomy and oophorectomy, the decline in estrogen is the main cause of neuroendocrine dysfunction. Reduced estrogen levels lead to various cognitive and emotional disturbances. Mood changes, depression, and anxiety are directly linked to estrogen deficiency. Moreover, diminished cognitive performance and memory impairment are also consequences of this condition.
2. **Thermoregulatory processes.** Estrogen deficiency alters the regulation of heat exchange in the body. This results in excessive sweating (hyperhidrosis) and fluctuations in blood pressure. Such manifestations are especially common in women during menopause.
3. **Metabolic disturbances.** A decline in estrogen negatively affects metabolic activity. Energy expenditure decreases, while fat accumulation increases. This contributes to obesity and predisposes to the development of various endocrine disorders.
4. **Vascular dysfunction.** Estrogen plays a crucial role in maintaining vascular integrity. Its deficiency leads to vascular damage, weakening of vessel walls, and increased cardiac workload.

Secondary changes are conditions not directly caused by estrogen deficiency but rather arising as consequences of the primary alterations.

1. **Hypertension.** Elevated blood pressure develops due to vascular dysfunction associated with estrogen deficiency. Hypertension poses a major risk to the cardiovascular system, as it places excessive strain on the heart.
2. **Obesity.** Metabolic disturbances contribute to excess weight gain. This not only affects physical appearance but also negatively impacts cardiovascular health. Obesity further increases the risk of diabetes mellitus and other endocrine disorders.
3. **Loss of bone density.** Estrogen is essential for maintaining bone strength. Its decline leads to osteoporosis, characterized by weakened bones and increased risk of fractures. This condition is particularly common among postmenopausal women.
4. **Cardiovascular complications.** Estrogen deficiency contributes to vascular weakening and the development of hypertension. As a result, the risk of heart disease increases, particularly when hypertension is present, further overloading the heart.

As demonstrated, primary changes resulting from neuroendocrine dysfunction affect multiple systems of the body and trigger secondary diseases and complications. Timely recognition and treatment of these processes are critically important for preserving overall health.

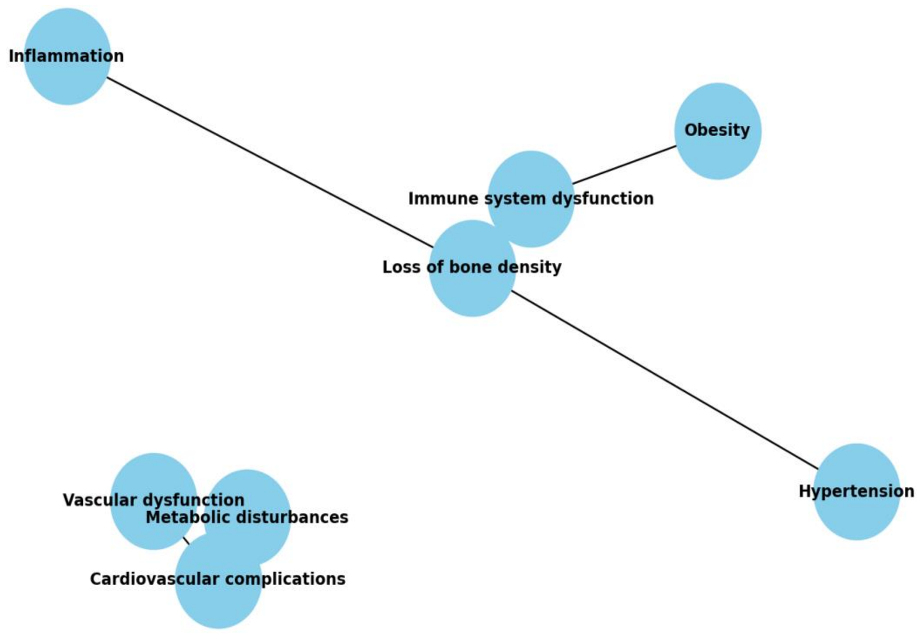


Figure 2.2. Types of various physiological and metabolic changes observed in neuroendocrine dysfunction

Neuroendocrine dysfunction, particularly estrogen deficiency, leads to a range of physiological and metabolic alterations in the human body**.** These changes significantly affect multiple systems, including the cardiovascular system, metabolism, the immune system, and bone tissue. Disorders arising from estrogen deficiency often weaken overall health and increase the risk of developing various diseases. The following section provides a detailed analysis of these processes.

Metabolic effects. Estrogen plays a central role in regulating metabolic processes. This hormone influences lipid breakdown, carbohydrate metabolism, and overall energy expenditure. Estrogen deficiency leads to a marked reduction in energy expenditure, resulting in fat accumulation. Excessive fat storage contributes to obesity, which in turn predisposes individuals to metabolic syndrome, insulin resistance, and type 2 diabetes mellitus. Metabolic disturbances related to estrogen deficiency also increase the risk of cardiovascular diseases, including atherosclerosis and hypertension. Moreover, excess fat deposition may lead to hepatic steatosis, further disrupting metabolic processes and impairing the body's overall energy balance.

Cardiovascular effects. Estrogen has profound effects on the cardiovascular system, helping to maintain vascular tone, regulate blood pressure, and provide antioxidant protection. Estrogen deficiency reduces the elasticity of vascular walls, thereby contributing to the development of hypertension. Elevated blood pressure places the cardiovascular system at risk, increasing the likelihood of myocardial infarction and stroke. In addition, estrogen deficiency accelerates the progression of atherosclerosis, since estrogen regulates lipid metabolism and helps maintain normal cholesterol levels. Consequently, decreased estrogen levels heighten the risk of myocardial infarction, ischemic heart disease, and cerebrovascular disorders.

Skeletal effects. Estrogen deficiency directly affects bone tissue, reducing bone mineral density and leading to the development of osteoporosis. Estrogen plays a key role in maintaining calcium and other minerals in the bone matrix, and its deficiency accelerates bone loss. This process is particularly pronounced in postmenopausal women, making osteoporosis a common complication in this population.

Osteoporosis and bone fragility. Osteoporosis leads to bone fragility, which significantly increases the risk of fractures. Fractures, particularly those occurring in the femur or vertebrae, have a profound impact on quality of life and severely restrict patient mobility. Osteoporosis that develops as a result of estrogen deficiency often results in serious injuries, making treatment more complex and challenging.

The immune system and estrogen deficiency. The immune system is the body’s defense mechanism, operating on two primary levels: humoral immunity and cell-mediated immunity. Humoral immunity acts through antibodies circulating in the blood and body fluids, while cell-mediated immunity directly combats intracellular pathogens. Estrogen deficiency significantly affects both levels of immune responses.

Humoral immunity and estrogen deficiency. In humoral immunity, B lymphocytes play a key role by producing antibodies against pathogens. Estrogen exerts broad immunomodulatory effects, and its deficiency can impair B-cell function. This slows the humoral immune response, reduces antibody production, and weakens defense against pathogens, thereby increasing susceptibility to infections. Normally, estrogen stimulates B-lymphocyte activity to ensure rapid and effective immune function. In its absence, B-cell numbers decline and antibody production decreases, leaving the body vulnerable to viruses and bacteria. Furthermore, estrogen deficiency can contribute to the development of autoimmune diseases. In these conditions, the immune system mistakenly attacks the body's own cells, leading to disorders such as rheumatoid arthritis and multiple sclerosis.

Cell-mediated immunity and estrogen deficiency. In cell-mediated immunity, T lymphocytes and macrophages play critical roles. Estrogen deficiency diminishes the activity of these immune cells. Reduced T-cell function limits cytokine production, impairing cell-to-cell communication essential for initiating and coordinating immune responses. Likewise, estrogen deficiency weakens macrophage function, slowing the process of engulfing and destroying intracellular pathogens. This facilitates the persistence and replication of infections within cells, heightening disease risk. Estrogen deficiency also amplifies inflammatory processes, contributing to chronic diseases and dysregulated immune responses.

Estrogen and autoimmunity. When estrogen levels decline, the immune system may lose its regulatory balance, increasing the risk of autoimmune diseases. Estrogen normally maintains immune homeostasis, but its deficiency promotes misdirected immune responses, in which immune cells attack self-tissues. This creates a predisposition to autoimmune conditions, including rheumatoid arthritis, systemic lupus erythematosus, and multiple sclerosis. In addition to weakening immune defense, estrogen deficiency enhances chronic inflammation, which may contribute to a wide spectrum of diseases, such as cardiovascular disorders, gastrointestinal diseases, and pulmonary conditions.

Clinical implications of hysterectomy and oophorectomy. Hysterectomy (removal of the uterus) and oophorectomy (removal of the ovaries), performed for tumors, fibroids, endometriosis, or other gynecological conditions, often result in a sharp decline in estrogen and other hormone levels. This hormonal disruption has profound effects on the immune system and overall health.

Estrogen is not only a reproductive hormone but also a key immunomodulator that regulates both humoral and cell-mediated immunity. Its deficiency compromises immune function, enhances susceptibility to infections, promotes autoimmune diseases, and contributes to chronic inflammatory processes with systemic health consequences.

1.Humoral immunity. Estrogen deficiency slows down the humoral immune response, particularly the process of antibody production. Antibodies produced by B lymphocytes play a critical role in defending the body against pathogens. In women who have undergone hysterectomy and oophorectomy, reduced estrogen levels weaken this process, diminishing the body's protection against infections and viruses. Consequently, the immune system becomes less efficient at rapidly identifying and neutralizing pathogens.

2. Cell-mediated immunity. Estrogen deficiency also impairs the activity of T lymphocytes, which are the primary defense cells against intracellular bacteria and viruses. Reduced estrogen levels weaken T-cell function, thereby creating favorable conditions for the proliferation of viruses and bacteria within cells.

Autoimmune disease risk. Estrogen is essential for maintaining immune system balance. Its deficiency may cause immune dysregulation, leading the immune system to mistakenly attack the body's own cells. This significantly increases the risk of autoimmune diseases such as rheumatoid arthritis, multiple sclerosis, and systemic lupus erythematosus. Estrogen also regulates cytokine production, thereby controlling inflammation. Estrogen deficiency enhances inflammatory processes, increasing susceptibility to chronic diseases and infections. Women who undergo hysterectomy or oophorectomy experience a sharp decline in estrogen, resulting in immune system imbalance. For this reason, hormone replacement therapy (HRT) is often recommended to restore estrogen levels, strengthen immunity, and reduce disease risk. However, HRT is not suitable for all patients, as it may carry risks of its own.

5. Vascular dysfunction

Estrogen is vital for maintaining vascular health and elasticity. It protects blood vessel walls and supports their structural integrity. Estrogen deficiency weakens vascular walls, disrupting vasodilation and vasoconstriction. This contributes to hypertension and circulatory problems. Reduced vascular elasticity negatively impacts the cardiovascular system and can impair blood supply to vital organs such as the heart and kidneys, potentially leading to cardiac and renal failure. Hysterectomy and oophorectomy, through the abrupt reduction of estrogen levels, substantially increase the risk of vascular abnormalities, since estrogen is one of the key protective hormones for blood vessels.

Estrogen and vascular function. Estrogen regulates vascular elasticity by modulating vasodilation and vasoconstriction, thereby helping to control blood pressure. Deficiency disrupts this balance, causing vascular stiffening, atherosclerosis, and hypertension. These changes elevate the risk of cardiovascular diseases, including myocardial infarction and stroke.

Blood clotting and thrombosis risk. In women who have undergone hysterectomy or oophorectomy, blood coagulation mechanisms may also be altered. Low estrogen levels increase the risk of thrombosis, particularly venous thrombosis and thromboembolism. This risk is especially pronounced in women who have had an oophorectomy, as ovarian removal completely halts estrogen production, thereby increasing blood clotting tendencies.

Inflammatory processes and vascular disease. Estrogen also plays an anti-inflammatory role by promoting the synthesis of anti-inflammatory mediators that protect vascular tissue from damage. Estrogen deficiency enhances vascular inflammation, leading to arterial stiffening and the development of inflammatory foci within vessel walls. These processes accelerate atherosclerosis and increase the risk of cardiovascular disease.

6. Inflammatory processes. Estrogen deficiency amplifies inflammatory activity. Reduced levels of this hormone impair anti-inflammatory responses, which in turn promotes chronic inflammation. This exacerbates the risk of long-term diseases, slows recovery after injury, and negatively affects cardiovascular and autoimmune conditions. Estrogen deficiency-driven inflammation particularly damages joints and cardiac tissue, leading to further systemic complications.

7. Hypertension. Estrogen deficiency is a direct cause of hypertension, as this hormone plays a central role in regulating vascular tone. Impaired control of vascular relaxation and contraction increases blood pressure, placing additional strain on the heart. The absence of estrogen reduces vascular elasticity, promotes vasoconstriction, and damages cardiovascular function. Hypertension significantly increases the risk of myocardial infarction and stroke. It also disrupts systemic circulation, limiting adequate blood supply to vital organs.

Estrogen and blood pressure regulation. Estrogen maintains vascular dilation and elasticity, supporting efficient circulation and blood pressure control. When estrogen levels decline, vascular walls stiffen and constrict, resulting in elevated blood pressure. Women undergoing hysterectomy and oophorectomy—particularly at a younger age—face an increased risk of developing hypertension due to the abrupt reduction in estrogen.

Post-surgical hypertension risk. Oophorectomy halts estrogen production entirely, which directly contributes to arterial constriction and raises the risk of hypertension. Research indicates that the risk of hypertension is strongly associated with postmenopausal declines in hormone levels, with the risk being especially high in women who have undergone oophorectomy.

Metabolic changes. The decline in estrogen affects not only the vascular system but also metabolic processes. This hormonal deficiency promotes fat accumulation, insulin resistance, and increased blood lipid levels. As a result, in addition to the development of hypertension, the risk of cardiovascular disease is also elevated.

Hormone replacement therapy (HRT). To reduce the risk of hypertension after surgery, hormone replacement therapy (HRT) is often prescribed. By restoring the estrogen deficit, HRT helps prevent hypertension and cardiovascular disease. However, HRT is not suitable for all women, as it may itself carry risks, such as thrombosis and myocardial infarction. Estrogen deficiency also disrupts metabolism and decreases energy expenditure, leading to excess fat deposition and obesity. Obesity not only negatively affects appearance but also places a heavy burden on multiple organ systems, particularly the cardiovascular system, thereby increasing the risk of heart disease. In addition, obesity is closely linked with insulin resistance, type

2 diabetes mellitus, hypertension, and other endocrine disorders, further compounding health risks.

Obesity and estrogen deficiency. The removal of the ovaries leads to a sharp decline in estrogen and other hormones, resulting in impaired lipid metabolism. Estrogen plays a key role in fat breakdown and distribution; its deficiency in women after hysterectomy or oophorectomy leads to increased visceral fat accumulation in the abdominal cavity and other areas. This condition significantly raises the risk of cardiovascular disease, diabetes mellitus, and other chronic disorders.

Estrogen deficiency also slows basal metabolic rate, reduces energy expenditure, and accelerates weight gain. It contributes to sarcopenia (loss of muscle mass), which further promotes obesity since muscle tissue is a primary consumer of energy. Fat accumulation in turn increases the production of pro-inflammatory cytokines, promoting metabolic syndrome and chronic disease. Thus, women undergoing hysterectomy or oophorectomy face a significantly higher risk of obesity due to hormonal imbalance and disrupted metabolism.

Preventive strategies, such as HRT or lifestyle modification, are crucial to reduce the risk of obesity and its complications.

Physiological changes. Estrogen deficiency is closely linked to profound physiological changes that disrupt essential body systems, contributing to serious illnesses. These pathological changes often create a cascade of disorders requiring long-term treatment. Early detection and management strategies are critical to maintaining women’s health. The mechanisms of hypothalamic and limbic system dysfunction after hysterectomy and oophorectomy are central to these changes and explain their clinical manifestations.

Depression and psychological disorders. Research shows that the post-surgical period is frequently associated with anxiety-depressive syndrome (ADS), which in women often correlates with arterial hypertension, cerebrovascular insufficiency, and cognitive decline. These effects are largely explained by dysfunction of dopaminergic and noradrenergic neurotransmitter systems, leading to disturbed neuropsychological balance in the central nervous system.

Depression (from Latin *depressio* — despondency) is a psychiatric disorder characterized by prolonged emotional imbalance that significantly reduces quality of life. It often develops as a psychological response to trauma or negative life events (e.g., bereavement, job loss). According to the World Health Organization (WHO), more than 110 million people worldwide suffer from depression, and the prevalence continues to rise. Between 1992 and 2020, about 5% of the global population was affected; since 2021, this number has reached 5.7%. Depression typically begins with mild symptoms such as sleep disturbance, irritability, and reduced interest in daily activities. When these symptoms persist for more than two weeks, they indicate a developing disorder. Without treatment, depression can progress to social withdrawal, suicidal ideation, and a marked reduction in quality of life.

Women after oophorectomy are particularly vulnerable to depressive states, including anxiety-depressive syndrome. Common symptoms include tearfulness, irritability, fatigue, and cognitive impairments such as memory loss and decreased concentration. These emotional disturbances negatively affect work performance and contribute to family and social conflicts.

Psychological assessment studies reveal an increased tendency toward anxiety-hypochondriac syndrome and affective reactivity in such patients. Dennerstein (2017) emphasized that depressive symptoms associated with menopausal syndrome are strongly linked to the sharp decline in estrogen levels. This hormonal imbalance disrupts emotional regulation in the central nervous system and worsens mood disorders.

III. CLINICAL-NEUROLOGICAL AND NEUROPSYCHOLOGICAL ASPECTS OBSERVED IN THE PROBLEM

Neurological and psychiatric changes after hysterectomy and oophorectomy

Hysterectomy (removal of the uterus) and oophorectomy (removal of the ovaries) are surgical procedures often followed by widespread clinical-neurological manifestations in women. These changes are primarily associated with a sharp decline in hormone levels, particularly estrogen and progesterone, which play an essential role in regulating both central and peripheral nervous system activity. The decrease in these hormones after ovarian removal may result in neurological disorders and a variety of psychiatric symptoms.

1. Neurological and psychiatric alterations. Women who undergo oophorectomy or hysterectomy frequently experience depression, anxiety, and cognitive decline. Estrogen deficiency is the primary trigger for these disturbances, as it is closely linked to central neurotransmitter systems, especially serotonin and dopamine. These neurotransmitters are vital for mood regulation, concentration, and motivation.

- **Memory and cognition.** Ovarian removal may cause impaired memory, reduced attention span, and difficulty in learning new information. Studies indicate that decreased estrogen lowers synaptic connectivity in neurons, adversely affecting memory and cognitive performance.
- **Emotional instability.** Hormonal decline leads to depression, anxiety, fatigue, and irritability. Reduced estrogen also decreases serotonin activity, thereby disturbing emotional stability and increasing mood fluctuations.

2. Autonomic dysfunction

Estrogen deficiency also impacts the peripheral nervous system by disturbing autonomic balance. Clinical manifestations include:

- **Sleep disorders.** Women often report insomnia, frequent awakenings, or disrupted sleep cycles. This is attributed to reduced production of melatonin and serotonin, both of which regulate sleep.

- **Hot flashes and night sweats.** Disruption of thermoregulatory processes due to estrogen deficiency commonly causes vasomotor symptoms similar to those observed during menopause.

3. Inflammation and nerve damage. Another important consequence of estrogen deficiency is enhanced inflammatory activity. Estrogen normally acts as an antioxidant, protecting neurons against oxidative stress. Its reduction weakens this defense, increasing vulnerability to neuronal and axonal injury. This may elevate the long-term risk of neurodegenerative disorders such as Alzheimer's and Parkinson's disease.

4. Osteoporosis and peripheral neuropathy. Estrogen is critical for maintaining bone mineral density. Its deficiency promotes osteoporosis, which not only weakens the skeleton but can also contribute to nerve fiber damage, potentially leading to peripheral neuropathy and chronic pain syndromes.

5. Cognitive function and Alzheimer's disease risk. Estrogen exerts neuroprotective effects in the brain and is essential for maintaining synaptic connections. Its deficiency contributes to β-amyloid accumulation, which increases the risk of Alzheimer's disease. Research shows that women who undergo oophorectomy face a higher risk of cognitive decline and dementia.

Overall, the decline in hormone levels following hysterectomy and oophorectomy leads to widespread clinical-neurological disturbances, including psychiatric symptoms, autonomic dysfunction, sleep disorders, hot flashes, cognitive decline, and skeletal fragility. While hormone replacement therapy (HRT) may alleviate some of these symptoms, it is not suitable for all patients. Therefore, women who undergo these procedures require regular neurological and psychiatric evaluation.

Cognitive function and disorders. Cognitive dysfunction is one of the most common neurological manifestations observed in post-surgical women. Since cognition represents the brain's integrative activity, its impairment often arises from diffuse or localized brain injury.

- **Epidemiology.** Cognitive disorders are especially frequent in older adults: 3–20% of individuals over 65 years suffer from dementia or significant cognitive impairment, and prevalence increases with age, reaching up to 80% in the elderly. The growing proportion of older populations makes cognitive dysfunction a pressing challenge for neurologists and healthcare providers.

Theoretical foundations of cognitive functions. Cognitive functions are the highest-level activities of the human brain, enabling rational acquisition of knowledge. They allow individuals to perceive, process, store, and apply information, underpinning thought, memory, speech, motor activity, and intellectual capacity. Stable cognitive function ensures quality of life and social adaptation, while cognitive impairment negatively affects daily functioning.

1. Memory. Memory is the central element of cognition, encompassing the ability to receive, store, and retrieve information. It is closely associated with hippocampal structures. Memory disorders (amnesia) significantly impair learning, work, and social interactions.

- **Retrograde amnesia:** loss of past memories.
- **Anterograde amnesia:** inability to form new memories.
- **Global amnesia:** combination of both deficits.

These can occur in Alzheimer's disease, traumatic brain injury, or other neurological conditions.

2. Gnosis. Gnosis refers to the ability to process sensory input and transform it into coherent perceptions. It includes:

- **Visual gnosis.** Recognition of objects by sight. Impairment leads to **visual agnosia**, often associated with occipital lobe damage.
- **Audiovisual gnosis.** Recognition of sounds and their sources. Impairment results in **auditory agnosia**, typically caused by temporal lobe lesions.
- **Tactile gnosis.** Recognition of objects by touch. Impairment results in **tactile agnosia**, associated with parietal lobe damage.
- **Prosopagnosia.** Inability to recognize familiar faces, associated with temporal or occipital lobe damage.
- **Astroagnosia.** Impairment of spatial relationships and distance perception, often linked to parietal lobe lesions.

Gnostic disorders (agnosia) profoundly impair daily functioning. Early recognition and diagnosis are essential for maintaining quality of life.

3. Speech. Speech is the primary means of expressing thoughts and establishing communication. It relies on coordinated activity of frontal and parietal cortical structures. Speech disorders manifest as **aphasia**, which may involve impaired comprehension, impaired production, or both. Aphasia frequently arises from stroke, brain trauma, or other neurological conditions.

Praxis. Praxis refers to learned motor skills, including the ability to acquire, retain, and apply purposeful movements. Normal praxis allows for the automation of complex actions such as writing, drawing, or performing daily tasks. Disturbances in praxis are known as **apraxia**, typically associated with damage to the frontal or parietal lobes. Apraxia manifests as difficulty in planning or executing movements correctly and is frequently observed in neurological disorders, especially in the post-stroke period.

Types of apraxia include:

- **Ideomotor apraxia.** Characterized by impaired ability to program and perform purposeful movements. The patient can mentally conceptualize the action but fails to execute it correctly. *Clinical presentation:* difficulty in gestures such as waving, saluting, or grasping objects. Often results from frontal or parietal lobe lesions.
- **Ideational apraxia.** Defined by disruption in sequencing and coordinating a chain of actions. Patients fail to perform complex tasks correctly, such as dressing or using tools. *Clinical presentation:* using objects incorrectly (e.g., attempting to write with a pen as if it were another tool). Typically linked to parietal lobe damage.
- **Constructive apraxia.** Involves visuospatial dysfunction, impairing the ability to reproduce shapes or understand spatial relations. *Clinical presentation:* difficulty with tasks involving drawing, building, or assembling objects. Usually associated with posterior parietal lesions.

- **Orofacial apraxia.** Inability to perform coordinated voluntary movements of the face, mouth, tongue, or pharynx. *Clinical presentation:* impaired speech articulation, difficulty producing facial expressions, and problems with feeding. Often co-occurs with aphasia.
- **Dynamic apraxia.** Characterized by impaired execution of rapid, complex, sequential movements. *Clinical presentation:* difficulties maintaining timing and continuity of motor actions.

Intellect. Intellect encompasses the capacity to analyze, compare, identify similarities and differences, evaluate, and draw conclusions. These higher-order intellectual abilities reflect the integrative function of the brain and are essential for complex intellectual activity. Intellectual impairment is most often associated with Alzheimer's disease and other dementias. Such impairments interfere with the ability to perform complex daily tasks and significantly reduce quality of life.

Neuropsychological tests are used to evaluate cognitive functions, including memory, image recognition, problem-solving, and praxis. While comprehensive neuropsychological assessments are not always feasible in outpatient practice, screening tools are widely employed. One such instrument is the **Abbreviated Mental Test Score (AMTS)**, a rapid screening tool for cognitive impairment that evaluates memory, attention, arithmetic, and language in 10–15 minutes.

Cognitive impairment syndromes. Cognitive impairment may result from focal brain lesions affecting one or several domains of cognition. Common causes include stroke, traumatic brain injury, and brain tumors. In cases of diffuse or multifocal brain damage, impairments are widespread, affecting all cognitive domains, often through multiple pathogenic mechanisms.

Dementia represents the most severe form of cognitive dysfunction, diagnosed when memory impairment is accompanied by deficits in praxis, gnosis, speech, or intellect, significantly interfering with daily life. A clear state of consciousness and evidence of organic brain pathology are required for diagnosis.

- **Alzheimer's disease (AD).** The leading cause of dementia, associated with progressive loss of cholinergic neurons, typically after age 65. Early signs include forgetfulness of recent events, followed by spatial disorientation, language impairment, and progressive loss of independence. Motor and sensory systems are usually preserved until later stages.

- **Vascular dementia.** The second most common cause, linked to recurrent strokes or chronic cerebral ischemia. Compared with AD, memory impairment is less pronounced, while intellectual slowing and attentional deficits dominate. *Clinical signs:* pseudobulbar syndrome, hypokinesia, spasticity, and dysarthria. Neuroimaging reveals cerebral infarctions and diffuse white matter changes (leukoaraiosis).
- **Mixed dementia.** A combination of neurodegenerative and vascular pathology, frequently observed in elderly patients. Clinical and morphological studies indicate mixed dementia is more prevalent than "pure" forms of AD or vascular dementia.

Other causes of dementia include traumatic brain injury, brain tumors, hydrocephalus, neuroinfections, and metabolic disorders, though these are less frequent.

Mild Cognitive Impairment (MCI). MCI is defined as cognitive decline greater than expected for age, but not severe enough to meet dementia criteria. Patients often present with memory and attentional deficits that are noticeable to themselves and their relatives. Epidemiological studies show that 10–15% of older adults are affected, and 55–70% of these cases progress to dementia within five years. The most frequent causes are neurodegenerative processes, cerebrovascular insufficiency, or their combination. In 1994, the **World Psychogeriatric Association** proposed the term **aging-associated cognitive decline (AACD)** to describe age-related cognitive changes. However, distinguishing AACD from early degenerative or vascular disease remains challenging.

Recent evidence highlights the connection between depression and cognitive impairment. Cognitive deficits are increasingly recognized as part of the clinical presentation in depressed patients, underlining the complex interplay between affective and cognitive disorders.

Sigma receptors and their role in depression and cognitive disorders. Sigma receptors play an important role in the development of depression and cognitive dysfunction in the brain. Sigma-1 receptors, first discovered in 1976 and cloned in 1996, are proteins located in the endoplasmic reticulum. They modulate glutamatergic neurotransmission by regulating NMDA (N-methyl-D-aspartate) receptors.

Binding sites for sigma receptors are widely distributed in key brain regions, including the hypothalamus, substantia nigra, red nucleus, and other critical structures. Sigma-1 receptors are of particular importance in the pathophysiology of neuropsychiatric disorders. They interact with multiple neurotransmitter systems, including dopamine, serotonin, norepinephrine, and acetylcholine. The highest concentrations of sigma-1 receptors are found in the hippocampus, amygdala, and frontal cortex. Research on the role of sigma-1 receptors in cognitive functions demonstrates that their agonists exert beneficial effects on cognitive deficits. Furthermore, antidepressants that act as sigma-1 receptor agonists may be effective in the treatment of cognitive impairment, depression, and anxiety disorders.

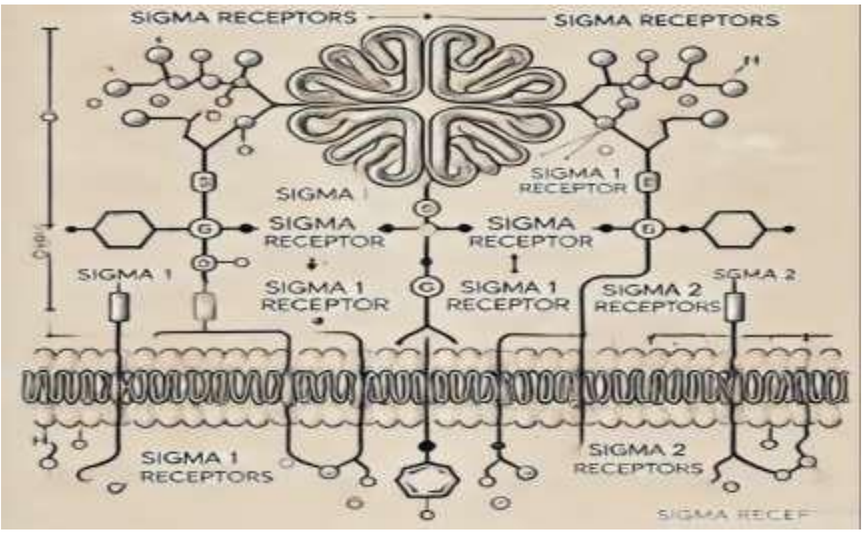


Figure 3.1. Localization and structure of sigma-1 and sigma-2 receptors in the cell membrane.

Examination of Patients with Cognitive Impairment. Complaints of memory decline or reduced intellectual performance constitute the basis for conducting a neuropsychological evaluation. Such complaints may be reported by the patient or by close relatives. Reports from relatives are generally considered more reliable, since the patient’s own assessment of cognitive function is often not objective.

In routine clinical practice, neuropsychological assessment can be performed using simple screening scales, such as the **Mini-Mental State Examination (MMSE)**. Extending the protocol is often impractical, as results are strongly influenced by the patient’s age and educational level; this may reduce specificity without necessarily improving sensitivity. Furthermore, in about half of patients with subjective memory complaints, basic screening scales fail to confirm objective cognitive impairment.

When subjective complaints of memory decline are not objectively verified, the most common cause is usually emotional disturbances such as heightened anxiety or depressed mood. Therefore, careful evaluation of emotional state is essential in patients presenting with memory complaints. Among young and middle-aged individuals, subjective memory decline is frequently associated with an increased risk of depression. In such cases, the absence of objective evidence of cognitive impairment often reflects the limited sensitivity of screening tools. For this reason, in addition to assessment and treatment of emotional disorders, regular follow-up and repeat psychological testing at 3–6 month intervals are recommended.

If objective cognitive impairment is confirmed, the underlying cause should be investigated, since cognitive disturbances are not always due to primary brain pathology. Systemic disorders or metabolic abnormalities may also contribute, such as hypothyroidism, hepatic or renal disease, or deficiencies of vitamin B12 or folic acid.

Selecting the appropriate therapeutic approach is crucial. Many medications—particularly those with psychotropic effects—can adversely affect memory and other cognitive functions. The greatest negative impact is seen with anticholinergics, antidepressants, neuroleptics, and benzodiazepines. Therefore, such drugs should be used cautiously in elderly patients. Likewise, uncontrolled alcohol consumption should be avoided.

It is important to assess not only the patient's somatic status but also their emotional state. Emotional disturbances, especially anxiety-depressive syndromes, may underlie subjective cognitive impairment. Severe depression, in particular, can lead to objective cognitive deficits and mimic dementia—a condition known as pseudodementia.

In elderly patients where depression is suspected, antidepressant therapy may be considered. However, agents with minimal anticholinergic activity—such as selective serotonin reuptake inhibitors (SSRIs)—are preferred.

IV. ANALYSIS OF VEGETATIVE, COGNITIVE, AND NEUROPSYCHOLOGICAL CHANGES IN PATIENTS: ISSUES OF TREATMENT STRATEGY AND PREVENTION

Treatment of cognitive impairment. The choice of therapeutic intervention is determined by the **severity** of cognitive impairment and its **etiology**. In cases of mild to moderate dementia associated with **Alzheimer's disease (AD)**, **cerebrovascular insufficiency**, or **mixed vascular-degenerative etiology**, the first-line pharmacological agents are **acetylcholinesterase inhibitors** (galantamine, rivastigmine, donepezil) or **memantine**.

These medications improve **memory and other cognitive functions**, help normalize behavior, and enhance adaptation in daily life. As a result, they contribute to improving the **quality of life** for both patients and their caregivers. Some evidence suggests these agents may slow the progression of cognitive decline, although this requires further investigation.

In mild and moderate cognitive impairment, however, the efficacy of acetylcholinesterase inhibitors and memantine has not yet been conclusively demonstrated. When global cognitive deficits have little effect on daily functioning, the primary therapeutic goal is not memory improvement, but rather the **prevention of further deterioration**. In such cases, **neuroprotective agents** are considered first-line therapy.

Vasotropic (Cerebrovascular) agents. Cerebrovascular agents can be divided into three major pharmacological groups:

- **Phosphodiesterase inhibitors**: eufillin, pentoxifylline, vinpocetine, ginkgo biloba, etc.
    - Their mechanism of action is based on increasing intracellular cyclic adenosine monophosphate (cAMP) levels in vascular smooth muscle cells, leading to vasodilation.
    - These agents predominantly improve **microcirculation** and do not produce a "steal effect".
- **Calcium channel blockers**: cinnarizine, flunarizine, nimodipine.

    - Their vasodilatory effect is due to inhibition of calcium influx into cells.
    - Some data suggest that calcium channel blockers may preferentially act on the **vertebrobasilar circulation**.
- **Alpha-2 adrenoreceptor blockers**: nicergoline.
    - These agents reduce the vasoconstrictive effects of adrenaline and noradrenaline, producing vasodilation.

Cerebrovascular insufficiency involves **ischemic and hypoxic processes** that trigger structural vascular changes and neurodegenerative mechanisms. Therefore, vasotropic agents are used not only in ischemia but also in other neurodegenerative conditions, including Alzheimer’s disease. Standardized **ginkgo biloba extract** is an example of such neuroprotective therapy.

Neuroprotective and metabolic agents. GABAergic drugs, **peptidergic agents**, **amino acids**, and certain metabolites optimize metabolic processes, enhance **neuronal plasticity**, and protect neuronal survival.

- These agents are employed in the **prevention of neurodegeneration**.

Antidepressant therapy. The concept of depression treatment has changed dramatically over the past 100 years. Early in the 20th century, Kraepelin described depression as a long-lasting and recurrent disorder.

More recently, it has become clear that discontinuation of antidepressants, particularly **SSRIs (selective serotonin reuptake inhibitors)**, may cause **“discontinuation syndrome”**. Distinguishing between relapse of depression and discontinuation symptoms can be challenging in clinical practice.

When SSRI therapy is stopped, **mild symptoms** may appear within 1–10 days, and in some cases persist up to 24 days (e.g., fluvoxamine). Reported symptoms include:

- Vestibular disturbances (postural instability, dizziness, ataxia)
- Gastrointestinal dysfunction (nausea, vomiting)
- Flu-like symptoms (tension, myalgia, fatigue)
- Sensory disturbances (paresthesias, “electric shock” sensations)
- Sleep disorders (insomnia, vivid dreams)
- Psychiatric symptoms (anxiety, irritability, fear)

According to the **UK Medicines Control Agency**, paroxetine and venlafaxine are most frequently associated with discontinuation symptoms, while mirtazapine and fluvoxamine have a lower incidence. Paroxetine increases the risk of discontinuation symptoms up to **100 times more** than fluvoxamine.

- Clinical studies comparing fluvoxamine and paroxetine confirm these findings.
- To prevent withdrawal syndromes, **tapering over 4–6 weeks** is recommended when discontinuing paroxetine or venlafaxine.
- The discontinuation process must be **closely monitored** to avoid relapse.

The main therapeutic goal in depression treatment is **remission**, defined as a reduction of symptoms below a threshold score (≤7 on the **Hamilton Depression Rating Scale**). Remission is achieved in 50–65% of patients. Evidence shows that SSRIs are at least as effective as **tricyclic antidepressants (TCAs)**, with a more favorable side effect profile.

Clinical findings after hysterectomy (HE) and oophorectomy (OE)

Analysis of clinical-neurological indicators in 40 patients post-surgery revealed a distinct clinical syndrome characterized by:

- Blood pressure fluctuations
- Cardiac pain (angina-like symptoms)
- Dyspnea and hyperventilation syndrome
- Hyperhidrosis
- Cold extremities
- Headaches
- Insomnia
- Anxiety and panic-like vegetative crises

These symptoms are closely linked to **cerebral circulation disturbances** following hysterectomy and oophorectomy.

Anxious-Depressive Syndrome (ADS)

- Identified in **30 patients (75%)** after surgery.
- Manifested as a spectrum from **mild neurotic reactions** to **severe depressive episodes**.

- Clinical features included:
    - Irritability
    - Mood swings
    - Depression
    - Anxiety and fear
    - Loss of interest (anhedonia)
- Symptoms were assessed using the **Spielberger Anxiety Scale**.

Comparative analysis was performed between two patient groups:

- Those with anxious-depressive syndrome
- Those without ADS but with post-hysterectomy/oophorectomy syndrome

The results are presented in the following tables.

Table №4.1

Comparative characteristics of cerebral complaints after нysterectomy (HE) and oophorectomy (OE)

Complaints	**Patients with HSD (%)**	**Patients without HSD (%)**	**p-**
Headache	75	50	0,001
Dizziness	50	50	0,03
Memory impairment	80	10	0,05
Fatigue	85	40	0,05
Insomnia	80	50	0,05
Tinnitus	50	50	0,034
Irritability	90	0	0,05

Note: *Statistically significant differences between the groups: $p < 0,05$.*

Based on the results of the conducted analytical study, it is noteworthy that patients suffering from anxiety-depressive syndrome (ADS) exhibit a significantly higher frequency of various somatic and psychological symptoms compared to those without

ADS. This finding contributes to a more precise understanding of the clinical picture of the syndrome.

Specifically, headache was reported in 75% of patients with ADS, compared to 50% in the non-ADS group, indicating that this symptom is 1.5 times more common in the presence of ADS. Dizziness was observed in 50% of cases in both groups, suggesting that this symptom may not be directly associated with ADS.

Memory impairment was identified in 80% of patients with ADS, whereas only 10% of patients without ADS reported this symptom. This indicates that memory-related cognitive disturbances are 8 times more common in individuals with ADS, highlighting the neuropsychological components of the syndrome.

Fatigue was recorded in 85% of patients with ADS, compared to 40% in the control group, showing a 2.1-fold increase. This may negatively affect patients' overall work productivity and daily functioning.

Insomnia was reported by 80% of ADS patients and 50% of those without ADS, indicating a 1.6-fold higher incidence of sleep disturbances in the presence of the syndrome. Given its widespread occurrence, insomnia has a direct impact on patients' quality of life and psycho-emotional state.

Tinnitus was found in 50% of patients in both groups, suggesting no significant association with ADS. One of the most remarkable symptoms was irritability, observed in 90% of patients with ADS and completely absent in the non-ADS group. This stark contrast confirms that irritability is one of the key symptoms of ADS.

In conclusion, cognitive (e.g., memory impairment, fatigue) and emotional (e.g., irritability, insomnia) disturbances are significantly more prevalent in patients with ADS. The most characteristic symptoms in these individuals include headache, memory decline, fatigue, insomnia, and irritability. These findings confirm that ADS is a complex, multi-component pathological condition and underscore the necessity of a comprehensive, multidisciplinary approach to its management.

Table №4.2

Comparative analysis of complaints in patients with and without ADS

Complaints	Patients with ADS (n)	Patients without ADS (n)	Subjective Rating (ADS)	Subjective Rating (Non-ADS)	p-
Headache	30	20	8	6	<0,05
Dizziness	20	20	6	5	<0,05
Memory impairment	32	4	9	3	<0,05
Fatigue	34	16	9	4	<0,05
Insomnia	32	20	8	5	<0,05
Tinnitus	20	20	6	5	<0,05
Irritability	36	0	10	3	<0,05

Statistically significant differences between groups: p < 0,05.

The conducted study compared key symptoms and their subjective evaluations between patients with and without anxiety-depressive syndrome (ADS). The analysis revealed that not only were symptoms more prevalent among ADS patients, but they were also subjectively rated as more severe. For example, headache was reported by 30 patients (75%) in the ADS group, compared to only 20 patients (50%) in the non-ADS group. Memory impairment was documented in 32 cases (80%) among ADS patients, while only 4 patients (10%) in the non-ADS group reported the same symptom. This stark contrast indicates a significantly higher incidence of cognitive dysfunction in individuals with ADS. Subjective symptom ratings also differed significantly between the two groups. ADS patients rated their symptoms in the range of 8–10 on a 10-point scale, whereas non-ADS patients rated them between 3–6. This suggests that symptoms are experienced as more intense by ADS patients, highlighting a stronger negative impact on their psychological and physical well-being. For all primary complaints, the p-value was < 0.05, indicating that the differences between the groups are statistically significant. These differences are not random but are clinically meaningful and directly associated with the presence of ADS.

Conclusion. Patients with ADS not only experience a higher number of symptoms but also perceive them as more severe. This condition significantly impairs overall health, emotional well-being, and quality of life. The findings underscore the importance of a comprehensive psychological and medical approach in the management of ADS patients.

Table №4.3

Analysis of clinical complaints and the impact of cognitive impairments in patients with and without anxiety-depressive syndrome

Complaints	Patients with ADS (%)	Patients without ADS (%)	Age Range (years)	p-value
Headache	75%	50%	35–44	<0.01
Dizziness	50%	50%	45–54	<0.05
Memory impairment	80%	10%	35–44	<0.05
Fatigue	85%	40%	45–54	<0.001
Insomnia	80%	50%	35–44	<0.05
Tinnitus	50%	50%	45–54	<0.05
Irritability	90%	0%	35–44	<0.05

Statistically significant differences between groups: p < 0,05.

The results of the study demonstrate significant differences in clinical complaints between patients with anxiety-depressive syndrome (ADS) and those without ADS. These findings help to better understand the impact of ADS by highlighting the frequency and severity of various symptoms. One of the main complaints among patients was headache, which was more commonly observed in those with ADS. However, other clinical manifestations such as memory impairment, insomnia, and irritability were also significantly more pronounced in ADS patients.

Headache is one of the most common symptoms in patients with ADS, occurring in 75% of cases according to the study results. This is a notably high rate compared to only 50% in patients without ADS, confirming that ADS has a stronger and more frequent influence on the presence of headaches. The headache in ADS patients is associated with specific pathogenic processes, including changes in the nervous and vascular systems of the brain, as well as increased muscle tone. These factors contribute to the intensity of headaches in ADS and seriously affect the quality of life, as persistent and severe headaches reduce daily functioning and work productivity.

Dizziness was recorded equally in both groups (50%), suggesting that this symptom is less associated with ADS. It may be more related to general autonomic and vascular system dysfunctions and therefore could be considered an independent symptom rather than one directly linked to ADS. The study findings indicate that dizziness is not a characteristic symptom of ADS, implying the need to consider other pathological processes when evaluating this complaint.

Memory impairment was observed in 80% of patients with ADS, while only 10% of those without ADS reported it. This large difference indicates a strong impact of ADS on cognitive functions. Memory decline is directly related to neuroendocrine imbalance and stress. Chronic exposure to stress in ADS patients disrupts cognitive function. Such impairments may cause difficulties in performing daily tasks and negatively impact quality of life. Cognitive dysfunctions related to memory are among the most serious symptoms and require early detection and treatment.

Fatigue was reported in 85% of ADS patients compared to just 40% in the non-ADS group. This significant difference points to a reduction in energy levels and work performance among ADS patients. The onset of fatigue is often linked to both physiological and psychological processes, including chronic stress, increased muscle tone, and sleep disturbances. As a result of fatigue, patients experience notable difficulties in their daily work, which negatively affects their social and professional quality of life.

1. Among patients with ADS, insomnia was observed in 80% of cases, compared to 50% in non-ADS patients. Insomnia is often associated with stress and hyperactivity in the brain. Sleep disturbances in patients with ADS negatively affect their daily life, concentration, and overall quality of life. Chronic insomnia may contribute to worsening symptoms such as depression, fatigue, and cognitive impairments. This highlights the importance of improving sleep hygiene as part of the treatment process.

2. Tinnitus was reported at an equal rate (50%) in both groups, indicating that this symptom is equally prevalent regardless of ADS status. Tinnitus may be primarily linked to autonomic and vascular system dysfunction. Since this symptom is also common in patients without ADS, it is likely not specific to the syndrome and suggests the need to explore other possible causes.

3. Irritability showed the greatest difference between the groups: it was present in 90% of patients with ADS but was completely absent in those without. This symptom reflects significant emotional and psychological disturbances in ADS patients. Irritability may be associated with chronic stress and persistent headaches, and it seriously affects emotional stability. If it becomes chronic, it can increase the risk of depression and psychological deterioration, emphasizing the need for psychotherapeutic interventions in ADS management.

4. The study findings revealed that clinical and cognitive impairments are significantly more frequent and severe in patients with ADS. Each of these symptoms holds distinct clinical significance, as they severely impact patients' quality of life. The physiological and psychological changes identified in these patients are directly linked to reductions in daily functioning and work productivity. Based on the study results, the following areas require closer attention: In patients with anxiety-depressive syndrome (ADS), headache was one of the most prevalent symptoms, reported in 75% of cases, which is significantly higher than in non-ADS patients. This symptom is associated with specific physiological processes related to ADS, such as increased muscle tone and cerebrovascular dysfunction, leading to persistent and intense headaches that negatively impact daily life and work productivity. Effective management requires understanding the underlying mechanisms and applying timely therapy. Memory impairment, seen in 80% of ADS patients, indicates significant cognitive dysfunctions that affect daily tasks, work performance, and social interactions, often resulting from cognitive stress and brain function disruptions. Early treatment and psychotherapeutic approaches are crucial to mitigate these impairments. Fatigue, recorded in 85% of ADS patients, reflects decreased energy levels and difficulty performing daily tasks, while insomnia, observed in 80%, exacerbates stress and fatigue, contributing to depression, memory issues, and declining mental health. Proper sleep hygiene and daily routine optimization are essential in managing these symptoms. Irritability, the most differentiated symptom, was found in 90% of ADS patients but absent in non-ADS patients, indicating severe emotional and psychological instability potentially linked to chronic stress and headaches, highlighting the importance of psychotherapy and stress management. These findings show that ADS involves not only physiological disturbances but also significant cognitive and emotional disorders, requiring comprehensive treatment including headache management, prevention of cognitive and emotional decline, and addressing insomnia and fatigue. Physicians must assess each ADS patient individually and apply multi-dimensional therapeutic strategies. The study also emphasizes the need for future research into the pathogenesis of ADS-related cognitive and physiological impairments, as well as the development of new therapeutic strategies. Early detection and effective treatment of cognitive and emotional disturbances are essential to improve quality of life in ADS patients.

Table №4.4

Clinical and neurological examination findings in patients with and without anxiety-depressive syndrome (ADS)

№	Symptoms	Without ADS	With ADS
1	Oral automatisms	Absent	Absent
2	Tremor of fingers and eyelids	Absent	Present
3	Skin flushing	Present	Absent
4	Blood pressure instability	Present	Severe
5	Reduced attention and memory	Absent	Severe
6	Thermoregulation disorders	Marked	Marked
7	Excessive tearfulness	Marked	Marked
8	Hyperhidrosis	Absent	Severe

The table above illustrates how several clinical symptoms—such as oral automatisms and finger tremors—manifest differently across two patient groups. In the first group (without ADS), neither oral automatisms nor finger tremors were observed, while in the second group (with ADS), finger tremors were recorded. This difference may be linked to a higher degree of autonomic nervous system dysfunction. Skin flushing and blood pressure instability were observed in the first group, but were reported as **severe** in the second group. These clinical differences reflect varying levels of autonomic insufficiency and confirm that the second group experienced more intense dysfunction. In particular, symptoms like blood pressure instability and hyperhidrosis may indicate broader involvement of autonomic components of the illness. Attention and memory impairments were absent in the first group but very pronounced in the second group. This supports the idea that central nervous system function and cognitive capabilities are more severely affected in ADS patients. Additionally, thermoregulatory disturbances and heat episodes were clearly evident in the second group, further confirming autonomic dysfunction.

Increased salivation and plantar sweating were either absent or mild in the first group, while both symptoms were significantly more pronounced in the second group. This suggests that peripheral autonomic dysfunction was more intense in ADS patients.
In summary, clinical symptom analysis demonstrates that vegetative and cognitive disturbances were more prominently expressed in the second group. These differences may reflect varying severities of the disorder and hold important implications for treatment strategies. Signs such as excessive tremor and hyperhidrosis were also more frequently observed in patients with ADS.

Symptoms of autonomic insufficiency—such as tinnitus, blood pressure instability, and fatigue—played a central role in the clinical picture. Our findings on autonomic nervous system function indicate that in approximately 75% of cases, post-surgical arterio-circulatory disturbances are associated with autonomic nervous system dysfunction. Such conditions are typically identified following radical surgical procedures.

In this study, we analyzed two groups: patients with anxiety-depressive syndrome (ADS) and those without the syndrome (non-ADS). The results are summarized in Table 4.

Table №4.5.

Mean autonomic nervous system (ANS) score according to questionnaire and scheme data (m±m)

Indicator	Group I (M±m)	Group II (M±m)	p-value
ANS Questionnaire	54,1±0.215	32,9±0.232	0,001
ANS Schematic	48,6±0.152	23,9±0.155	0,02

Statistically significant differences between groups: $p < 0,05$.

The table presents the mean scores of the autonomic nervous system (ANS) based on validated questionnaire and schematic data (M±m). The findings clearly show that in both patient groups—those with and without anxiety-depressive syndrome (ADS)—ANS scores were significantly elevated compared to normative values. Moreover, the difference between the two groups was minor, indicating that even patients without ADS exhibited clear signs of vegetative dysfunction.

This observation is clinically important. It suggests that autonomic dysregulation is not exclusive to individuals with ADS but may also develop as a result of physiological and psychological stress induced by surgical menopause. Surgical removal of reproductive organs, such as the uterus and ovaries, is a major physical stressor for the female body. The autonomic nervous system often reacts with compensatory overactivation, leading to vegetative dystonia symptoms, even in the absence of underlying psychiatric pathology.

Such findings highlight the importance of using dynamic assessment methods (questionnaires and schematics) to evaluate ANS function postoperatively. These tools allow for better understanding of the systemic physiological disruptions that occur after gynecological surgeries, especially those involving small pelvic organs. Surgeries such as hysterectomy and oophorectomy disrupt the hypothalamic-pituitary-ovarian-uterine axis, leading to downstream dysfunction of hypothalamic and pituitary structures. This neuroendocrine imbalance manifests clinically through autonomic instability, often referred to in literature as Post-Gynecological Endocrine Syndrome (PG-OES).

In addition to physiological symptoms, patients also exhibited significant changes in emotional state, primarily anxiety, which was assessed using standardized psychometric tools. Anxiety was evaluated as two distinct psychological constructs:

- Trait Anxiety (TA): A stable personality characteristic that predisposes individuals to perceive situations as threatening. It reflects the baseline emotional tone of a person and is considered a constitutional vulnerability.
- State Anxiety (SA): A temporary emotional condition caused by external stressors, such as medical illness or surgery. It is situational and reflects the individual's acute psychological response to stress.

In our study, all post-surgical patients had anxiety levels that exceeded normal reference values. Compared to the non-ADS group, surgical patients had markedly elevated scores in both types of anxiety. Specifically:

- The State Anxiety (SA) score averaged 72.6, nearly double the normal range.

- The Trait Anxiety (TA) score averaged 51.3, also significantly higher than normative values.

By contrast, patients in the control group (non-ADS, non-surgical) had average anxiety scores of 32.5 for TA and 33.8 for SA, which are only slightly elevated but still clinically relevant. This discrepancy underscores that surgical intervention itself—regardless of ADS status—can trigger substantial emotional and autonomic disturbances. These findings support the hypothesis that postoperative anxiety may be a form of reactive psychological distress, emerging as a response to the invasive nature of surgical procedures, hormonal disruption, and recovery challenges. They also suggest the need for pre- and post-operative psychological screening and support to mitigate the risk of chronic emotional and autonomic complications.

Furthermore, clinical symptoms such as tremor, hyperhidrosis, tinnitus, blood pressure instability, and chronic fatigue were more commonly observed in the ADS group, reinforcing the idea that autonomic imbalance often overlaps with emotional dysregulation. According to functional assessments of the autonomic nervous system, it was found that in approximately 75% of patients, post-surgical arterio-circulatory disturbances were directly associated with autonomic dysfunction. These conditions were most commonly diagnosed after radical gynecological surgeries, further supporting the systemic impact of such interventions. In conclusion, our analysis of questionnaire and schematic data reveals that both physiological (ANS) and psychological (anxiety) systems are deeply interconnected, especially in the context of surgical stress and hormonal shifts. Monitoring these systems in a dynamic, individualized manner is critical for developing comprehensive treatment plans and improving long-term outcomes for female patients undergoing reproductive surgeries.

Table №4.6

Psychoemotional state analysis of postoperative patients using the Spielberger-hanin method (in scores)

Anxiety type	Primary group (M±m)	Comparison group (M±m)	Norm	p-value
Reactive anxiety (RA)	72.6±0.243	33.8±0.115	30	<0.05

Trait anxiety (TA)	51.3±0.178	32.5±0.124	30	<0.05

Statistically significant differences between groups: p < 0,05.

According to the data presented, patients who underwent surgery demonstrated significantly higher levels of reactive anxiety (RA) compared to trait anxiety (TA), indicating that surgical intervention affects not only the physiological but also the emotional and psychological dimensions of the patient's condition. This difference suggests that the rise in reactive anxiety is likely a result of the body's acute response to surgical stress, rather than an inherent personality trait, as trait anxiety reflects a stable personal predisposition while reactive anxiety arises in direct response to external stressors.

In all patients who underwent surgery, both RA and TA were elevated beyond normal limits, but RA levels were markedly higher, with an average of 72.6 points versus 51.3 for TA. This discrepancy highlights a pronounced emotional reactivity likely triggered by the psychological stress associated with procedures such as hysterectomy and oophorectomy, especially in those diagnosed with anxiety-depressive syndrome (ADS). These elevated anxiety levels reflect systemic changes in the body postoperatively, including increased sympathetic nervous system activity, neuroendocrine dysfunction, and a disruption in psychoemotional regulation. The central pathological mechanism in Post-Gynecological Endocrine Syndrome (PG-OES) appears to be hypothalamic-pituitary insufficiency, which develops as a response to the absence of ovarian hormones following oophorectomy.

Even in cases where only the uterus is removed, and estradiol production continues via the ovaries, many patients still report heightened anxiety, suggesting that the psychological impact of losing reproductive capacity may serve as a key psychovegetative trigger. This emotional component is not solely attributed to hormonal imbalance but may stem from deeper psychosocial factors related to femininity, fertility, and self-perception. Elevated levels of both types of anxiety in the surgical group support the clinical presence of ADS, which is often associated with early cognitive impairments such as memory problems, difficulty concentrating, mood disturbances, and behavioral changes. In our analysis of cognitive function, patients frequently reported symptoms like memory decline, attentional deficits, dizziness, and emotional instability. These findings underscore the interconnectedness of emotional and cognitive dysfunction following gynecological surgery and emphasize the importance of proactive, multidisciplinary postoperative care that addresses not only physical recovery but also psychological and neurocognitive well-being.

Table №4.7.

Comparison of mean cognitive complaint severity in patients with PG and OES syndromes

Symptom	ADS Patients (%)	Non-ADS Patients (%)	p-value
Memory impairment	80	10	<0,001
Attention deficits	85	20	0,05
Decreased work capacity	78	20	0,001
Vertigo (dizziness)	50	0	0,05
Behavioral disturbances	30	0	<0,05
Mood instability	100	20	<0,05

Statistically significant differences between groups: $p < 0,05$.

As seen in the table, cognitive-related complaints were more prominently expressed in patients diagnosed with anxiety-depressive syndrome, particularly those who developed the condition following the loss of reproductive capacity. This indicates a strong psychological and neurocognitive response to the physiological and emotional impact of surgical menopause or reproductive organ removal.

Table №4.8

Characteristics of cognitive function according to the MOCA assessment

Parameter	ADS Patients	Non-ADS Patients
Orientation	8,6	10
Sensory Perception	2,8	3
Attention and Calculation	3,2	4
Memory	2,0	3
Speech Functions	7,8	8,9
Total Score	24,6	28,9

The mean MoCA score in the primary group was 24.6, while in the comparison group it was 28.9, indicating the presence of mild cognitive impairment in the primary group. In contrast, no such impairments were observed in the comparison group. The primary group demonstrated lower performance across all cognitive domains, including orientation, perception, attention, and memory, suggesting a broader decline in cognitive functioning.

Memory assessment. Given that memory is a critical cognitive function, its evaluation is essential in understanding its influence on women's work capacity, social adaptability, and overall quality of life. In this study, memory was assessed using Luria’s 10-word memorization test, a validated neuropsychological tool used to evaluate immediate and delayed recall performance.

Table №4.9

Average memory assessment scores

Repetition	ADS Patients (Mean±SD)	Non-ADS Patients (Mean±SD)	p-value

1st Repetition	5,0±1,6	8,6±0,71	<0,001
5th Repetition	5,4±1,2	9,6±1,04	<0,001
10th Repetition	4,5±0,87	8,3±0,61	<0,001

Statistically significant differences between groups: - p<0,001.

In the group with ADS (Anxiety-Depressive Syndrome), an average of 5.0±1.6 words were recalled during the first repetition, while in the comparison group this figure was 8.6±0.71. This finding indicates impaired working memory. During the fifth repetition, the main group recalled 5.4±1.2 words, compared to 9.6±1.04 in the group without ADS, suggesting impaired dynamic memory. By the tenth repetition, the ADS group recalled 4.5±0.87 words, whereas the non-ADS group recalled 8.3±0.61 words, indicating long-term memory impairment in the main group.

Comprehensive treatment of anxiety-depressive syndrome after hysterectomy and oophorectomy. During the study, all patients received standard therapy, which included the following components: hormone replacement therapy, vegetotropic therapy, neurometabolic agents, vasoprotectors, antioxidants, and general tonic treatments. These components primarily aimed to effectively treat vegetative and cognitive disorders, by maintaining optimal hormonal balance in the body, restoring the function of the autonomic nervous system, reducing cognitive impairments, and improving the general condition of the patients.

The study showed that cognitive impairments and anxiety-depressive syndrome, resulting from radical surgical interventions, are common among women and require appropriate postoperative therapeutic strategies. The pathogenesis of cognitive impairments following radical surgery was studied, with psychological and cognitive disorders due to the removal of reproductive organs identified as the primary factor. Cognitive impairments are often accompanied by anxiety-depressive syndrome, which frequently develops after radical surgeries and significantly affects the psycho-emotional health of patients. The main goals of treatment are to maintain hormonal balance, restore cognitive abilities, and ensure emotional stability.

Efficacy of fluvoxamine as an antidepressant. Research evidence indicates that the pathogenesis of cognitive impairments following radical surgeries is well understood, and Fluvoxamine has been selected as an antidepressant for their treatment. Radical surgeries—particularly the removal of reproductive organs—can lead to severe cognitive and psychological disturbances in patients, due to the significant impact of hormonal changes on brain function. These cognitive disorders often manifest alongside anxiety and depression, leading to a reduced quality of life.

In such cases, antidepressants play a crucial role in improving cognitive functions, primarily by stabilizing emotional states and regulating neurochemical changes in the brain. Fluvoxamine is widely used internationally to treat anxiety and depressive syndromes and acts primarily as a selective serotonin reuptake inhibitor (SSRI). Its use is recommended based on multiple international guidelines and clinical protocols.

International Guidelines on Fluvoxamine. Fluvoxamine is included in key international recommendations, notably the World Health Organization (WHO) Essential Medicines List. In this list, it is recommended for the treatment of F41.2 (mixed anxiety and depressive disorder) and F42 (obsessive-compulsive disorder). Based on international guidelines and clinical trials, Fluvoxamine has been proven to be a safe and effective medication.

WHO expert recommendations. The 24th WHO Expert Committee reviewed the efficacy and safety of Fluvoxamine. They emphasized that Fluvoxamine is particularly effective in treating anxiety disorders, mixed anxiety and depressive states, and obsessive-compulsive disorder. Clinical trials have shown that selective serotonin reuptake inhibitors (SSRIs), including Fluvoxamine, are distinguished by their high safety profile.

Clinical recommendations for fluvoxamine use. Fluvoxamine dosing is determined individually for each patient. The initial dose typically ranges from 50–100 mg, and if necessary, can be increased up to 300 mg. Clinical trials have demonstrated Fluvoxamine's effectiveness in the treatment of depression, obsessive-compulsive disorder, and various anxiety conditions. Fluvoxamine is included in the official list of registered medicines in the Republic of Uzbekistan and is prescribed based on protocols approved by the Ministry of Health. Its use complies with the requirements of the State Pharmacopoeia of Uzbekistan, which includes processes for quality control, state registration, and certification.

Pharmacological properties of fluvoxamine. The main active substance, Fluvoxamine, inhibits the reuptake of the neurotransmitter serotonin, thereby improving the patient’s mental state. Serotonin is a key chemical regulator of mood in the brain. A decrease in its levels is often associated with symptoms such as depression, anxiety, and impaired cognitive functions. By increasing the concentration of serotonin at the synaptic level, Fluvoxamine helps stabilize emotional and cognitive functioning.

One of Fluvoxamine's key advantages is its selective action on serotonin, with minimal effects on other neurotransmitters such as noradrenaline and dopamine. Although these neurotransmitters also play roles in emotional regulation, their imbalance can lead to negative side effects. Fluvoxamine helps maintain the balance of these chemicals, ensuring both safety and efficacy. The drug also has minimal influence on the autonomic nervous system and supports overall improvement in the patient's condition.

Several studies have shown that Fluvoxamine is effective in treating anxiety-depressive syndrome and cognitive impairments following radical surgeries. Notably, significant clinical improvement can be observed within one week, confirming the drug’s mechanism of action and its positive impact on patient outcomes.

Dosing strategy and treatment course. In the study, 30 patients were prescribed Fluvoxamine, with an initial evening dose of 100 mg. Depending on symptom severity, the dose was gradually increased to a maximum of 300 mg. Initial doses were increased gradually to allow the body to adapt and reduce potential side effects. The maximum recommended dose is 300 mg per day, typically reached through a titration schedule—for example, starting at 50 mg and gradually increasing to 150 mg or higher.

Patient condition is closely monitored throughout this process to adjust dosing accordingly. The treatment course usually lasts 6 months, but may be extended until the patient reaches a stable condition. Continuous therapy plays a key role in maintaining long-term emotional and cognitive health. During the maintenance phase, a daily dose of 100 mg is used to reinforce treatment results. By inhibiting serotonin reuptake, Fluvoxamine stabilizes mood and improves cognitive function. It is a safe and effective option for long-term use, contributing significantly to improved quality of life in patients.

Adverse effects of fluvoxamine. During the first two weeks of Fluvoxamine therapy, patients may experience some transient side effects. These are generally short-lived and are linked to the medication's impact on the neurotransmitter system. The most commonly reported adverse effects include nausea, dry mouth, drowsiness, headache, and tremors. These symptoms typically resolve quickly and are associated with the body's adaptation to long-term Fluvoxamine use.

A mild increase in heart rate (by approximately 2–6 beats per minute) has also been observed in some cases. This can result from both the drug’s pharmacological action and the underlying depression itself. However, Fluvoxamine has minimal influence on the sympathetic nervous system, making such side effects transient and generally harmless as the body adapts.

Safety of long-term use. Research has shown that Fluvoxamine is safe even at higher doses, and no serious risks have been associated with prolonged use. Its complex mechanism of action primarily targets emotional stability while exerting minimal effects on other body systems. This makes it a reliable option for long-term therapy.

Efficacy of fluvoxamine in treating cognitive impairments. Clinical findings indicate that anxiety-depressive syndrome occurs in approximately 75% of women following radical surgical procedures. These psychological conditions are often closely tied to cognitive impairments, significantly lowering patients' quality of life. Patients who received Fluvoxamine treatment experienced marked emotional and cognitive improvements, confirming the drug's effectiveness.

Fluvoxamine was administered over a 60-day period, with clinical and neurological assessments performed at the end of this duration. Improvements were seen in patients' emotional well-being, concentration, and memory. The antidepressant effect of Fluvoxamine extends beyond merely alleviating depressive symptoms; it also plays a significant role in restoring cognitive abilities and enhancing brain function.

Studies show that Fluvoxamine reduces cognitive impairments by lowering anxiety and depressive symptoms, without heavily affecting overall brain function. This targeted action supports the recovery of cognitive performance, especially in the post-surgical period.

Fluvoxamine therapy has proven to be effective in facilitating psycho-emotional and cognitive recovery after radical surgeries. In the study, cerebral symptoms in patients suffering from anxiety-depressive syndrome and cognitive impairment after surgery were assessed before and after Fluvoxamine treatment. These symptoms included headache, dizziness, memory and concentration decline, sleep disturbances, head and ear noise, fatigue, and irritability.

Below is a table summarizing the symptoms before and after treatment, including the percentage of improvement:

Table №4.10.

Changes in cerebral complaints before and after fluvoxamine therapy

Complaint	Before Treatment	After Treatment	Effectiveness (%)	p-value	r-value

Headache	22	9	59%	0,05	-0,5
Dizziness	15	3	80%	0,001	-0,5
Memory and attention decline	24	3	87%	0,005	-0,5
Sleep disturbances	28	0	100%	0,005	-0,5
Head and ear noise	15	6	60%	0,005	-0,5
Fatigue	26	2	93%	0,005	-0,5
Irritability	28	0	100%	0,005	-0,5

*Statistically significant differences between groups: - *p<0,05, **- p<0,001.*

Significant reduction in cerebral complaints after fluvoxamine therapy

Following Fluvoxamine therapy, a marked reduction in cerebral complaints was observed. Headache was reported in 22 cases before treatment, which decreased to 9 cases after therapy, reflecting an effectiveness rate of 59%. Dizziness was initially present in 15 patients, which dropped to 3 cases post-treatment, indicating 80% effectiveness. Memory and attention impairment—considered among the most severe cognitive dysfunctions—was noted in 24 patients before treatment, and decreased to only 3 cases after therapy, achieving an 87% improvement rate. Sleep disturbances were one of the areas most effectively addressed by Fluvoxamine, with a 100% success rate, indicating complete resolution of these complaints post-treatment.

The symptom of head and ear noise (tinnitus) was reported in 15 patients initially, and decreased to 6 cases after treatment, showing 60% effectiveness. Fatigue, observed in 26 patients prior to therapy, was reduced to just 2 cases following treatment, with an effectiveness rate of 93%. Irritability was noted in 28 patients before treatment. The negative correlation coefficient (r = –0.5) indicates a strong inverse relationship between pre- and post-treatment symptoms. This means that patients who initially had more severe symptoms showed the greatest improvement after therapy. Post-treatment, 100% effectiveness was achieved in eliminating irritability, with all symptoms fully resolved. According to the study results, Fluvoxamine significantly reduced cerebral complaints and demonstrated high efficacy in improving patients' cognitive and emotional status. The complete resolution of symptoms such as sleep disturbances and irritability, along with significant improvement in other complaints, supports the recommendation of Fluvoxamine as an effective treatment for anxiety-depressive syndrome during the postoperative period following radical surgical interventions.

Table №4.11.

According to the questionnaire and schematic data, the presence and severity of anxiety-depressive syndrome (ADS) were assessed.

Parameter	Mean ± SE (Before → After Treatment)	Improvement (%)	p-value
Questionnaire	54,1 ± 0,241 → 28,8 ± 0,1	49%	<0,001
Schema evaluation	41,6 ± 0,1 → 27,2 ± 0,01	46%	0,05

*Statistically significant differences between groups: -*p < 0,05.*

Analysis of the tabulated data demonstrates that the indicators measured after treatment with Fluvoxamine improved significantly. These findings indicate a substantial reduction in Anxiety-Depressive Syndrome (ADS) scores**,** as evaluated through both questionnaire-based and schematic assessment tools**.** The observed improvements reflect the treatment's strong impact on psycho-emotional well-being.

The results suggest that the underlying dynamics of ADS are closely related to vascular system function and peripheral vasomotor regulation**.** These components of autonomic function appear to be particularly responsive to treatment, as higher therapeutic efficacy was noted in complaints linked to cerebrovascular instability, fatigue, and vegetative symptoms**.**

Thus, administration of Fluvoxamine was associated with clinically significant improvements in both vegetative and emotional parameters**,** supporting its role in managing ADS—particularly when these symptoms emerge after radical surgical procedures (e.g., hysterectomy, oophorectomy).

Table №4.12

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Type of anxiety	Before treatment (M±m)	After treatment (M±m)	p-value	r-value
Reactive (State anxiety)	72,6 ± 0,243	42,1 ± 0,0143	<0,01	0,85
Personal (Trait anxiety)	51,3 ± 0,178	33,4 ± 0,0113	<0,01	0,78

*Statistically significant differences between groups: -*p < 0,05.*

A comprehensive evaluation of anxiety symptom dynamics revealed a significant therapeutic effect of Fluvoxamine on both reactive (state) and trait (personal) forms of anxiety. These findings are particularly important in the context of post-surgical anxiety-depressive syndrome (ADS), where emotional instability and cognitive dysfunction are often exacerbated. Reactive anxiety refers to the individual’s acute emotional response to external stressors, such as medical procedures, uncertainty, or physical discomfort. It is often situational and can vary greatly depending on environmental stimuli. Baseline Measurement: Prior to treatment, the mean reactive anxiety score was 72,6 points according to the Spielberger state-trait anxiety inventory (STAI). This score falls within the severe anxiety range, indicating that patients were experiencing heightened emotional reactivity, likely due to the psychological burden of their medical condition and its treatment.

Post-Treatment Measurement: After a structured course of Fluvoxamine therapy, the reactive anxiety score decreased to 42,1 points. This represents a 42% reduction, suggesting a substantial attenuation of acute stress reactivity. Patients reported feeling calmer, less overwhelmed in challenging situations, and more emotionally balanced. Statistical ignificance: The correlation coefficient (r = 0,85) demonstrates a strong positive association between pre- and post-treatment scores. This indicates that individuals with the highest baseline anxiety experienced the most pronounced improvements, reflecting the targeted and proportional efficacy of the treatment. Additionally, the p-value (<0,01) confirms that the improvement is statistically significant, meaning the likelihood that these results occurred by chance is less than 1%.

These outcomes suggest that Fluvoxamine has a robust anxiolytic effect on acute stress-related emotional disturbances, making it a reliable pharmacological option for managing state-dependent anxiety symptoms, particularly during the early recovery phase after surgery or serious illness. Trait anxiety represents a long-term, personality-linked tendency to perceive various situations as threatening and to respond with chronic worry, fear, or tension. Unlike reactive anxiety, it is relatively stable over time and reflects a person's baseline emotional state. Baseline Measurement: The average trait anxiety score prior to treatment was 51,3 points, which is moderate-to-high and indicates that patients possessed a heightened predisposition toward chronic anxiety, irrespective of immediate stressors. Post-Treatment Measurement: After Fluvoxamine therapy, the trait anxiety score decreased to 33,4 points, yielding a 35% reduction. This shift reflects a notable improvement in patients' underlying emotional resilience and psychological well-being. This reduction in trait anxiety is clinically important. While state anxiety may fluctuate with circumstances, trait anxiety is more resistant to change and often requires longer-term intervention. The observed improvements suggest that Fluvoxamine not only alleviates situational anxiety but also exerts a stabilizing effect on persistent, deeply rooted psychological vulnerabilities.

The ability to reduce trait anxiety implies that Fluvoxamine may promote adaptive neurochemical and cognitive restructuring, allowing patients to reframe negative thought patterns, better regulate emotions, and cope with everyday stress more effectively.

Clinical relevance andiImplications-the findings from this analysis support the use of Fluvoxamine as a dual-acting antidepressant and anxiolytic, capable of improving both acute and chronic anxiety dimensions. Given the significant improvement in both reactive and trait anxiety scores, and the strong statistical backing (r values and p-values), Fluvoxamine demonstrates high therapeutic potential, especially in post-operative and high-stress clinical populations.

This dual-action profile is particularly advantageous in treating women recovering from radical gynecological surgeries, who are at elevated risk for both situational distress and persistent anxiety-depressive disorders.

Cognitive performance was evaluated using the Montreal Cognitive Assessment (MoCA), a widely validated tool designed to screen for mild cognitive impairment and assess various domains including memory, attention, language, executive function, and orientation.

- Pre-treatment Evaluation: The average MoCA score before treatment was 24.6 points, which is slightly below the normal cognitive threshold. Scores in the 22–25 point range typically indicate the presence of mild cognitive impairment.
- Post-treatment Evaluation: After completing Fluvoxamine therapy, the average score increased to 28 points, moving into the normal cognitive function range (25–30 points). This shift strongly indicates a clinically significant improvement in cognitive performance.

The data show that a majority of patients experienced an upward shift from borderline scores (22–25) to within the normal range, further confirming enhanced functioning across multiple cognitive domains.

Correlation Between Anxiety Reduction and Cognitive Improvement

The observed reduction in anxiety levels, particularly in reactive anxiety, signifies a decrease in the intensity of patients' emotional responses to stress. This suggests that individuals became more emotionally stable and less prone to situational distress after treatment.

Moreover, trait anxiety, reflecting chronic and personality-related anxiety, also showed a significant decline. This indicates a broad improvement in patients' baseline psychological condition, contributing to greater emotional resilience and adaptability.

Importantly, these emotional improvements were accompanied by measurable cognitive gains. Enhanced scores on the MoCA scale confirm that Fluvoxamine therapy contributed to improvements in attention, memory, orientation, and executive functions—all of which are often impaired in individuals with anxiety-depressive syndrome.

Fluvoxamine therapy not only reduced both state and trait anxiety levels, but also led to significant cognitive recovery. The marked increase in MoCA scores—from borderline impairment to normal range—demonstrates the drug's effectiveness in restoring neurocognitive stability. These outcomes reinforce Fluvoxamine's role as a multifunctional psychotropic agent, capable of addressing both emotional dysregulation and cognitive decline in patients with post-surgical anxiety-depressive syndrome.

Table №4.13.

Average MoCA scores for cognitive domains during treatment

Parameter	**Before treatment**	**After treatment**	**% change**	**p-value**
Orientation	8,6	9,2	6,5%	0,05
Comprehension	2,8	3,0	6%	0,001
Attention and calculation	3,2	3,8	6%	0,001
Memory	2,0	2,3	15%	0,05
Language function	7,8	8,0	2,5%	0,05

Total score	24,6	28,0	12%	0,05

*Note: * Statistical significance between groups – p < 0.05.*

Clinical data indicate that Fluvoxamine therapy significantly improves not only anxiety-depressive symptoms but also plays a critical role in restoring the human factor—that is, the patient's cognitive capacity, psychological stability, and overall functional well-being.

Cognitive functions were assessed using the Montreal Cognitive Assessment (MoCA), a validated tool covering five major cognitive domains: orientation, perception (comprehension), attention and calculation, memory, and language functions. These domains represent core components of cognitive health and are closely linked to the individual's ability to function independently and meaningfully in daily life.

1. Orientation. Orientation refers to a person's awareness of time and place. It is fundamental for maintaining connection with reality and is crucial for everyday functioning. Score before treatment: 8.6, Score after treatment: 9.2 Improvement: 6.5%.

This change reflects an enhanced ability to accurately interpret temporal and spatial cues, signaling recovery of fundamental awareness.

2. Perception (Comprehension). This domain involves the brain's ability to receive, process, and interpret sensory information from the environment. Score before treatment: 2.8, Score after treatment: 3.0, Improvement: 6%.

An improvement in this area suggests better environmental adaptation and quicker response to external stimuli, contributing to improved emotional resilience.

3. Attention and calculation. These functions are critical for logical thinking, decision-making, and planning. Score before treatment: 3.2, score after treatment: 3.8, improvement: 6%.

Enhanced attention and mental arithmetic ability indicate strengthened executive functioning and greater mental control.

4. Memory. Memory plays a vital role in both personal life and professional activities. Score before treatment: 2.0, score after treatment: 2.3, improvement: 15%

This domain showed the highest rate of improvement, indicating that Fluvoxamine has a strong positive influence on memory consolidation and retrieval.

5. Language function. Language is key to effective communication and social interaction. Score before treatment: 7.8, score after treatment: 8.0, improvement: 2.5%

While modest, the improvement suggests better fluency and clarity in verbal expression.

The total MoCA score increased from 24.6 to 28.0, reflecting an overall 12% improvement. This shift marks a transition from borderline cognitive impairment to normal cognitive function, demonstrating the comprehensive neurocognitive benefit of Fluvoxamine therapy. Fluvoxamine's therapeutic effect extended beyond cognition to include a profound impact on emotional regulation and the human experience of anxiety. Reactive anxiety, related to situational stress responses, dropped from 72.6 to 42.1 points (42% reduction), indicating greater emotional resilience and reduced stress sensitivity. Trait anxiety, a more stable and personality-related form, decreased from 51.3 to 33.4 points (35% reduction), pointing to lasting improvements in baseline emotional well-being.

These changes highlight Fluvoxamine's dual role in stabilizing mood and restoring cognitive and emotional equilibrium.

Table№4.14

Average scores of the 10-word memory recall test during the treatment process

Repetitions	1st recall	5th recall	10th recall	p-value
Before treatment	5,0	7,4	7,5	0,05
After treatment	7,6	8,3	8,3	0,05

Note: * The difference between groups is statistically significant at p < 0,05.

Evaluation of memory improvement using luria's method during fluvoxamine therapy. To assess changes in memory performance during treatment, the Luria 10-word recall test was employed. This method involves presenting patients with a list of 10 words and asking them to recall as many as possible across multiple repetitions. The test evaluates short-term (operational) and long-term (delayed) memory performance.

1st Recall (Initial Repetition)

- Before treatment: Patients could recall an average of 5.0 words, indicating low initial memory retention and impaired operational memory.
- After treatment: The recall improved significantly to 7.6 words. This substantial increase in the first repetition shows a rapid and marked improvement in initial encoding and attention-related memory processes, suggesting that Fluvoxamine positively influences early-stage memory acquisition.

5th Recall (Mid Repetition)

- Before treatment: The average recall was 7.4 words.

- After treatment: This score increased to 8.3 words. This improvement reflects an enhanced learning curve and memory consolidation. Patients were able to retain more information across repetitions, indicating that working memory capacity and information processing efficiency were improved following therapy.

10th Recall (Final Repetition)

- Before treatment: Patients recalled an average of 7.5 words.
- After treatment: The score increased to 8.3 words. This result highlights enhancement in long-term memory, indicating that Fluvoxamine supports memory retention over time. The stability of this score after multiple repetitions confirms improved cognitive resilience and sustained mental performance.

Interpretation of the Results

These findings indicate that Fluvoxamine contributes to the restoration of both operational (short-term) and long-term memory functions. Notably:

- By the 5th repetition, patients' average recall rose close to normal values, improving from 7.4 to 8.3 words.
- By the 10th repetition, the sustained increase in recall further confirms the recovery of delayed recall ability, a key component of long-term memory.

This dynamic enhancement of memory performance reflects the cognitive benefits of Fluvoxamine, particularly in patients with postoperative anxiety-depressive syndrome caused by hysterectomy and oophorectomy.

The analysis confirms that Fluvoxamine therapy is effective in improving memory function, which in turn leads to better cognitive performance and emotional stability. The recall scores observed before and after treatment show clear clinical efficacy, with the restoration of both dynamic and delayed memory functions.

Moreover, the observed cognitive improvements contributed to the overall stabilization of the patient's condition, reflecting better adaptation, orientation, and quality of life. These findings support the inclusion of Fluvoxamine in comprehensive treatment protocols for cognitive impairment and anxiety-depressive syndrome following radical gynecological surgeries.

Cognitive impairments that develop following radical surgical procedures on the reproductive organs in women represent one of the significant and pressing issues in modern neurology. There is an urgent need to find comprehensive solutions and develop appropriate therapeutic strategies tailored to these cases. Women frequently experience anxiety-depressive syndromes in the postoperative period, particularly following the removal of reproductive organs through radical surgeries such as hysterectomy and oophorectomy.

The broad biological influence of sex hormones on the female body explains the emergence of various adverse symptoms and complications when these hormones are abruptly diminished or cease entirely. The deficiency of sex hormones resulting from the surgical removal of reproductive organs can lead to a wide spectrum of psycho-emotional, cognitive, and autonomic disturbances. These outcomes underscore the importance of establishing precise treatment and preventive measures to mitigate severe postoperative complications.

The post-hysterectomy and post-oophorectomy syndrome constitutes a complex symptomatology, comprising neuropsychiatric, autonomic–vasomotor, and endocrine disorders. These disturbances arise after the surgical removal of reproductive organs and are especially pronounced in women of reproductive age who are still in the active phase of their reproductive life. The underlying cause is often surgical intervention, such as oophorectomy (removal of the ovaries) or hysterectomy (removal of the uterus).

Currently, there is no universally accepted, reliable set of criteria for accurately assessing and diagnosing such conditions. Furthermore, although various therapeutic approaches are employed to manage these syndromes, the majority lack robust validation through long-term clinical trials. This creates a need for well-structured, evidence-based methodologies both for diagnosis and for evaluating the efficacy of treatment interventions. Enhancing our understanding of the human dimension of this issue remains crucial in preserving the overall quality of life and psychological well-being of affected women.

Post-hysterectomy and post-oophorectomy syndromes in women frequently lead to complications involving the cardiovascular, endocrine, and nervous systems. The surgical deprivation of sex hormones results in a disruption of homeostasis, causing a wide range of physical and psychological changes. These changes significantly diminish the quality of life in affected women.

Following hysterectomy and oophorectomy, the female body attempts to adapt to neuroendocrine restructuring. During this transitional phase, post-hysterectomy and post-oophorectomy syndromes may develop. At this stage, the use of antidepressants such as **Fluvoxamine** is considered an effective therapeutic approach. Research has demonstrated that antidepressants like Fluvoxamine contribute to the alleviation of cognitive and psycho-emotional disturbances.

The use of Fluvoxamine has shown effectiveness in the treatment of various cognitive impairments. Its impact has been associated with improvements in memory, attention, orientation, and other cognitive functions. The enhanced cognitive and psycho-emotional state of patients has led to a noticeable improvement in their overall quality of life.

The clinical efficacy of Fluvoxamine has been confirmed through clinical studies. According to research findings, patients experienced significant improvements following treatment, underscoring the clinical value of Fluvoxamine. In many cases, patients nearly completely recovered from cognitive impairments post-surgery, indicating the important role this medication may play in addressing postoperative complications associated with radical gynecological surgeries.

Based on recent research, it has been established that women often develop various cognitive impairments following radical surgical procedures such as oophorectomy and hysterectomy. In such cases, treatment with Fluvoxamine has shown effective results. During the treatment process, patients demonstrated improvements in memory, attention, and overall psychoemotional state. This therapeutic approach is important not only for mitigating cognitive dysfunctions but also for enhancing overall quality of life.

Hysterectomy and oophorectomy significantly affect the autonomic nervous system and the state of neuroendocrine regulation in women. These surgical interventions, particularly in women of active reproductive age, disrupt homeostasis and lead to profound neuroendocrine imbalances. Therefore, studying the effects of these operations on the female body—especially regarding the autonomic nervous system—is of great importance.

A comprehensive clinical study was conducted to assess the condition of the autonomic nervous system and psychoemotional status in women post-surgery. The aim of this study was to investigate neurological disturbances arising from surgical interventions and to develop differential therapeutic approaches to their treatment.

Patients were monitored closely over a two-month period. Throughout the examinations, signs of vegetative dystonia (VD) and psychoemotional conditions were assessed, alongside evaluations of cognitive functions. Some patients underwent two courses of treatment with the antidepressant Fluvoxamine, and their clinical status was carefully monitored and evaluated.

As a result of the study, anxiety-depressive syndrome was identified in 75% of the female patients, while 25% did not exhibit this syndrome. Anxiety-depressive manifestations were primarily associated with autonomic and vascular system disturbances, as well as psychoemotional dysfunctions.

Har bir bemorda vegetativ nerv tizimining funksional holati va VDSning mavjudligi aniqlandi. Bu VD ning belgilari anketalar va sxemalar yordamida baholandi. Tadqiqotlar shuni koʻrsatdiki, VD har ikki guruhda ham birdek uchraydi, ya’ni jismoniy va sifatli belgilarga koʻra VD belgilarining har ikki guruhda bir xil darajada ekanligi aniqlandi. Bu natijalar shuni anglatadiki, kichik chaqalaklar organlarida radikal operatsiyalar oʻtkazilgan ayollarda psixovegetativ sindromning rivojlanishi katta darajada namoyon bo‘ladi. Bachadon va tuxumdonlarni olib tashlash xirurgik aralashuvlar natijasida vegetativ-vazomotor buzilishlarni keltirib chiqaradi, bu esa ayollarning umumiy holatiga salbiy ta’sir qiladi.

Gipotalamus-gipofiz-yo‘qolgan organlar zanjiri buzilganda gipotalamus-gipofizyo‘qotilgan reproduktiv organlar tizimining to‘liq disfunksiyasi paydo bo‘ladi. Bu disfunksiya vegetativ nerv tizimiga va ayollarning umumiy emotsional holatiga ta’sir ko‘rsatib, PG-OES (postgisterektomiya va ovarektomiya sindromi) kabi qiyin asoratlarga olib keladi. Ushbu sindromning belgilari ko‘pincha gipotalamusgipofiz-regulyatsiya tizimida buzilishlarni namoyon qiladi. Xavotirlik darajasini o‘rganish davomida, tashvish-depressiya sindromiga chalingan bemorlarda xavotir ko‘rsatkichlari GE va OE bo‘yicha guruhlar bilan solishtirilganda ancha yuqori ekanligi aniqlandi. Har bir bemorda operatsiyadan keyin reaktiv tashvishlilik (RT) va shaxsiy tashvishlilik (SHT) darajalari tekshirildi. Natijalar shuni ko‘rsatdiki, barcha bemorlarda operatsiyadan keyin RT va SHT ko‘rsatkichlari pasaygan bo‘lsa-da, RT ko‘rsatkichi SHTga nisbatan ko‘proq o‘zgarish ko‘rsatgan. Bu holat shaxsiy xususiyatlar va shaxsning operatsiyaga bo‘lgan reaksiyasi bilan bog‘liq bo‘lishi mumkin. Xususan, operatsiyaning emotsional ta’siri bemorlarda yuqori darajadagi reaktiv xavotir bilan namoyon bo‘ladi. Shaxsiy xavotir darajasi esa kamroq o‘zgarish ko‘rsatgan.

In each patient, the functional state of the autonomic nervous system and the presence of vegetative dystonia syndrome (VDS) were evaluated. The signs of VDS were assessed using standardized questionnaires and diagnostic scales. The findings revealed that VDS was present equally in both groups under investigation, indicating that the frequency and severity of vegetative symptoms were comparable regardless of group assignment. These results suggest that radical surgeries on the internal reproductive organs in women lead to a significant manifestation of psychovegetative syndrome.

Surgical removal of the uterus and ovaries induces vegetative–vasomotor disturbances, which negatively affect the overall health status of women. When the hypothalamic–pituitary–target organ axis is disrupted due to the loss of reproductive organs, a complete dysfunction of this neuroendocrine system occurs. This dysfunction has a direct impact on the autonomic nervous system and the emotional stability of women, resulting in complex complications such as post-hysterectomy and oophorectomy syndrome (PG-OES).

The clinical signs of this syndrome often reflect regulatory disruptions within the hypothalamic-pituitary system. In the evaluation of anxiety levels, it was found that patients diagnosed with anxiety-depressive syndrome exhibited significantly higher anxiety scores compared to those in the hysterectomy (HE) and oophorectomy (OE) groups without the syndrome.

Each patient's level of state anxiety (SA) and trait anxiety (TA) was assessed postoperatively. Results indicated that although both SA and TA levels decreased following surgery, state anxiety showed a more pronounced change compared to trait anxiety. This finding may be associated with individual psychological characteristics and the patient’s emotional response to the surgical procedure. Specifically, the emotional impact of the surgery appeared to manifest in the form of high levels of reactive anxiety, whereas trait anxiety remained relatively stable.

In our study, postoperative cognitive functions were evaluated using the Montreal Cognitive Assessment (MoCA) and the 10-word memory recall test. The results clearly demonstrated that patients exhibited significant cognitive changes following surgery. These impairments were predominantly observed in patients diagnosed with anxiety-depressive syndrome (ADS), whereas no notable cognitive deficits were found in patients outside this group.

These findings provide valuable insight into the pathogenesis of cognitive dysfunctions following radical surgical procedures, such as hysterectomy and oophorectomy. The data suggest that such surgeries result in systemic changes in women that heavily affect the nervous system and emotional regulation.

Clinical, autonomic, and neuropsychological assessments confirmed that systemic dysfunctions develop after surgery on internal reproductive organs. These changes primarily manifest as vegetative, psychoemotional, and cognitive disturbances. Surgeries like hysterectomy and oophorectomy can disrupt the autonomic nervous system and cause multiple psychoemotional issues, particularly through their impact on the hypothalamic-pituitary-ovarian-uterine (HPOU) axis. The removal of the uterus and ovaries leads to neuroendocrine dysregulation, which induces deep physiological and psychological alterations with a substantial impact on women’s quality of life.

Studies on anxiety and depressive disorders reveal that patients with ADS exhibit very high levels of anxiety, which persist even in the postoperative period. Specifically, state anxiety (SA) was found to be significantly higher than trait anxiety (TA), indicating that surgical stress elicits strong reactive emotional responses in susceptible individuals. The heightened SA compared to TA suggests that many women respond acutely to the emotional impact of surgery, while elevated TA values point toward long-term anxiety vulnerability in this patient population. In addition to anxiety, the study found a strong association between psycho-vegetative and neuroendocrine changes and cognitive impairment in patients with ADS. Within the scope of the study, the effectiveness of Fluvoxamine in managing symptoms of ADS was also analyzed. Comparison of the clinical status of patients before and after treatment with Fluvoxamine showed clinically significant improvements, affirming the drug's therapeutic efficacy in this context.

The findings of this research indicate that women with ADS not only suffer from physiological disruptions but also from significant cognitive and emotional impairments. As such, a comprehensive, multidisciplinary treatment approach is required. This should include interventions to manage headaches, cognitive deficits, sleep disturbances, fatigue, and mood instability. For clinicians, accurate individual assessment and tailored therapeutic strategies are essential to effectively support recovery and improve overall quality of life in this patient group. The results of this study demonstrate that patients with anxiety-depressive syndrome (ADS) experience not only physiological disturbances but also clinically significant cognitive and emotional impairments. This underscores the necessity for a comprehensive treatment approach for ADS patients. Key components of such treatment should include management of headaches, prevention of cognitive and psychological dysfunction, and reduction of insomnia and fatigue. For healthcare providers, it is critical to perform individualized assessments of patients with ADS and implement multidimensional therapeutic interventions that address their unique needs.

This study serves as a foundational investigation into the clinical and cognitive effects of ADS and provides a basis for further exploration. Future research should focus on the pathogenesis of physiological and cognitive disturbances in ADS patients and the development of effective treatment protocols. It is essential to conduct additional studies aimed at clarifying the mechanisms behind these impairments and establishing targeted therapeutic strategies to better manage the core symptoms.

Furthermore, the identification and effective treatment of cognitive and emotional disorders (CEDs) associated with ADS are of great importance for improving patients' overall quality of life. Continued investigation in this area will support the creation of more personalized, evidence-based interventions and advance clinical practices in the management of postoperative neuropsychological complications.

CONCLUSIONS

1. Radical surgeries performed on the pelvic organs in women lead to the development of neurological complications. These complications manifest through various clinical syndromes, with the most common being anxiety-depressive syndrome (ADS), identified in 75% of patients.
2. Anxiety-depressive syndrome contributes to a significant decline in cognitive functions, particularly impairing memory and attention. The presence of ADS in 75% of patients reflects the severity of emotional and psychological changes experienced postoperatively.
3. The cognitive abilities of patients were evaluated using the MoCA test and the 10-word memory recall test. The results showed that 85% of patients experienced a decline in memory, and 65% showed reduced attention, both of which had a substantial negative impact on daily functioning.
4. Fluvoxamine not only alleviates symptoms of anxiety and depression but also plays a significant role in improving overall cognitive and psychoemotional states in more than 87% of patients, while exerting minimal impact on the autonomic nervous system.

Thus, Fluvoxamine has been evaluated as an effective and broad-spectrum therapeutic agent in the treatment of postoperative cognitive impairments.

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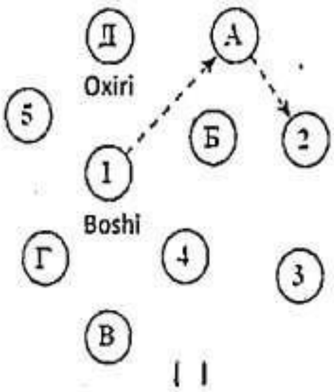
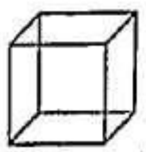
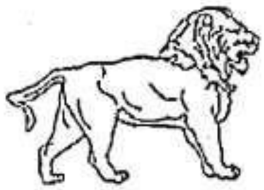
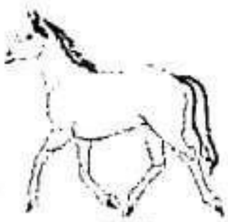
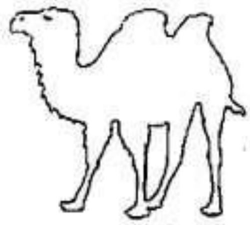
I L O V A - 1

MONREAL SHKALASI
(kognitiv funksiyalarni baholash uchun)

F.I.SH:………………………………………………………
Ma'lumoti:……………………………………………………
Tug'ilgan yili va kuni:………………………………………
Tekshiruv o'tkazilgan kun:…………………………………

Optik-fazoviy funksiyalarni bajara olish qobiliyati		Raqamli soatni chizing va unda 11 dan 10 daqiqa o'tgan bo'lsin (3 ta punkt)	Ball
 [ ]	 Kubikni xuddi shunday chizing [ ]	[ ] [ ] [ ] Shakli Raqamlari Strelkalari	__/5

Nomini aytish			
 [ ]	 [ ]	 [ ]	__/3

Xotira Sinaluvchi vrach o'qigan so'zlarni takrorlaydi va natija 1-qatorga yoziladi. Vrach shu so'zlarni yana		Oyna	Yap-roq	Ma-chit	Dar-yo	Pax-ta	Ball qo'-yil-may-di
	1-imko-niyat						

takrorlaydi va natija 2-qatorga yoziladi. Sinaluvchidan bu so'zlarni eslab qolish so'raladi.							
	2-imko-niyat						

Diqqat. Raqamlarni o'qing (1 soniyada 1 raqam). To'g'ri tartibda takrorlang [ ] 2 1 8 5 4 Teskari tartibda takrorlang [ ] 7 4 2							__/2
Harflarni o'qing. Har gal A ni o'qiganda sinaluvchi qo'li bilan stolga urib qo'yadi. Sinaluvchi 2 marotaba xatoga yo'l qo'ysa, ball qo'yilmaydi. [ ] F B A V M N A A J L L B A F A K D E A A A J A M O F A A B							__/1
100 dan 7 ni ayirib boring. [ ] 93 [ ] 86 [ ] 79 [ ] 72 [ ] 65							__/3
Nutq. Takrorlang: Men shuni bilamanki, Karim – bu menga yordam beradigan odam. [ ] Hovlida kuchuk paydo bo'lganida, mushuk ko'chaga qochib ketardi. [ ]							__/2
Nutq tezligi/bir daqiqa ichida «L» bilan boshlanadigan so'zlarni iloji boricha ko'proq ayting. [ ]__(N≥11)							__/1
Abstrakt fikrlash Olma – apelsin = meva; [ ] poyezd – velosiped [ ] spidometr – soat							__/2
Qayta esga tushirish (eslatmasdan)	Avvalgi so'z-larni qayta esga tushirish	**Oyna** [ ]	**Yaproq** [ ]	**Machit** [ ]	**Daryo** [ ]	**Paxta** [ ]	__/5
To'ldirish shart emas (ball ham qo'yil-maydi)	O'xshash so'zlarni eslatish						
	Taklif etilgan so'zlardan tanlash						
Orientatsiya	[ ] Sana [ ] Oy [ ] Yil [ ] Hafta kuni [ ] Joy [ ] Shahar						__/6
Ballar yig'indisi ____/30	Norma 26 – 30						

(Z.R.Ibodullaev "Asab kasalliklari"kitobidan ,2013y)

L O V A- 2

SPILBERG ShKALASI

Tashvish turi	Savollar	Ballar shkalasi	Tashvish turi	Savollar
Reaktiv tashvish (STAI-S)	Men tinchman.	1 — Hech qachon, 2 — Bir oz, 3 — O‘rtacha darajada, 4 — Juda ko‘p	Reaktiv tashvish (STAI-S)	Men tinchman.
Reaktiv tashvish (STAI-S)	Men xavotirdaman.	1 — Hech qachon, 2 — Bir oz, 3 — O‘rtacha darajada, 4 — Juda ko‘p	Reaktiv tashvish (STAI-S)	Men xavotirdaman.
Reaktiv tashvish (STAI-S)	Men ko‘proq xavotirlanganman.	1 — Hech qachon, 2 — Bir oz, 3 — O‘RXacha darajada, 4 — Juda ko‘p	Reaktiv tashvish (STAI-S)	Men ko‘proq xavotirlanganman.
Reaktiv tashvish (STAI-S)	Men o‘zimni xotirjam his qilmoqdaman.	1 — Hech qachon, 2 — Bir oz, 3 — O‘rtacha darajada,	Reaktiv tashvish (STAI-S)	Men o‘zimni xotirjam his qilmoqdaman.

		4 — Juda ko‘p		
Reaktiv tashvish (STAI-S)	Men zo‘riqib turibman.	1 — Hech qachon, 2 — Bir oz, 3 — O‘rtacha darajada, 4 — Juda ko‘p	Reaktiv tashvish (STAI-S)	Men zo‘riqib turibman.
Reaktiv tashvish (STAI-S)	Men juda xavotirdaman.	1 — Hech qachon, 2 — Bir oz, 3 — O‘rtacha darajada, 4 — Juda ko‘p	Reaktiv tashvish (STAI-S)	Men juda xavotirdaman.
Reaktiv tashvish (STAI-S)	Men o‘zimni tinch his qilmoqdaman.	1 — Hech qachon, 2 — Bir oz, 3 — O‘rtacha darajada, 4 — Juda ko‘p	Reaktiv tashvish (STAI-S)	Men o‘zimni tinch his qilmoqdaman.
Reaktiv tashvish (STAI-S)	Men asabiyman.	1 — Hech qachon, 2 — Bir oz, 3 — O‘rtacha darajada, 4 — Juda ko‘p	Reaktiv tashvish (STAI-S)	Men asabiyman.
Reaktiv tashvish (STAI-	Men juda tinchman.	1 — Hech qachon, 2 — Bir oz, 3 —	Reaktiv tashvish (STAI-	Men juda tinchman.

S)		O‘rtacha darajada, 4 — Juda ko‘p	S)	
Reaktiv tashvish (STAI-S)	Men o‘zimni xavotirsiz his qilmoqdaman.	1 — Hech qachon, 2 — Bir oz, 3 — O‘rtacha darajada, 4 — Juda ko‘p	Reaktiv tashvish (STAI-S)	Men o‘zimni xavotirsiz his qilmoqdaman.
Reaktiv tashvish (STAI-S)	Men o‘zimni hozir juda yaxshi his qilyapman.	1 — Hech qachon, 2 — Bir oz, 3 — O‘rtacha darajada, 4 — Juda ko‘p	Reaktiv tashvish (STAI-S)	Men o‘zimni hozir juda yaxshi his qilyapman.
Reaktiv tashvish (STAI-S)	Men xavotirlanganman.	1 — Hech qachon, 2 — Bir oz, 3 — O‘rtacha darajada, 4 — Juda ko‘p	Reaktiv tashvish (STAI-S)	Men xavotirlanganman.
Reaktiv tashvish (STAI-S)	Men o‘zimni xotirjam his qilmayman.	1 — Hech qachon, 2 — Bir oz, 3 — O‘rtacha darajada, 4 — Juda ko‘p	Reaktiv tashvish (STAI-S)	Men o‘zimni xotirjam his qilmayman.
Reaktiv tashvish	Men juda juda	1 — Hech qachon, 2	Reaktiv tashvish	Men juda juda
		ko‘p	tashvish darajasi	

H.M. Halimova, Y.R. Parpiyeva

KOGNITIV FUNKSIYALARNING GISTEREKTOMIYA VA OVARIEKTOMIYADAN KEYINGI ASPEKTLARI VA DAVOLASHNI MUQOBILLASHTIRISH

(Monografiya)

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