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O.V. Kravets, V.V. Yekhalov, V.V. Gorbuntsov
Dnipro State Medical University, Dnipro, Ukraine

# Anesthetic problems in concomitant systemic lupus erythematosus (literature review)

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**Abstract.** *Based on available literature sources, we have analyzed the impact of systemic lupus erythematosus (SLE) on organs and body systems, the main pathological aspects of the interaction of dosage forms used for its treatment with anesthetic drugs for the purpose of increasing the anesthetic safety of such patients during surgical interventions. Patients with SLE have different abnormalities of varying intensity. Numerous features of the course of the pathological condition create significant problems in the anesthetic provision of various surgical interventions. Anesthesia in emergency surgery in such patients can be difficult, since the time for developing perioperative strategies is limited. SLE is a very complex systemic autoimmune disease that presents major challenges in the surgical context; in the field of anesthesia, such patients should always be considered at higher risk. Given the heterogeneity of manifestations, it is difficult to establish unified management protocols, so the anesthesia plan must be appropriate for each specific case. Patients with SLE still have a higher incidence of postoperative complications and a 2- to 7-fold higher risk of in-hospital postoperative mortality compared to people without SLE. Anesthesiologists must be knowledgeable about the pathophysiology and potential organic lesions, which often affect airway patency, ventilation therapy, hemodynamic control, and renal function support. Therefore, multidisciplinary management and adequate preoperative assessment are essential for surgical risk stratification and readiness to avoid or treat serious complications such as bleeding, thrombotic, cardiovascular and other events early. A well-planned perioperative management and a skilled multidisciplinary team approach can contribute to a positive outcome of surgery and anesthesia in patients with SLE. Currently, there are no clear regulatory documents on anesthetic care for patients with concomitant SLE. This topic remains poorly studied, so there is an important problem in developing high-quality research that allow a better understanding of the anesthetic consequences of SLE with the creation of appropriate clinical protocols; facilitate the management of these patients; optimize the safety of anesthesia; reduce the rate of complications and associated mortality.*

**Keywords:** *antiphospholipid syndrome; drug interactions; regional and general anesthesia; systemic lupus erythematosus; review*

*Experience shows that lupus erythematosus may be accompanied by dangerous constitutional symptoms. Death may result from conditions resulting from the local disease.*
**M. Kaposi**

## Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease. Its variable course makes it difficult to standardize patient management [1]. The estimated prevalence of SLE worldwide is 100 patients per 100 thousand people. The disease can manifest at any age, although it primarily affects women of reproductive age. The ratio of female to male patients is 9 : 1. Blacks and Hispanics are affected 3 times more often than Europeans. Although the classic features of SLE have been described, there are plenty of clinical variations of the disease; for example, elderly patients tend to have a less severe form that involves fewer organ systems overall; men tend to experience less photosensitivity but have a higher mortality rate [2].

**Purpose.** Based on available literature sources, to analyze the impact of SLE on organs and body systems, the main pathological aspects of the interaction of dosage forms used for its treatment with anesthetic drugs in order to increase the anesthetic safety of such patients during surgical interventions.



Для кореспонденції: Єхалов Василь Віталійович, кандидат медичних наук, доцент, кафедра анестезіології, інтенсивної терапії та медицини невідкладних станів ФПО, Дніпровський державний медичний університет, вул. В. Вернадського, 9, м. Дніпро, 49044, Україна; e-mail: sesualiy@gmail.com; тел.: +380 (63) 276-64-35, +380 (50) 779-23-03
For correspondence: Vasyl Yekhalov, PhD in Medicine, Associate Professor, Department of Anesthesiology, Intensive Care and Emergency Medicine, Postgraduate Education Faculty, Dnipro State Medical University, Volodymyr Vernadsky st., 9, Dnipro, 49044, Ukraine; e-mail: sesualiy@gmail.com; phone: +380 (63) 276-64-35, +380 (50) 779-23-03
**Full list of authors' information is available at the end of the article.**

## Evidence synthesis

Patients with SLE have different anomalies of varying intensity [3]. Numerous features of the course of the pathological condition create significant problems of anesthetic support for various surgical interventions [2]. Preoperative examination aims to identify lupus activity, organ damage, drug effects, thorough preanesthetic assessment, laboratory results.

Management of high-risk patients requires a multidisciplinary approach [3]. As general measures, risk factors such as hypertension, hyperlipidemia, hyperhomocysteinemia should be carefully monitored and corrected; at that hypoalbuminemia, anemia, lymphopenia, and aspirin use are independent risk factors [4–6].

The mean modified comorbidity index for SLE is 0.49 ($p < 0.001$) [5]. Anesthesia care should consider:

— systemic organ involvement;

— thrombocytopenia;

— perioperative steroid replacement.

Laboratory examinations, including coagulation tests, platelet count, hemoglobin, and renal function, should be performed before anesthesia [7]. As SLE is a multisystem disorder, careful preanesthetic assessment is essential for safe anesthesia. The anesthetic plan should be individualized based on the degree of involvement of different organs and systems, side effects and competing drug effects, laboratory results [8].

Anesthesia in emergency surgery in such patients can be challenging since the time to develop perioperative strategies is limited [9].

Central and peripheral nervous system complications may occur in 37 to 95 % of patients with SLE. The American College of Rheumatology recommends the term "neuropsychiatric SLE" to encompass all possible manifestations which can range from headache, mild cognitive dysfunction, and mental confusion to cramps, cerebrovascular diseases, psychoses, demyelinating diseases, and states of coma [3, 8, 10–13]. Chorea may be the initial manifestation of SLE or antiphospholipid syndrome (APS); it is most commonly described in young adult women in association with other manifestations of SLE [14].

Neuropsychiatric manifestations of SLE are common, range from mild to severe, and are often difficult to diagnose and distinguish from those of other diseases [13]. Psychosis occurs in approximately 5 % of patients diagnosed with SLE; to alleviate the symptoms, it often requires lower doses of glucocorticoids, the addition of antipsychotics, pulse dose of cyclophosphamide [15].

Cotard's syndrome, also known as "zombie syndrome," is a clinical condition characterized by a fixed delusion that the person is already dead or dying, including relatives and acquaintances. It is a neuropsychiatric sign of brain pathology that affects the nondominant frontotemporal and parietal lobes, particularly the fusiform gyrus. The etiology of Cotard's syndrome may include structural changes associated with brain trauma, tumors, and SLE [16]. Electrophysiological studies have shown axonal neuropathy in 70 % of patients and evidence of demyelination in 20 % of people with severe SLE [12]. Fibromyalgia is common under SLE and occurs much more frequently than in the general population [17, 18].

Although thyroid disease can occur in lupus, thyrotoxicosis is rare. In a cohort study, the overall rate of hyperthyroidism in the SLE group was 6.4 %. Thyroid storm is an acute manifestation of thyrotoxicosis, often triggered by a physiologically stressful event (surgery). Thyrotoxic crisis often presents as a high-grade fever that requires emergency treatment, and in the absence of appropriate medical intervention, it can lead to cardiovascular collapse and death. These symptoms may be misdiagnosed in cases of high lupus activity, so the diagnosis is often missed. Fortunately, thyroid storm is uncommon in highly active SLE [19].

Cardiovascular risk is significantly increased under SLE, and systemic use of corticosteroids contributes to its elevation [20]. Myocarditis affects 5–10 % of the patients with SLE; of these, 80 % have a reduced ejection fraction. The process can progress to arrhythmias, dilated cardiomyopathy, and chronic heart failure, especially in the presence of hypertension, valvular disease, atherosclerosis, and renal failure, in particular during treatment with cyclophosphamide and hydroxychloroquine.

Cardiotoxicity is a recognized complication of high-dose cyclophosphamide therapy with acute decompensation and reversible decline in systolic function, although the rare cases have also been documented with hydroxychloroquine. However, cardiac dysfunction may be a consequence of myotoxicity, consistent with the more common neuromyotoxicity observed in case reports, where the predominant feature is proximal myopathy with or without peripheral neuropathy or cardiomyopathy. Based on the structural similarity of hydroxychloroquine to anesthetics, its potential to act through an anesthetic-like mechanism is currently being investigated [21].

Cardiac involvement under SLE is manifested by pericarditis and Libman-Sacks endocarditis. Coronary vasculitis and rapidly progressing atherosclerosis led to a high prevalence of ischemic heart disease in these patients. Rhythm and conduction disturbances are common. Patients are prone to potentially fatal intraoperative events such as intraoperative myocardial infarction [2, 3, 10–12, 21–24].

Pulmonary involvement can range from acute lupus pneumonitis, dry and exudative pleurisy to diffuse alveolar hemorrhage and interstitial lung disease [3, 11]. The late pulmonary sequela of SLE is diaphragmatic pathology. This complication is known as "shrinking lung syndrome", which is characterized by a decrease in total capacity, lung volume and frequent pneumonia (due to leukopenia) [2, 7]. Interstitial pneumonitis, alveolitis, alveolar wall damage, edema and hemorrhages are commonly observed in these patients. Immunoglobulin and complement deposition in the pulmonary vascular walls are induced. Chronic interstitial lung disease may occur in 50 % of cases and is characterized by interstitial lymphoid aggregates and fibrosis, thickening of the septum, hyperplasia of type 2 pneumocytes [23, 24].

SLE is characterized by the second highest prevalence of pulmonary hypertension [15]. Alveolar hemorrhage is a rare complication in these patients, in which pulmonary capillary wedge pressure, oxygenation, and airway pressure should be monitored; mechanical suction should be readily available [2].

Lupus nephritis is a glomerulonephritis, usually with proteinuria and erythrocyturia, and with the presence of erythrocyte casts in the urine [25]. People with SLE are at high risk of renal hypertension, which may increase if the patient is receiving more than 30 mg of prednisolone per day. Renal involvement is manifested by proteinuria and kidney failure. The general principle of protecting the kidneys should be followed, even in the absence of overt acute injury, when serum creatinine and urinalysis are normal. Drugs excreted by the kidneys include certain opioids, benzodiazepines, and neuromuscular blocking agents, which can accumulate in the body. Retained toxic metabolites result in prolonged sedation, paralysis, and delayed recovery from anesthesia. Important goals during anesthesia in patients with SLE are to avoid the use of nephrotoxic drugs, to maintain and control urine output [2, 3, 7, 8, 11].

Both azathioprine and methotrexate have the potential for hepatotoxicity, at that methotrexate tending to cause fibrosis and cirrhosis in severe cases. Pulmonary toxicity caused by methotrexate has also been documented, usually in the form of drug-induced pneumonitis with pulmonary infiltrates. Mycophenolate mofetil, a lymphocyte-selective immunosuppressant that acts by inhibiting purine synthesis, is a new drug that is increasingly being used for the treatment of lupus nephritis. It has a favorable toxicity profile compared with older drugs, but, like most other medicines, can cause clinically significant myelosuppression [21].

Women with SLE are at higher risk of complications during pregnancy [26] such as:

— spontaneous abortion;
— intrauterine growth retardation or fetal death;
— preeclampsia, eclampsia;
— preterm labor.

Steroids facilitate the synthesis and action of catecholamines, modulate the synthesis and response of β-receptors, and contribute to normal vascular tone and cardiac contractility [7]. Most patients with SLE are on long-term steroid therapy, which can lead to suppression of the hypothalamic-pituitary-adrenal axis when steroids are abruptly withdrawn. In these patients, abrupt withdrawal of glucocorticoids or the stress response associated with surgery may precipitate an Addisonian crisis [21]. The integrity of the hypothalamic-pituitary-adrenal axis can be assessed by plasma cortisol levels, the 250-μg ACTH stimulation test [3, 7, 8].

Preoperative evaluation should focus on optimizing glucocorticoid dosing by reducing the preoperative dose at most 20 mg/day whenever it is possible [27].

Patients with Cushingoid features who receive long-term high-dose steroid therapy are more likely to develop adrenal suppression [11]. To prevent it, adequate analgesia and corticosteroid therapy [3, 7, 8] should be provided during surgery, with additional doses of corticosteroids given to the patients who have been receiving them routinely [28, 29]. Long-term use of corticosteroids in patients with SLE often results in underdiagnosed and undertreated osteoporosis, leading to osteoporotic fractures. Other complications of long-term corticosteroid use include avascular necrosis, glaucoma, cataract, weight gain, and poor control of diabetes. High-dose steroid use may also be associated with opportunistic infections and acute psychosis; in addition, glucocorticoids cause hyperglycemia, hypercholesterolemia, osteoporosis, and hypertension [21].

Musculoskeletal involvement is the most common presentation (70–95 %) or even the first symptom in 50 % of patients with SLE and can range from mild arthralgias to deforming arthritis; of these, over 90 % have arthralgia or non-erosive symmetric inflammatory polyarthritis, which predominantly affects the small joints of the hands, knees, and wrists, although any joint may be involved. Jaccoud arthropathy results from laxity of the joint capsule and leads to non-erosive deformities of the hands, including elbow deviation and subluxation of the metacarpophalangeal joints, which may mime rheumatoid arthritis [23, 30, 31]. Long-term use of glucocorticoids for immunosuppression can cause osteoporosis, which is prevalent in up to 23 % of patients with SLE; osteoarthritis, avascular necrosis of the femoral head, and vertebral fractures have been observed. Careful intraoperative positioning is necessary to avoid stress on the joint, prevent osteoporotic bone fractures and peripheral nerve compression [3, 5, 21, 32].

Joint deformities make it difficult to palpate anatomical landmarks, assess vessels, and administer regional anesthesia [5, 32]. Limited cervical spine mobility complicates central venous access [32].

Esophageal symptoms are commonly reported, with 1–13 % of patients with SLE experiencing dysphagia, 11–50 % having heartburn.

Manometric studies have shown a frequent prevalence of peristaltic dysfunction, particularly in the upper third of the esophagus, which may explain some of the symptoms. However, no studies have shown abnormalities of the lower esophageal sphincter, and patients with SLE do not appear to be at increased risk for gastroesophageal reflux. Gastric disease secondary to SLE is controversial. Peptic ulceration or gastric perforation may occur as a consequence of using nonsteroidal anti-inflammatory drugs (NSAIDs) and corticosteroids rather than being directly caused by SLE [21]. Patients taking steroids and NSAIDs are at risk of gastrointestinal bleeding and should receive antacids [33].

Mycophenolate mofetil is an inhibitor of inosine monophosphate dehydrogenase and is also cytotoxic to T- and B-lymphocytes. This also has implications for anesthesia, as it results in anemia, leukopenia, and thrombocytopenia [34]. Nausea, vomiting, diarrhea, leukopenia, and septicemia are common when mycophenolate mofetil is combined with cyclosporine and corticosteroids.

Procainamide and hydralazine have the highest rates of causing and exacerbating SLE, with risks reaching 30 % for procainamide, 5–10 % for hydralazine.

All anti-TNF agents have been associated with SLE, at that the risk being higher with etanercept and infliximab; other drugs that have been associated with SLE include interferon-α, minocycline, isoniazid, rifampicin, phenytoin, penicillamine, quinidine, methyldopa, chlorpromazine, carbamazepine, ethosuximide, propylthiouracil, sulfasalazine [35].

Several other drugs have been implicated as possible causes of SLE: statins, antiarrhythmics, angiotensin-converting enzyme inhibitors, proton pump inhibitors, gold salts, NSAIDs, oral contraceptives [35].

Hematologic manifestations commonly seen under SLE include anemia, thrombocytopenia, leukopenia [3, 11].

Anemia is present in approximately half of patients with SLE, with anemia of chronic disease being the most common cause; however, other causes include autoimmune hemolytic anemia, iron deficiency anemia, anemia of chronic renal failure, complications of cyclophosphamide myelotoxicity. This anemia may be exacerbated by the dilutional anemia of pregnancy [5, 8].

Such patients often take medications such as anticoagulants, antiplatelet agents, disease-modifying agents, immunosuppressants. This regimen may increase the risk of postoperative complications, including bleeding, thrombosis, delayed healing, and postoperative infections.

There are currently no established guidelines for tooth extraction in individuals with APS associated with SLE [36].

Thrombocytopenia is commonly observed in patients with SLE [7, 11].

***Antiphospholipid syndrome*** (Hughes syndrome) may occur as a consequence of SLE and is clinically characterized by recurrent pregnancy loss and the presence of lupus anticoagulant (LA) antibodies, which may falsely prolong the activated partial thromboplastin time in such individuals [3, 37]. Antiphospholipid antibodies (aPL) are detected in one third of patients with systemic lupus erythematosus. The risk of thrombosis increases to 60– 70 % in the presence of aPL, decreases to 10–15 % in its absence.

LA production is not accompanied by bleeding, but paradoxical thrombosis [37]. The risks in patients with antiphospholipid antibody syndrome should be assessed before the surgery to reduce the possibility of massive bleeding and thromboembolic events [38]. LA, one of the diagnostic aPLs for APS, biochemically target epitopes of the negatively charged phospholipid-binding protein, leading to prolongation of phospholipid-dependent coagulation *in vitro*, as it is reflected in the results of tests such as activated partial thromboplastin time. However, a positive LA result is itself a risk factor for thrombosis, so patients with a positive LA should be suspected of having a hypercoagulable state, regardless of prolonged or normal activated partial thromboplastin time. Regardless of whether the patients receive antithrombotic therapy, careful anesthesia is mandatory for the patients with a positive LA to prevent thrombotic complications. In an aPL-positive patient, a provoking factor such as infection, trauma, and prolonged immobilization is needed to develop thrombosis; therefore, surgery itself may be the primary trigger of thrombosis [9]. Given the characteristic hypercoagulability seen in patients with APS, treatment focuses on preventing thrombosis. However, in such patients undergoing surgery, attention should be paid to the risk of not only thrombotic complications, but also perioperative bleeding [39, 40].

In cases of previous venous thrombosis, anticoagulant therapy is carried out with a target international normalized ratio (INR) of 2.0 to 3.0. Catastrophic APS is rare and accounts for approximately 1 % of all cases of antiphospholipid syndrome, but it has a mortality rate of 50 % [40]. The combination of glucocorticoids, heparin, and plasmapheresis is recommended as the first-line treatment instead of single-agent therapy. In refractory cases, rituximab may improve survival [40].

Direct oral anticoagulants include:

— direct thrombin inhibitors such as dabigatran etexilate;

— direct factor Xa inhibitors:

1) rivaroxaban;

2) apixaban;

3) edoxaban.

In contrast to warfarin, they have the advantages of predictable anticoagulant effects at a fixed dose without the need for blood level monitoring, and few drug-food interactions, making them acceptable for patients with APS. In the preoperative period, warfarin discontinuation should be extended from 5 to 7 days in patients receiving highly intensive anticoagulation therapy with a target INR no less than 3.0 [40]. Preoperative safe INR is usually corrected to no less than 1.5. The incidence of bleeding events has been reported to be significantly lower in patients with a preoperatively corrected INR to no less than 1.5 than in patients with an INR over 1.5. It is important to avoid rapid and excessive correction, as the former causes immediate thrombosis and the latter makes it difficult to restore anticoagulation to the therapeutic range after surgery and increases the risk of postoperative thrombotic complications. Low-dose oral vitamin K (1–2 mg) is generally recommended. Even if the INR is no less than 3.0, slow correction with low-dose vitamin K or slow infusion of fresh frozen plasma is safer than rapid correction before emergency surgery [40].

In the perioperative period, not only the risk of thrombosis but also of bleeding increases. Therefore, among perioperative considerations, appropriate discontinuation of anticoagulants and switching anticoagulant therapy are important to prevent possible bleeding while reducing the risk of thrombosis. In addition to pharmacological interventions, the continuous use of physical preventive methods in the perioperative period is quite important, and optimal anesthesia management and coagulation monitoring should be carried out according to the state of the blood coagulation system [40].

The presence of LA is associated with prolonged partial thromboplastin time, but the risk of bleeding is quite rare, so, according to some authors, regional anesthesia can be practiced safely. Local anesthetic techniques are contraindicated in targeted anticoagulant therapy and priority administration of anticoagulants after surgery [9, 11, 21].

As long as the coagulation profile is not disturbed after preoperative anticoagulant therapy for an appropriate period, neuraxial anesthesia can be safely performed in APS, except in cases where massive blood transfusion is planned or in patients who require emergency surgery immediately after heparin administration [40]. Obstetric and cardiac anesthesia, especially in those with aPLs or APS, requires multidisciplinary management in a specialized center [21].

***Neuraxial anesthesia*** can be safely performed in pregnant women with platelet counts of 80–100 thousand/mm$^3$ [40]. The ASA II Task Force recommends platelet transfusion if the platelet count is over 20 thousand/mm$^3$ and there is clinical evidence of bleeding [7, 11].

Anesthesiologists should carefully consider the choice of anesthetic technique, ensuring a balance between the appropriate risks and benefits, and always be prepared for dangerous situations [41].

Researchers have concluded that continuous epidural anesthesia is safer than general anesthesia and should be performed to the VI thoracic dermatome to provide adequate analgesia with hemodynamic stability, or a low-dose combined spinal-epidural anesthesia should be used [26, 41, 42]. If an epidural catheter is left *in situ* after surgery, it should be for the shortest possible time [31]. Systemic diseases that can damage the nervous system are easily underestimated when neurological disorders occur after epidural anesthesia [41].

Regional anesthesia decreases sympathetic nervous system activity and provides adequate pain control through sensory blockade, thereby reducing the adverse effects on the cardiovascular system. However, a single spinal anesthesia is contraindicated since it can rouse vasodilation, causing marked hypotension. This results in a significant decrease in systemic vascular resistance. Since vasodilation must be controlled immediately after the administration of local anesthetics, vasopressor agents such as phenylephrine or noradrenaline should be prepared in advance [42].

Appropriate anticoagulant treatment is also necessary for pregnant women who will receive epidural or neuraxial anesthesia. Regarding needle/catheter placement for neuraxial blockade, the 2018 American Society of Regional Anesthesia and Pain Medicine (ASRA) guidelines suggest no restrictions when patients receive low-dose aspirin alone. Needle/catheter placement should be performed at least [43]:

— 4 to 5 hours after unfractionated heparin;

— 12 and 24 hours after the last bolus for low- and high-dose low-molecular-weight heparin, respectively.

The combined use of low-dose aspirin with heparin or another antiplatelet agent affecting coagulation mechanisms requires caution due to the risk of bleeding complications or spinal hematoma formation [40, 43].

Catheter removal is recommended 1 hour before re-administration of unfractionated heparin or 4 hours before re-administration of low-molecular-weight heparin according to the ASRA guidelines [40, 43].

If patients with APS develop sudden hypotension, fever, or back pain in the postoperative period, adrenal infarction or bleeding should be suspected [40].

During an acute episode of SLE, it is reasonable to postpone selective surgery until recovery from the flare-up [21].

Active preoperative albumin elevation may be of greater benefit to the postoperative recovery of patients with SLE [44].

Antibiotic prophylaxis should be mandatory when patients are receiving steroids and immunosuppressive agents [3, 20, 21, 31, 40, 44].

Anticoagulant prophylaxis is indicated in patients at high risk of thromboembolism (antiphospholipid syndrome) [9, 11, 21].

The anesthetic plan should be individualized, based on the degree of involvement of various systems, the patient's medication regimen, and laboratory results [3].

***General anesthesia*** is preferred in critically ill patients requiring airway control. Intraoperative transesophageal echocardiographic monitoring is advisable whenever possible. This is because the sudden change in hemodynamics that can occur immediately after delivery can lead to rapid decompensation. In addition, the obvious advantages of general anesthesia are: first, the ability to control ventilation (avoiding hypoxia or hypercarbia), second, the lack of influence on perioperative anticoagulant therapy.

General anesthesia is reserved for patients having contraindications to neuraxial anesthesia or for urgent cesarean section [45, 46].

The anesthesiologist's primary concern is to avoid hypoxia at all stages of the procedure [28]. Airway manipulations require special care because of the potential for laryngeal edema, vocal cord paralysis, cervical spine arthritis, atlanto-occipital subluxation, which can lead to unexpected airway obstruction [3, 7, 8, 10, 11, 28].

The goals of anesthesia are:

— to create a safe airway while maintaining cervical spine integrity (through careful positioning and manipulation);

— to successfully treat any systemic disease;

— to prevent progression of SLE complications.

The anesthesiologist should be aware that the standard position for endotracheal intubation during direct laryngoscopy involves hyperextension of the neck, which may result in neurological damage [32]. Patients with SLE may suffer from cervical spine fusion and severe osteochondrosis/osteoporosis, which increases the risk of spinal dislocation or fracture during endotracheal intubation [27].

Laryngeal complications of SLE are potentially fatal; they have been reported under SLE with a frequency ranging from 0.3–0.5 to 30 % for over 50 years. Secondary upper airway obstruction due to cricoarytenoid arthritis has also been observed [21, 31]. Laryngeal involvement can range from mild inflammation to laryngeal and epiglottic edema and right up to acute airway obstruction. Left-sided vocal cord paralysis in SLE patients is thought to be secondary to neural vasculitis, and an association with pulmonary hypertension has been reported, probably due to right atrial/pulmonary artery dilatation, which causes compression of the recurrent laryngeal nerve [3, 8]. The pathophysiology of laryngitis under SLE is poorly understood, although tissue deposition of immune complexes with complement activation is a less likely cause. Facial skin lesions, oral or nasopharyngeal ulcers, hemorrhages, and gingivitis can interfere with face mask ventilation and oral manipulation. Epiglottitis and rheumatoid nodules have also been described. Most cases of SLE respond to immunosuppressive therapy, although emergency endotracheal intubation or surgical tracheostomy is rarely required in the postoperative period [2, 3, 8, 28]. Endotracheal intubation may be complicated by cricoarytenoid arthritis and tracheal stenosis. Cricoarytenoid arthritis is accompanied by hoarseness, stridor, and a foreign body sensation; if any of these symptoms occur, the larynx should be examined directly. Given the potential for laryngeal and tracheal involvement, all patients with SLE should be expected to have difficulty in the airway. Endotracheal intubation should be avoided where possible; if it is not possible, a laryngeal mask airway or the smallest possible endotracheal tube should be used. In these patients, awake endotracheal intubation under fiberoptic bronchoscopy is sometimes recommended, although surgical tracheostomy under local anesthesia is an alternative. Active SLE may also contribute

to the development of post-intubation subligamentous stenosis even after short-term intubation, which in extreme cases may also require tracheotomy [2, 3, 7, 10, 11, 21, 28, 32, 45, 47].

Complications of endotracheal tube insertion and positive pressure ventilation include increased intrathoracic pressure and pulmonary vascular resistance. This mainly occurs during intubation and extubation, when sympathetic nerve stimulation is greatest [42]. Careful monitoring for edema-induced airway obstruction is necessary during recovery from extubation [32].

Arterial cannulation for blood gas analysis and placement of a pulmonary artery catheter to assess pulmonary hypertension may be indicated [2].

Long-term use of hydroxychloroquine can sometimes lead to maculopathy and retinopathy, which are irreversible, and careful ophthalmological examination is recommended. Patients with SLE are immunocompromised and are at significantly higher risk of infections, which are important causes of morbidity and mortality. Most of these patients are on long-term steroids and other immunosuppressants such as rituximab, which should be continued before surgery [1, 3, 7, 23, 24, 48].

The problems of anesthesia in patients with SLE arise from the nature of the disease itself and the interaction of anesthetics and muscle relaxants with anticholinesterase drugs used in routine therapy [49]; pharmacological interactions between anesthetics and immunosuppressants must be considered [10].

Cyclosporine is a calcineurin inhibitor that suppresses the early phase of T-cell activation without affecting suppressor T-cells or antibody-mediated immunity. Perioperative considerations include several drug interactions. The duration of action of nondepolarizing muscle relaxants may be prolonged, and systemic lidocaine may result in decreased clearance of myoplegic drugs [50].

Tacrolimus, a potent calcineurin inhibitor, is not officially approved for use in SLE, but some experts recognize its effectiveness in kidney damage, especially in combination with mycophenolate mofetil. Tacrolimus inhibits the first phase of T-cell activation and may suppress humoral immunity. It is a substrate of cytochrome P450 CYP3A4, so concomitant use of opioids (buprenorphine, fentanyl, tramadol, methadone), barbiturates, benzodiazepines, dexamethasone, lidocaine may increase tacrolimus toxicity. Tacrolimus prolongs the QT interval, so halogenated anesthetics and ondansetron should be used with caution due to the risk of arrhythmias. As a potent immunosuppressant, it causes an increased risk of infection [50].

Voclosporin is an immunosuppressant, a calcineurin inhibitor, approved for the treatment of lupus nephritis. Potential adverse effects associated with the postoperative period include hypertension, increased risk of infection, neurotoxic effects, acute kidney injury. Voclosporin prolongs the QT interval, so the same considerations as for tacrolimus should be followed [50].

The anesthesiologist should be particularly vigilant in people receiving mycophenolate, azathioprine, cyclosporine, and tacrolimus; in patients with mild SLE, they should be discontinued 1 week before surgery and resumed approximately 3 to 5 days after it, unless there is evidence of local or systemic infectious complications [27, 50].

Azathioprine, an antimetabolite immunosuppressant, may interact with muscle relaxants, reducing their effect through interaction with phosphodiesterases and increased presynaptic acetylcholine release. One study required an increase in dose, for atracurium — 37 %, vecuronium — 20 %, pancuronium — 45 %. In case of pancuronium and vecuronium, the possibility of increasing doses was negated by the presence of renal failure [49].

In patients treated with cyclophosphamide, prolonged apnea may occur after succinylcholine administration due to its cholinesterase inhibitory effect. Normally, 90–95 % of succinylcholine is metabolized in the blood by plasma cholinesterase, leaving only 5–10 % to reach the neuromuscular junction. Thus, a deficiency of plasma cholinesterase will result in a relative overdose, which may even cause a double neuromuscular block. Many other drugs hydrolyzed in the circulation by plasma cholinesterase have been found to prolong the action of succinylcholine to varying degrees. These interactions should be anticipated, especially in short-term procedures, and it would therefore be advisable to avoid succinylcholine, if possible, for at least 3 weeks after high-dose cyclophosphamide chemotherapy [3, 11 ,21, 50, 51].

The concomitant use of NSAIDs with methotrexate has known adverse effects described in several reports of acute renal failure and pancytopenia. In addition, administration of the drug against the background of nitrous oxide (currently not used in Ukraine) may lead to side effects of methotrexate and suppression of the hematopoietic function of the bone marrow [2, 10, 31, 32].

Halothane and methoxyflurane are contraindicated under SLE [28]. Concomitant use of halothane and cyclophosphamide has been associated with increased toxicity and mortality in both animal and clinical studies [52].

Propofol-induced exacerbation of SLE has been reported in several cases. This side effect may be related to the fact that propofol increases the number of T-helper cells and induces B-cell differentiation and plasmablast formation, which further promotes the secretion of autoantibodies, especially in patients with positive tests for anti-DNA and antinuclear antibodies, although there are still insufficient studies to demonstrate the clinical significance of this. Thus, propofol may aggravate the symptoms of SLE in some patients, highlighting the importance of identifying potential immune-related factors before using it, especially in people with autoimmune diseases [29, 31, 53]. Cardiotoxicity of anthracyclines and monoclonal antibodies such as trastuzumab can cause fatal arrhythmias if QT-prolonging drugs (propofol) or β2 agonists are administered [52, 54].

Finally, angioedema secondary to drug interactions, esterase deficiency, or hypocomplementemia may occur; the most common causes are NSAIDs, angiotensin-converting enzyme inhibitors [31].

NSAIDs increase the risk of allergic reactions and aseptic meningitis; in the presence of lupus nephritis, they increase the risk of acute kidney injury and death when used in patients with end-stage CKD [20].

For intravenous induction of anesthesia, the following preparations are recommended:

— etomidate — to maintain systemic vascular resistance and minimize hypotension;

— fentanyl — to prevent adverse hemodynamic reactions to laryngoscopy, intubation, and intraoperative pain.

Isoflurane maintains pulmonary and systemic pressure within the preoperative range provided nitroglycerin and dobutamine are readily available [46]. Barbiturates should be used with caution. Depolarizing muscle relaxants have been shown to be superior to nondepolarizing ones [28].

During surgery, normothermia should be maintained, fluids should be given, and exposed areas should be covered, as patients with SLE are more prone to Raynaud's phenomenon [7, 11, 40].

Monitoring for SLE during anesthesia includes five-lead ECG, noninvasive blood pressure measurement, pulse oximetry. Invasive monitoring should be used in patients with myocarditis, valvular disease, or intracardiac conduction abnormalities if hemodynamic stability is adequately maintained [2, 3, 10, 11, 21–24].

Intraoperative coagulation monitoring methods include activated clotting time, measurement of heparin concentration by protamine titration, measurement of viscoelasticity by thromboelastography (or rotational thromboelastometry) [40].

Since the symptoms of SLE are nonspecific, laboratory tests become the mainstay of monitoring:

— all patients should have a complete blood count with coagulation profile;

— platelet count should be repeated monthly because of the high risk of thrombocytopenia;

— electrocardiography is advisable in cases of suspected pericarditis and myocarditis;

— chest X-ray is performed urgently in extreme cases when pleural effusion or interstitial pneumonitis is clinically observed;

— creatinine clearance and 24-hour urinary protein should be monitored monthly for patients with renal involvement.

Anticardiolipin antibodies, LA, anti-β2 glycoprotein should be tested to rule out any secondary lesions in the following months [3, 8].

If the patient is taking steroids, careful monitoring of blood glucose levels is recommended. Hydroxychloroquine has been shown to cause severe hypoglycemia, including loss of consciousness, which can be life-threatening in patients treated with or without antidiabetic medications. Patients receiving hydroxychloroquine should be warned about the risk of hypoglycemia and the associated clinical signs and symptoms. In patients with clinical symptoms suggestive of hypoglycemia, blood glucose levels should be monitored during hydroxychloroquine treatment, and therapy should be reviewed as necessary [33].

Patients with SLE still have a higher incidence of postoperative complications and a 2- to 7-fold higher risk of in-hospital postoperative mortality compared to patients without SLE [4, 5].

SLE is a very complex systemic autoimmune disease that presents major challenges in the surgical context; in the field of anesthesia, such patients should always be considered at higher risk. Given the heterogeneity of manifestations, it is difficult to establish unified management protocols, so the anesthetic plan must be appropriate for each specific case. Anesthesiologists must be knowledgeable about the pathophysiology and potential organic lesions, which often affect airway patency, ventilation therapy, hemodynamic control, and renal function support. Therefore, multidisciplinary management and adequate preoperative assessment are essential for surgical risk stratification and readiness to avoid or treat serious complications such as bleeding, thrombotic, cardiovascular and other events early [31, 45]. A well-planned perioperative management and a skilled multidisciplinary team approach can contribute to a positive outcome of surgery and anesthesia in patients with SLE [11].

Currently, there are no clear regulatory documents on anesthetic care for patients with concomitant SLE. This topic remains poorly studied, so there is an important problem of developing high-quality research that:

— allow a better understanding of the anesthetic consequences of SLE with the creation of appropriate clinical protocols;

— facilitate the management of these patients;

— optimize the safety of anesthesia;

— reduce the rate of complications and associated mortality.

## Conclusions

1. Systemic lupus erythematosus is a chronic autoimmune disease that has a variable course, which complicates the standardization of patient treatment.

2. SLE causes damage to many organs and systems of the body, which makes it difficult to provide anesthesia for surgical interventions in such patients.

3. Drugs used in the planned treatment of SLE can interact with anesthetics, hypnotics and muscle relaxants, distorting the effect of anesthetic drugs and causing a negative effect on the body as a whole.

4. The development of appropriate recommendations and clinical protocols will increase the perioperative safety of patients with SLE.

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**Information about authors**

Olha Kravets, MD, DSc, PhD, Professor, Head of the Department of Anesthesiology, Intensive Care and Emergency Medicine, Postgraduate Education Faculty, Dnipro State Medical University, Dnipro, Ukraine; e-mail: 535951@ukr.net, 602@dmu.edu.ua; https://orcid.org/0000-0003-1340-3290

Vasyl Yekhalov, PhD in Medicine, Associate Professor, Department of Anesthesiology, Intensive Care and Emergency Medicine, Postgraduate Education Faculty, Dnipro State Medical University, Dnipro, Ukraine; e-mail: sesualiy@gmail.com; phone: +380 (63) 276-64-35, +380 (50) 779-23-03; https://orcid.org/0000-0001-5373-3820

Viacheslav Gorbuntsov, MD, DSc, PhD, Professor, Department of Skin and Venereal Diseases, International Faculty, Dnipro State Medical University, Dnipro, Ukraine; e-mail: gorbuntsovvv@gmail.com; https://orcid.org/0000-0001-7752-0993

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*Кравець О.В., Єхалов В.В., Горбунцов В.В.*
*Дніпровський державний медичний університет, м. Дніпро, Україна*

## Анестезіологічні проблеми при супутньому системному червоному вовчаку (літературний огляд)

**Резюме.** На основі доступних літературних джерел проаналізовано вплив системного червоного вовчака (СЧВ) на органи та системи організму, основні патологічні аспекти взаємодії лікарських форм, що застосовуються для його лікування, з анестетиками з метою підвищення анестезіологічної безпеки в таких пацієнтів під час хірургічних втручань. В осіб із СЧВ можуть спостерігатися різні порушення, що відрізняються за тяжкістю. Численні особливості перебігу хвороби створюють значні проблеми в анестезіологічному забезпеченні операцій. Анестезія в невідкладній хірургії може бути непростою, оскільки час на розробку періопераційних стратегій обмежений. СЧВ є дуже складним системним автоімунним захворюванням, що створює труднощі в хірургічному контексті; таких пацієнтів завжди слід вважати групою підвищеного ризику при виконанні анестезії. З огляду на неоднорідність проявів важко встановити уніфіковані протоколи ведення, тому план анестезії має відповідати кожному випадку. У пацієнтів із СЧВ досі спостері-

гається більша частота післяопераційних ускладнень і у 2–7 разів вищий ризик внутрішньолікарняної післяопераційної смертності порівняно з особами без цього захворювання. Анестезіологи повинні мати знання про патофізіологію та можливі органічні ураження, які часто впливають на прохідність дихальних шляхів, вентиляційну терапію, гемодинамічний контроль і підтримку функції нирок. Тому багатосторонній підхід й адекватна передопераційна оцінка є важливими для стратифікації хірургічних ризиків і готовності до запобігання серйозним ускладненням, як-от кровотеча, тромботичні, серцево-судинні та інші події, або їхнього раннього лікування. Добре спланований періопераційний менеджмент та кваліфікований підхід міждисциплінарної команди сприятимуть позитивному результату хірургічного втручання й анестезії в пацієнтів із СЧВ. На сьогодні немає чітких регламентуючих документів щодо анестезіологічного забезпечення осіб із супутнім СЧВ. Ця тема залишається недостатньо вивченою, тому існує важлива проблема розробки якісних досліджень, що дозволять краще зрозуміти анестезіологічні наслідки СЧВ зі створенням відповідних клінічних протоколів, полегшити ведення таких пацієнтів, оптимізувати безпеку анестезії, знизити рівень ускладнень і пов'язану з ними смертність.

**Ключові слова:** антифосфоліпідний синдром; взаємодія між фармакологічними засобами; регіонарна та загальна анестезія; системний червоний вовчак; огляд