Muscle examination may reveal weakness from myositis, neuropathy (in vasculitis and SLE), myopathy (in steroid myopathy), or synovitis (in rheumatoid arthritis, SLE, and spondyloarthropathy). A complete neurologic examination may reveal carpal tunnel syndrome, peripheral neuropathy such as mononeuritis multiplex (i.e., asymmetrical sensory or motor neuropathy seen in many vasculitides), and central nervous system disease (in SLE and vasculitis). Recurrent miscarriages, livedo reticularis, Raynaud's phenomenon, and recurrent thrombotic events indicate antiphospholipid antibody syndrome (in primary or secondary SLE).

The initial evaluation must determine whether diagnosis and treatment of the patient's problem requires urgent attention. Infectious processes need immediate treatment. Acute joint inflammation, fever, and systemic signs such as chills, night sweats, and leukocytosis provide supporting evidence for infection. Gouty arthritis may share some or all of these clinical features, but its onset tends to be more abrupt. Inflammation extending beyond the margins of the joint is characteristic of septic arthritis and is otherwise seen only in crystal disease and rheumatoid arthritis. Nonarticular processes such as cellulitis, septic bursitis, tenosynovitis, and phlebitis may mimic infectious arthritis. Analysis of synovial fluid is the key to diagnosis.

Acute nerve entrapment or spinal cord compression, tendon rupture, and fractures may occur in the absence of obvious trauma. Spinal cord compression may be the result of a herniated disk or vertebral subluxation. Tendon rupture may occur in inflammatory arthritides, particularly in the wrist of patients with rheumatoid arthritis. Pelvic and other insufficiency fractures may be seen in patients with osteoporosis or osteomalacia. Careful musculoskeletal and neurologic examinations help in the detection of these disorders, all of which require urgent treatment.

The onset of systemic rheumatic diseases is usually insidious, and the clinical course is prolonged. Treatment is usually not urgent and can be safely deferred, particularly if the diagnosis is uncertain. However, potential threats to life or the possibility of serious and irreversible organ damage may require urgent therapy. Patients with SLE or systemic vasculitis may have central or peripheral nervous system disease, including brain and peripheral nerve infarcts; glomerulonephritis; inflammatory or hemorrhagic lung disease; coronary artery involvement; intestinal infarcts; and digital infarcts. Threatened digit loss may also be seen in cases of scleroderma and Raynaud's disease. Renal crisis may occur in scleroderma, with vasculopathy leading to renal infarcts, azotemia, microangiopathy, and severe hypertension. Acute blindness is a potential complication of giant cell arteritis, and the diagnosis requires urgent therapy even before confirmatory biopsy.

Acute inflammatory myositis should be promptly treated because it may progress rapidly and involve the respiratory musculature. In some cases, major organ involvement may be occult. When systemic disease is suggested, the patient's lungs and kidneys should be carefully evaluated.

LABORATORY TESTING

Synovial fluid analysis is an important part of the evaluation of arthritis (Table 76-3). It helps to distinguish between inflammatory and noninflammatory arthritis, and results can be diagnostic of infectious arthritis or crystal disease.

Synovial fluid consists of an ultrafiltrate of plasma plus hyaluronic acid that is secreted by synovial lining cells. Evaluation of synovial fluid should include a cell count and differential, examination for sodium urate and calcium phosphate dehydrate crystals, Gram stain, and culture. Synovial fluid glucose and protein levels are not useful tests. Synovial fluid examination should be performed for all acute arthritides and all situations in which joint infection is likely. It should be performed at least once to evaluate chronic inflammatory arthritis. Aspiration and analysis of fluid before therapy are essential for appropriate decision making.

Although autoantibodies are often considered the hallmark of rheumatic diseases, their utility in diagnosing individual patients is much less than commonly assumed. Although almost 95% of patients with SLE have antinuclear antibodies (ANAs), as do most patients with scleroderma and autoimmune myositis, the proportion of patients with other rheumatic diseases who have positive test results is much lower. Conversely, 15% to 25% of healthy persons have ANAs, sometimes in high titers, when commercial test kits are used. Older persons and patients with nonrheumatic systemic diseases such as malignancies and nonrheumatic autoimmune diseases such as thyroiditis or hypothyroidism have even higher frequencies of ANAs.

The very low specificity of a positive ANA result in the absence of clinical findings for an autoimmune disorder precludes its use as a screening test for disease in the general population. Other autoantibodies may be more useful and are discussed in subsequent chapters.

TABLE 76-3 CLASSIFICATION OF SYNOVIAL EFFUSIONS BY SYNOVIAL WHITE BLOOD CELL COUNT

GROUP	SAMPLE DIAGNOSES	APPEARANCE	SYNOVIAL FLUID WBC COUNT (MM^3)*	PMN CELLS (%)
Normal		Clear, pale yellow	0-200	<10
I. Noninflammatory	Osteoarthritis; trauma	Clear to slightly turbid	50-2000 (600)	<30
II. Mildly inflammatory	Systemic lupus erythematosus	Clear to slightly turbid	100-9000 (3000)	<20
III. Severely inflammatory (noninfectious)	Gout	Turbid	2000-160,000 (21,000)	≈70
	Pseudogout	Turbid	500-75,000 (14,000)	≈70
	Rheumatoid arthritis	Turbid	2000-80,000 (19,000)	≈70
IV. Severely inflammatory (infectious)	Bacterial infections	Very turbid	5000-250,000 (80,000)	≈90
	Tuberculosis	Turbid	2500-100,000 (20,000)	≈60

PMNs, Neutrophils; WBC, white blood cell.
*Range, with mean values in parentheses.