Chapter 9
Postanesthesia and Postoperative Care

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DEFINITIONS

Hospital-acquired pneumonia (HAP)—Pneumonia that develops 48 hours or more after hospital admission because of organisms that were not incubating at the time of admission.

Hypoxic pulmonary vasoconstriction—Local reflex in the lung that diverts blood away from poorly oxygenated regions.

Low molecular weight heparin (LMWH)—Product that acts by inhibiting factor Xa.

Tissue plasminogen activator (t-PA)—Compound used for nonsurgical thrombolysis.

Venous thrombotic event—A venous thromboembolism that includes vascular clotting, such as deep vein thrombosis, and pulmonary embolism.

Virchow triad—Factors that increase the risk of vascular thromboembolism, for example, hypercoagulability, stasis, trauma to vessels.

V/Q abnormalities—Ventilation/perfusion abnormalities, which can lead to mismatching of pulmonary blood flow and ventilation.

Postoperative complications are the most important factors in defining the outcome of the first 72 hours following a patient's surgical procedure. It is critical to monitor basic physiologic parameters—such as renal, cardiovascular, and respiratory functions—and laboratory tests to optimize and sustain recovery from surgery and anesthesia.

Postoperative morbidity can be minimized by an appropriate preoperative assessment of the surgical patient. This should include emphasis on identifying the patient at risk for venous thromboembolic complications and administering prophylactic anticoagulation. Optimized nutritional status and support has also been shown to improve wound healing and decrease the postoperative recovery time and length of hospital stay.

POSTOPERATIVE VASCULAR COMPLICATIONS

About 3 million venous thrombotic events, or venous thromboembolisms (VTEs), occur in the United States each year. About 2,000,000 plus of these events are deep vein thrombosis (DVT) in the hospital, and as many as 600,000 are pulmonary embolisms (PEs). Twenty-five percent of PEs are fatal, and VTE has been recognized as the biggest preventable cause of morbidity and mortality in United States hospitals. Ten percent of hospital deaths in the US are due to PE, and some patient groups, especially gynecologic malignancy patients, have a higher-than-usual risk for VTE, with a general incidence of about 15% to 20%. The risk of fatal postoperative PE among these patients is closer to 40% with no prophylaxis. More than 90% of PE patients have a lower or upper extremity DVT concomitantly.

PE has few defining characteristics, but the onset of respiratory distress compounded by hypotension, chest pain, and cardiac arrhythmias can be harbingers of impending death and are complications that convert an otherwise successful surgery into a postoperative fatality. Only 70% of patients who die of a PE have it considered in their differential diagnosis.

Modern diagnostic studies have provided more accurate information about the frequency of vascular complications and can identify those patients at risk of an embolic event. Pre- and postoperative prophylaxis with heparin or low molecular weight heparins (LMWHs) and concomitant use of embolic stockings and intermittent pneumatic compression (IPC) devices have significantly reduced the risk of VTE in the moderate- and high-risk patients. Clarke-Pearson and colleagues, using univariate and regression analysis, designed a prognostic model to evaluate the risk of postoperative VTE for an individual patient. In a group of 411 gynecology patients, the prognostic factors they identified included type of surgery, age, leg edema, non-Caucasian ethnicity, severity of varicose veins, previous radiotherapy, and a prior history of DVT.

More than 130 years ago, Rudolph Virchow conceptualized the factors leading to postoperative thrombosis. These included venous stasis, changes in the blood constituents, and impaired function of the vessel wall. The blood clotting process is complicated (Fig. 9.1), but it is initiated by the actions of tissue factor (TF) on factor VIIa after injury to vessels exposes the subendothelium and promotes platelet adhesion and aggregation to form a primary platelet plug. The process is completed by the actions of multiple components and factors in the blood that generate thrombin, the potent rate-regulating enzyme, which then interacts with fibrinogen and factor XIII to form an insoluble clot (Fig. 9.2). Much recent evidence has focused on the role of cell-derived circulating microparticles (MPs) as potent etiologic agents for enhanced risk of VTE. This is because these MPs carry TF and other procoagulant phospholipids on their surfaces that potentiate activation of the coagulation pathways and may promote angiogenesis.

When patients have cancer or sustain venous damage from the surgical procedure, such as occurs with skeletonization of the pelvic vasculature, the up-regulation of thrombin generation has more profound effects. TF, fibrin, and thrombin

all have angiogenic properties that can interfere with tissue structural properties by degrading matrix metalloproteinases, promoting cell migration, and enhancing metastasis. Tumors also up-regulate the production of TF and its inclusion into MPs as well as plasminogen activator inhibitor-1 (PAI-1), thus promoting the generation of procoagulant activity. This multifactorial derived activity helps to explain the high incidence of VTE in the gynecologic cancer patient.

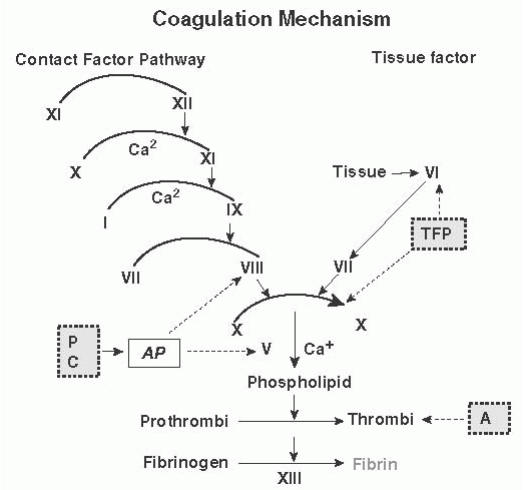

FIGURE 9.1 Formation of venous thrombus following various surgical procedures with the activation of clotting factors and aggregation of platelets. AP, activated protein, PC,

protein C, TFP, tissue factor peptide.

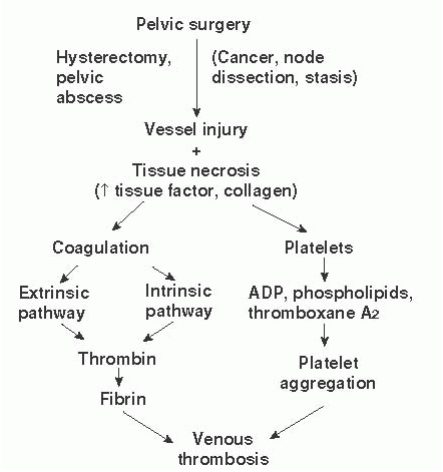

FIGURE 9.2 Schematic representation of the cascade clotting mechanism, illustrating the role of extrinsic and intrinsic factors. Increases in tissue thromboplastin-like substance and collagen-activated factor XII initiate the formation of fibrin through the extrinsic and intrinsic pathways, principally by the activation of factor X. ADP, adenosine diphosphate.

These tumorigenic effects on coagulation occur in addition to the typical acute postsurgical reactions seen in many hemostasis proteins. These include increases in fibrinogen, factor V, factor VIII, and von Willebrand factor, which promotes platelet adhesion and function. There is usually an increase in platelet number in the postoperative period, and the normal fibrinolytic response is blunted by the increase in PAI-1 and thrombin-activatable fibrinolysis inhibitor. Essentially, the fibrinolytic system is nonfunctional for several days following surgery, which down-regulates the ability of plasmin to impede wound healing by preventing degradation of fibrin and other matrix proteins.

Venous stasis is thought to be the cornerstone of postoperative thrombosis. Venous stasis in the pelvis and lower extremities results in platelet activation, promoting the adhesion of platelets to the endothelial cells lining the vessel, which are already stressed in a procoagulant mode. This results in conditions that encourage the development of a thrombus. These physiologic changes in venous hemodynamics occur in the pre-, peri-, and postoperative periods. Doran has shown that venous return from the lower extremities is decreased by half during surgical procedures because of the impact of muscle relaxation from anesthetic agents. Scanning using I^{125} fibrinogen has demonstrated that venous thrombosis is initiated during the surgery in 50% of patients who subsequently manifest a DVT. Lower extremity blood flow has been shown to decrease to about 75% of the normal drainage flow in the immediate postoperative period. This is an important reflection of Virchow triad on the role of adequate vessel flow. This reduction in flow persists for about 14 days after surgery because of the loss of muscle-pumping function in the legs. The major site of thrombus formation is the soleal venous sinuses of the calf, a portion of the venous arcade that joins the posterior tibial and peroneal veins draining the soleal muscle. Thrombi from these sinuses often occur posterior to valves located at the junction where these sinuses drain into the collecting veins. Thrombi often occur in these sinuses and in valve cusps in bedridden patients.

Another contributing factor to venous stasis during prolonged surgery is the use of tight packing of the intestines in the upper abdomen with obstruction of the underlying vena cava. The type and length of operation are directly related to the incidence of postoperative VTE, as outlined in **Table 9.1**.

Diagnosis of Venous Thromboembolism

The traditional clinical methods used to diagnose venous thrombosis of the lower extremities are of limited value, with error rates approaching 50% for both false-negative and falsepositive rates. Most of the diagnostics problems occur because of the insidious nature of venous thrombosis in the lower extremity, which takes place in the soleal veins.

Modern imaging methods have evolved considerably in recent years, ranging from I^{125} fibrinogen scanning to venography to Doppler duplex ultrasound, impedance plethysmography (IPG), and magnetic resonance imaging (MRI) technique studies. Although for DVT venography remains the gold standard, modern compression ultrasonography is now the dominant technique, having a good negative predictive value (98%) for proximal DVT and slightly lower (*96%*) for calf DVT. However, it is still inferior to venography, which remains the reference source (**Table 9.2**).

Venography

The venogram has had the most extensive and rigorous use in clinical practice of all imaging techniques. However, it is no longer routinely used because it is invasive, uses contrast dye, has limitations, and provides an increased risk in many

patients who have renal compromise. However, the use of computed tomography (CT) venography in conjunction with CT angiography had proven to increase the sensitivity of DVT diagnosis from 83% to 90% in the PIOPED-II study.'

TABLE 9.1 Levels of Thromboembolism Risk in Surgical Patients without Prophylaxis

LEVEL OF RISK EXAMPLES	CALF DVT, %	PROXIMAL DVT, %	CLINICAL PE, %	FATAL PE, %	SUCCESSFUL PREVENTION STRATEGIES
Low risk	2	0.4	0.2	0.002	No specific measures
Minor surgery in patients <40 y with no additional risk factors					Aggressive mobilization
Moderate risk	10-20	2-4	1-2	0.1-0.4	LDUH q12h, LMWH, ES, or IPC
Minor surgery in patients with additional risk factors; nonmajor surgery in patients aged 40-60 y with no additional risk factors; major surgery in patients <40 y with no additional risk factors					
High risk	20-40	4-8	2-4	0.4-1.0	LDUH q8h,

					LMWH, or IPC
Nonmajor surgery in patients >60 y or with additional risk factors; major surgery in patients >40 y or with additional risk factors					
Highest risk Major surgery in patients >40 y plus prior VTE, cancer, or molecular hypercoagulable state; hip or knee arthroplasty, hip fracture surgery; major trauma; spinal cord injury	40-80	10-20	4-10	0.2-5	LMWH, oral anticoagulants, IPC/ES + LDUH/LMWH, or ADH

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I^{125}-Labeled Fibrinogen Scanning

This technique first developed in the 1960s was used widely for many years. It involves the intravenous injection of isotopelabeled fibrinogen, which is expected to be incorporated into the evolving thrombus and can be imaged by a scintillation scanner. Because of the use of isotopes, it is technically cumbersome and rarely used, despite many large studies validating its use in the 1970s and 1980s and its high correlation with venography.

Impedance Plethysmography

Impedance plethysmography is based on the principal of electrical resistance in specific areas of the body. When there is resistance to blood flow that is due to a thrombus, there is marked reduction in the electrical resistance over that vessel. IPG is most useful in proximal venous thrombosis but is relatively poor in visualizing thrombi below the knee because of the small caliber and slow flow rates through the soleal sinuses. The technique is only about 50% accurate compared with venography in detecting DVT below the popliteal vessels.

Huisman and colleagues evaluated 471 outpatients clinically suspected of acute-onset DVT. Four sequential IPGs were obtained on days 1, 2, 5, and 10 of the study. Of the 137 patients with abnormal results, 117 (85%) had abnormal results on day 1, with the other 20 patients becoming positive by day 10. When compared with venography, serial IPG had a specificity of 92% and a sensitivity of 100%. The use of serial testing clearly improved the ability to diagnose DVT. Another similar study by Vaccaro and associates involving 252 patients using single-test IPG gave a sensitivity of 84% and a specificity of 78%, confirming the superiority of the serial testing protocol. Given the short length of hospital stays now, it is unlikely that serial IPG would be possible despite its proven high correlation with venography.

Doppler Ultrasound

The use of Doppler ultrasound, often with computer color enhancement, has become the most widely used imaging technique for diagnosis of DVT. Its major physiologic use is in the measurement of flow velocity in larger blood vessels. In this technique, a reflected sound signal is converted to both an audible form and visual image on a computer screen. In the presence of a thrombosis, there is a decrease in the reflected signal that can be heard or, more likely, can be visualized. Most modern ultrasound machines use color enhancement to identify arteries (red) and veins (blue). This technique is again very useful to identify DVTs in the iliac, femoral, or popliteal veins, but its sensitivity falls off markedly when applied to the small vessels in the calf, usually to less than 60%.

Real-Time Ultrasound

Real-time ultrasound has been compared with venography, and in a study by Aitken and Godden, it demonstrated a sensitivity of 94% and a specificity of 100% in a small study of

46 patients. In a slightly larger study of 121 patients by Appelman and colleagues, the sensitivity was found to be *96%*, and specificity was 97%.

TABLE 9.2 Diagnosis of Deep Venous Thrombosis

METHOD	SENSITIVITY AND SPECIFICITY	INDICATION AND COMMENTS
Clinical history and physical examination		Classic symptoms often absent in proven DVT <60% with suggestive symptoms proven to have DVT Absence of symptoms does not exclude PE
D-dimer ELISA (plasma)	Sensitivity 97% Specificity: poor	Useful to exclude the diagnosis if D-dimer ELISA is <500 µg/mL Many conditions increase D-dimer levels (false-positive results)
B-mode compression ultrasonography ± Doppler	Symptomatic proximal DVT: Sensitivity 93%-97% Specificity 98% Asymptomatic DVT: Sensitivity 38%-59% Specificity: high	Noninvasive test First-line modality for confirming diagnosis in symptomatic patients Not useful for screening of asymptomatic patients Compression component best for thigh DVT
IPG	Symptomatic DVT: Sensitivity 90% Specificity 95% Asymptomatic DVT: Sensitivity 22% Specificity 98%	Noninvasive test Limited to *serial* examination in symptomatic patients with proximal DVT Insensitive to calf-vein thrombi and nonocclusive thrombi
MR venography	Proximal DVT: Sensitivity 100% Specificity 96% Calf-vein DVT: Sensitivity 87%	Noninvasive but expensive Ability to screen for DVT in asymptomatic patients Can also image lungs for PE in same setting

	Specificity 97%	
Contrast-enhanced CT venography	Distal and proximal DVT: Sensitivity 100% Specificity 96%	Noninvasive test Superior to venography in evaluating the great vessels Expensive Less contrast than conventional venography Can image lungs for PE in same setting
Ascending contrast venography phlebography	Reference standard	Invasive test Reference standard but expensive Risk for contrast nephropathy and allergic reactions Risk for thrombogenicity (usually superficial veins) Negative test does not exclude PE Equivocal results in recurrent DVT

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Compression Ultrasound

This technique has been used in some large DVT trials, such as the PREVENT trial, but there have been few studies directly comparing it with venography. In a recent small study by Tomkowski and colleagues involving 160 medically ill patients, 12 patients had venographically proven DVT. Compression ultrasound technique had a sensitivity of 28% and a specificity of 98%, but despite the small numbers of venographically confirmed patients, the method performed poorly, having both false-positive and false-negative findings.

Duplex Doppler Ultrasound

This is a combination technique using real-time and Doppler methods in a procedure known as B mode or duplex Doppler imaging. It allows a radiologist to visualize the vessel and identify any thrombus in it. In a study by Langsfeld and colleagues, 431 patients were examined; 86 patients had a DVT. This gave a sensitivity of 100%, but two false positives dropped the specificity to 78%. Technical issues may have given one incorrect result, the second patient was pregnant, and the study was considered falsely positive because of aortocaval compression from the pregnant uterus.

In a study by Kristo and associates comparing duplex Doppler, venography, and single bilateral IPG, the respective sensitivities and specificities were as follows: ultrasound, 92% and 100%; venography, 100% and 75%; and IPG, 50% and 83%.

For many reasons, duplex B mode imaging has become the noninvasive imaging method of choice, essentially replacing venography as the “practical” gold standard.

Light Reflection Rheography

Light reflection rheography (LRR) uses infrared light directed at the skin. The backscattered rays are quantitated, which allow an estimation of blood volume. A decreased venous emptying rate of 0.35 is considered positive for DVT. In a study in patients with gastrointestinal problems in which *69* limbs were tested by venography and LRR, the sensitivity for LRR was *96%*, the specificity was 83%, the positive predictive value was 79%, and the negative predictive value was 97%.

Light reflection rheography could prove to be a low-cost, sensitive tool for DVT detection. Further studies are needed to determine if the technique fulfills its early promise. However, in one study of 411 asymptomatic pregnant women in the second and third trimesters who did not have DVT, the use of LRR denoted a significant false-positive rate of 25% and an inadequate study rate of 19%. This gave an overall specificity of only 45%, indicating that LRR is not for use in DVT diagnosis in pregnant women.

Radioisotope Imaging

Various imaging methods have been tried using radioisotopes to try to detect thrombi in both arteries and veins by labeling components of the clotting system, such as platelets or fibrinogen. These labeled components became incorporated into the developing thrombus, and the focused radioactivity would then allow “visualization” by a detector. Radioactive-tagged antibodies to both platelets and factors have also been used for DVT diagnosis.

Indium^{111}-labeled platelets have demonstrated good success with high sensitivity and specificity. The same is true for fibrinogen^{125}- and technetium^{99m}-labeled platelets.

Although all of these radioisotope-labeled methods have some proponents, the advances in computer software technology and imaging methods will probably outweigh significant development of radioisotopic methods for routine clinical VTE diagnosis.

Indirect Computed Tomography Venography

The new and evolving technique of indirect CT venography, using intravenous contrast medium injection followed by CT scanning of the limbs or chest, has a high potential for detecting DVT or PE. Some early studies have indicated detection of thrombi at least to calf level and perhaps lower. There are few studies comparing CT venography and CT pulmonary angiography. A recent study by Nchimi evaluated 1,408 patients with both techniques for PE detection and included lower-extremity DVT as a secondary finding. They found that in 48% of patients with DVT, the upper end of the thrombus was between the ankle and the knee. Comparing the two techniques, CT venography detected 17% more VTE than did CT pulmonary angiography.

Magnetic Resonance Imaging/Magnetic Resonance Imaging Venography

The use of MRI techniques with or without contrast media is another new approach to VTE detection. There are several different technologies using MRI, but all basically use the differences in signal intensities to distinguish flowing blood from stagnant blood (i.e., clot). Although there have been few largescale studies published, the major advantage of MRI is that no contrast medium is required, allowing the technique to be used in pregnant women. One study by Carpenter and colleagues indicated no statistical differences between contrast venography and MRI in DVT diagnosis. Magnetic resonance imaging techniques for routine VTE and arterial thromboses diagnosis will likely continue, as improvements in computer software will provide for the advancement of MRI-based method as tools for vascular diagnostics.

Evolving Imaging Techniques

Because of progress in instruments and computer software in conjunction with ongoing concerns about radiation exposure to patients, several new technical modifications are being evaluated, especially for PE diagnosis. These include multidetector computed tomography angiography (MDCTA), electrocardiogram (ECG)-gated CT angiography, and dual-energy/dual-source CT angiography. Early studies suggest that these methods, especially the dual-source CT angiography, may have significantly enhanced accuracy using the very latest detection technology to minimize the radiation required to provide good diagnostic images.

Nonimaging Methods

The use of laboratory tests as a means of exclusion of VTE has gained momentum in the last decade. The use of the *automated quantitative D-dimer assays* has gained widespread acceptance, especially in emergency rooms, to exclude VTE. In a study by Wells and associates comparing IPG and D-dimer with contrast venography, the combination of IPG and a negative (normal) D-dimer test gave a negative predictive value of 97%. In this same study, the combination of positive IPG and positive D-dimer had a positive

predictive value of 93% for any DVT and 90% for proximal DVT.

The use of an appropriate D-dimer assay in isolation has been shown to have about 98% negative predictive value for exclusion of DVT. Despite numerous studies, there is still no proven consistent correlation between a positive D-dimer assay and the presence of venous thrombosis.

The utility and power of D-dimer assays for exclusion of PE and DVT have been enhanced by the use of pretest probability scores. Several of these exist, namely, the Wells model criteria for DVT and PE diagnosis as well as the Geneva score or other modifications such as the Pisa score. All of these scoring systems are simple and applicable to most emergent VTE diagnostic situations. In conjunction with D-dimer assays, they all enhance the negative predictive value for VTE exclusion and hence the need for unnecessary imaging studies.

Because the diagnosis of PE can be difficult in many older patients, especially those with heart failure or other cardiovascular complications, some preliminary studies have been done using combinations of D-dimer, B-type natriuretic peptide, and cardiac troponins to determine if a better distinction can be made between a PE and an underlying cardiac complication. These studies are likely to be the forerunners for other combinations of biomarker lab tests to identify more specific negative or even positive predictive markers for VTE. This is especially important since many older patients with VTEs have some degree of renal impairment, and the use of contrast dyes in imaging can be problematic.

Risk Factors for Vascular Complications

Several clinical factors are known to identify the patient with an increased risk for VTE (**Table 9.3**). The most prevalent and important include age >40 years, obesity >20% above ideal weight, prolonged surgery, and immobility in the pre-, peri-, and postoperative periods. Pelvic malignancy, prior VTE, known thrombophilia risk, severe diabetes, heart failure, prior radiation therapy, and chronic obstructive pulmonary disease all increase the VTE risk.

TABLE 9.3 Profile of Patient at High Risk for Venous Thrombosis

FACTOR		CONDITION
Age		<40 Major surgery
Age		>60 Nonmajor surgery
Obesity		
	Moderate	75-90 kg or >20% above ideal weight
	Morbid	115 kg or >30% above ideal weight with reduced fibrinolysin and immobility
Immobility		
	Preoperative	Prolonged hospitalization; venous stasis
	Intraoperative	Prolonged operative time; loss of pump action of calf muscles; compression of vena cava
	Postoperative	Prolonged bed confinement; venous stasis
Trauma		Damage of wall of pelvic veins
Radical pelvic surgery		
Malignancy		Release of tissue thromboplastin[a]
Activation of factor X; reduced fibrinolysin		
Radiation		Prior radiation therapy
Medical diseases		Diabetes mellitus
Cardiac disease; heart failure		
Severe varicose veins		
Previous venous thrombosis with or without embolization[a]		
Chronic pulmonary disease		
Molecular hypercoagulable state		

[a]Highest risk.

Age

An autopsy study by Sevitt and Gallagher demonstrated that DVT was most prevalent in patients older than 60 years. Several studies have shown a linear risk of fatal PE with increasing age. Approximately 10% of hospitalized patients' deaths are due to PE, and only about 35% of these are diagnosed ante mortem. Contributing factors include degenerative changes in the vascular tree, increases that occur in the concentration of many coagulation factors, and, possibly, increased platelet adhesiveness.

Immobility

Prolonged inactivity in the preoperative patient promotes an impairment of venous flow in the lower extremities. Many diagnostic techniques also produce a decrease in muscle tone with a secondary decrease in venous flow. These hemodynamic changes promote sludging of red cells and activation of platelets, setting the stage for VTE during the operative period. This is one of the main risk factors described by Virchow for the etiology of thrombosis.

Studies done using I^{125} fibrinogen scanning presurgery and immediately postsurgery have indicated that in 50% of patients who subsequently developed VTE, the initiation of clot formation occurred during the surgical procedure. This is amplified during prolonged anesthesia, with generalized muscle relaxation further promoting venous stasis in the lower extremities, which compounds the thromboembolic risk. For this risk, the judicious use of prophylactic anticoagulation in the high-risk patient should include the operative phase and continue at a minimum until the patient is fully ambulatory.

Postoperative immobility also promotes VTE risk by continuing venous stasis, and studies have shown that *66%* of patients who develop a DVT do so in the first 48 hours after surgery. Other compounding issues include sitting with legs crossed or dangling over the bed or the exaggerated Fowler position. These positions all produce impairment of lower extremity venous return. Postoperative patients should be ambulated early and aggressively; if ambulation is not possible, they should have their legs elevated to 15 degrees above the horizontal.

Other Factors

Other factors include previous VTE, varicose veins, severe diabetes, cardiac failure, chronic obstructive pulmonary disease, and underlying thrombophilia. Given the high incidence of factor V Leiden (5%) and the G20210A prothrombin gene mutations (3%) in the Caucasian populations, which are wellrecognized risk factors for venous thrombosis (**Table 9.4**), these genetic risk factors are common enough to make a major contribution to preoperative thrombosis, even in the patient with no prior VTE history.

Underlying malignancy is a huge contributor to risk, most likely because of the significant up-regulation of TF that is known to occur with many malignancies. Up-regulation of TF increases thrombin generation by several mechanisms, promotes platelet activation, and enhances the generation of MPs and angiogenesis, as discussed earlier.

A review of these risk factors clearly identifies the high-risk VTE patient, and it is essential that these surgical risk variables are identified and understood in designing appropriate thromboprophylaxis and monitoring parameters to prevent venous thrombosis. Many countries including the United States have produced national consensus documents from their clinical

oncology societies for both treatment and prophylaxis for VTE in cancer patients.

TABLE 9.4 Risk of Thromboembolism

Deficiency/dysfunction
Antithrombin
Protein C
Protein S
Heparin cofactor II
Factor V Leiden
Prothrombin variant 20210A
Antiphospholipid antibodies
Lupus anticoagulant
Anticardiolipin
Hyperhomocystinuria
Dysfibrinogenemia
Decreased levels of plasminogen
Decreased levels of plasminogen activators
Heparin-induced thrombocytopenia

Prophylaxis

Prevention remains the most effective tool in the treatment of VTE. Between 5% and 45% of gynecologic surgery patients develop DVT in their legs; of these, 20% have popliteal or femoral involvement; and of these, 40% will progress to PE with its high mortality. It is imperative that methods for prophylaxis are planned and implemented before surgery (**Table 9.5**).

TABLE 9.5 Agents Used in Venous Thromboembolism

AGENT	MECHANISM OF ACTION	COMMENTS
Heparin	Combines with AT-III and neutralizes activated factors: IIa (thrombin activity) Xa (responsible for thrombin generation) XIIa, XIa, IXa	Prevention and treatment of VTE Risk of heparin-induced thrombocytopenia Requires monitoring (APTT) when used for treatment

LMWH Ardeparin Dalteparin Enoxaparin	Combines with AT-III and prevents thrombin generation through its anti-factor *Xa* effect	Prevention and treatment of VTE Risk of heparin-induced thrombocytopenia No anti-IIa activity (if molecular weight <5.6 kDa) APTT does not reflect anticoagulation state More predictable pharmacokinetic profile Renal failure and dehydration increase effective plasma concentration
Heparinoid Danaparoid	Same as LMWH High anti-Xa/IIa ratio	Prevention and treatment of VTE Similar to LMWH but may be used for anticoagulation when heparin-induced thrombocytopenia is present
Direct thrombin inhibitors and hirudin	Directly inhibits thrombin activity	Prevention and treatment of VTE May be used for heparin-induced thrombocytopenia
Plasminogen activators: Nonselective Streptokinase Urokinase	Activates plasminogen, which leads to the formation of plasmin, which dissolves fibrin clot (no effect on polymerized fibrin clot) Also degrades fibrinogen, which leads to fibrinogen degradation products and decreases in plasma fibrinogen	Treatment of life-threatening DVT or PE High risk of bleeding Many contraindications such as recent surgery or trauma
Thrombus-selective tissue plasminogen activator	Activates fibrin-bound plasminogen Degrades fibrinogen (to a lesser extent)	
Warfarin	Inhibits correct synthesis of vitamin K-dependent coagulation factors (II, VII, IX, X) These factors cannot bind calcium and therefore remain inactive Inhibits protein C (vitamin K dependent)	Long-term treatment and prevention of VTE Contraindicated in pregnancy (teratogenic) High risk of bleeding Requires anticoagulation monitoring Numerous drug interactions
Inferior vena caval filters	Trap larger emboli	Used as prevention of PE when anticoagulation fails or is contraindicated Used prior to pulmonary embolectomy or pulmonary endarterectomy
External pneumatic leg compression	Prevents venous stasis Stimulates fibrinolytic system	Used as prophylaxis for DVT Possibly contraindicated in peripheral arterial disease

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In the Sixth American College of Chest Physicians (ACCP) Consensus Conference, Geerts and colleagues—in a review of PE in 7,000 gynecologic surgery patients in prospective clinical trials—reported a reduction in the rate of fatal PE of 75% using thromboprophylaxis (Table 9.6). The current 2012 Chest (ACCP) guidelines recommend the routine use of LMWH or unfractionated heparin (UFH) and mechanical compression stockings given the high risk in this population. They do not recommend the use of inferior vena cava (IVC) filters.

TABLE 9.6 Prevention of Deep Venous Thrombosis after Gynecologic Surgery[a]

REGIMEN	NO. OF TRIALS	NO. OF PATIENTS	INCIDENCE OF DVT, %	95% CI	RELATIVE % REDUCTION
Untreated control subjects	12	945	16	14-19	—
Oral anticoagulants	5	183	13	8-18	22
IPC	3	253	9	6-13	44
LDUH	11	1092	7	*6-9*	56
ES	1	104	0	0-3	*"99"*

[a]Pooled data from randomized trials that used routine I^{125} fibrinogen-uptake test (FUT) as the primary outcome. Reprinted with permission from Geerts WH, Heit JA, Clagett GP, et al. Prevention of venous thromboembolism (Sixth ACCP Consensus Conference on Antithrombotic Therapy) *Chest* 2001;119(1 suppl):132S. Copyright © 2008, American College of Chest Physicians.

Low-Dose Unfractionated Heparin

Low-dose UFH has been the mainstay of prophylactic treatment for many years, with numerous prospective randomized clinical trials validating a risk reduction in DVT incidence from 35% to 45% to about 7% in the high-risk patient. In one large study by Kakkar and colleagues (Table 9.7) incorporating 4,000 patients at multiple centers, patients were randomized to 5,000 U USP calcium heparin subcutaneously starting 2 hours before surgery and subsequently every 8 hours thereafter for the next 7 days. The reduction in VTE between the control group (25%) and the treatment group (8%) was highly significant. The most important finding was the decrease in fatal PE from 16 patients in the control group to 2 in the treatment group confirmed by autopsy.

Because there is no change in the activated partial thromboplastin time (APTT) because of the low level of subcutaneous heparin, there was no increase in postoperative bleeding.

This is because the main impact of the low-dose heparin is exerted via antithrombin through factor Xa, as well as directly on thrombin. There is also a secondary effect in which heparin releases tissue factor pathway inhibitor (TFPI), which also helps to down-regulate factor Xa by forming a complex involving TF:F Xa:F VIIa:TFPI.

TABLE 9.7 Pulmonary Embolism and Deep Venous Thrombosis in Patients on Low-Dose Heparin and in Controls

	LOW-DOSE HEPARIN	CONTROL
Number of patients	2045	2076
Number of deaths from all causes	80	100
Deaths caused by PE (verified at autopsy)	2	16
Deep venous thrombosis	8%	25%

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These studies, as well as those shown in Table 9.8, clearly validate the efficacy of low-dose UFH in reducing VTE in surgical patients using an initial dose of 5,000 U 2 hours before surgery and then 5,000 U every 12 hours for the next 5 days. For the truly high-risk patient, such as those with a prior VTE or multiple risk factors, 5,000 U every 8 hours should be used. In current clinical practice, the use of UFH has been superseded by the use of LMWHs.

Low-Dose Unfractionated Heparin/Dihydroergotamine

The combination of low-dose UFH and dihydroergotamine (DHE) treatment was shown to work well by adding the known effect of DHE as a selective venous vasoconstricting agent to the anticoagulant properties of UFH.

Dextran 70/Dextran 40

In 1972, Bonnar and Walsh described the use of dextran 70 to prevent thrombosis after pelvic surgery. A subsequent study by Bernstein and colleagues involving radical hysterectomy patients using dextran 70 as prophylaxis showed a decrease in DVT incidence from 33% to 5%. Dextran works by interfering with platelet function, interacts with factor V and VIII, and inhibits fibrinolysis. Despite some comparable studies between dextran and low-dose UFH, the Sixth ACCP Consensus Conference in 2001 recommended against using dextran products in VTE prophylaxis.

Low Molecular Weight Heparins

Low molecular weight heparins (LMWHs) act by primarily inhibiting factor Xa with a small component of activity against thrombin. They have become mainstay of anticoagulant prophylaxis and treatment and continue to replace all other forms of drug therapy. The drugs have a longer half-life than does UFH and are much more biopredictable. If LMWH levels are measured in patients using an anti-Xa assay, there is a remarkable homogeneity of response. This has led the U.S. Food and Drug Administration to recommend against the need to monitor when LMWHs are used for VTE prophylaxis. There are currently four LMWH drugs available in the United States (Fragmin, Lovenox, Innohep, and Arixtra). They are subtly different in molecular weights and in manufacturing processes but in essence are almost identical in clinical efficacy. They are not, however, dosed in the same way, some using milligrams and other units, or even units per kilogram. Pharmacists can provide accurate dosage information about any of the products available.

TABLE 9.8 Results of Prophylactic Treatment of Venous Thrombosis after Gynecologic Surgery[a]

				VENOUS THROMBOSIS (%)					
INVESTIGATORS	YEAR	TYPE OF SURGERY	NUMBER OF PATIENTS	CONTROL	HEPARIN	LMWH	DEXTRAN	AC	PNEUMATIC CALF COMPRESSION
Bonnar et al.	1973	Simple hysterectomy	260	15.0	—	—	0.1	—	—
		Radical malignant	62	33.0	—	—	5.0	—	—
Ballard et al.	1973	Major gynecologic, age 40	110	29.0	3.6	—	—	—	—
McCarthy et al.	1974	Major gynecologic	130	—	10.9	—	16.2	—	—
Baertschi et al.	1975	Major gynecologic	458	—	2.3	—	—	4.7	—
Gjonnaess and Abildgaard	1976	Major gynecologic, age 50	95	8.0	2.0	—	—	—	—
Adolf et al.	1978	Major gynecologic	454	29.3	7.0	—	—	—	—
Taberner et al.	1978	Major gynecologic	146	23.0	6.0	—	—	6.0	—
Clarke-Pearson et al.	1983a	Gynecologic malignant	185	12.4	14.8	—	—	—	—
Clarke-Pearson et al.	1984	Gynecologic malignant	107	34.6	—	—	—	—	12.7
Borstad et al.	1992	Major gynecologic	141	—	0.00	0.00[b]	—	—	—

		malignant								

[a]Detected by ^{125}I-labeled fibrinogen scan.

[b]One patient had PE 3 days after discontinuation of LMWH.

AC, anticoagulants; LMWH, low-molecular-weight heparin.

Multiple studies reported in the literature essentially show equivalence or better for the LMWHs compared with UFH and/or Coumadin in the prevention of VTE, but almost all of these show a much lower bleeding risk for the LMWH treatment groups, even with hard data for bleeding risk being quantitated by transfusion requirements. LMWHs have also been compared with dextran and used in combination with DHE with good outcomes, but the reality is that *in normal clinical practice, LMWHs by themselves provide adequate protections with minimal complications.* One other advantage of the LMWH preparations is their much lower incidence of heparin-induced thrombocytopenia (HIT) when used as de novo therapy. However, if a patient has had HIT in the past, these preparations should not be used because there is a 90% cross-reactivity between UFH and LMWH for the antibody causing HIT. The one exception to this is Arixtra, the synthetic factor Xa inhibitor, which has been shown to cause clinical HIT in two to three cases to date.

It is highly likely, given the once-per-day dosage requirement and the lack of HIT risk, that LMWHs will continue to dominate for thromboprophylaxis therapy in VTE.

Newer Oral Anticoagulants

In the past 18 months, three new oral anticoagulants have been approved for use in various medical conditions. One of these drugs, dabigatran (Pradaxa), is an anti-IIa (thrombin) inhibitor approved for atrial fibrillation similar to a new oral anti-Xa inhibitor apixaban (Eliquis). However, the third new drug, an oral anti-Xa inhibitor, rivaroxaban (Xarelto), has been approved for treatment and prophylaxis of VTE although not specifically tried in cancer-related VTE.

The fact that Xarelto is oral, does not require any monitoring, and has few side effects is likely to promote increased use especially from the patient compliance perspective where the need for subcutaneous injections remains a problem.

Compression Modalities

As long ago as 1944, Stanton et al. used static compression to decrease venous stasis by decreasing the luminal diameter of the veins, thereby increasing blood flow velocity. In the mid-1970s, Sigel and colleagues showed an increase in blood velocity of 20% using graduated compression stockings but a 200% increase in velocity using intermittent sequential compression.

Mittelman and colleagues showed that uniform intermittent calf compression was not as effective as intermittent sequential compression at increasing thigh blood flow. This is another example of a component of Virchow triangle, namely, stasis being involved in the VTE protection mechanism.

It is possible that another component of the triad—the coagulation system—is also influenced by IPC because several groups have shown that it stimulates fibrinolysis, perhaps by increasing prostacyclin production. Prostacyclin is a potent natural vasodilator and antiplatelet agent released from endothelial cells. Guyton and colleagues found increased quantities of 6-keto prostaglandin $F_{1\alpha}$ in patients undergoing IPC compared with controls. The 6-keto prostaglandin $F_{1\alpha}$ is a

specific breakdown product of prostacyclin. Frango and associates have shown a 16-fold increase in prostacyclin production in cultured endothelial cells submitted to pulsatile shear stress compared with a twofold increase with contact shear stress.

Graduated Compression Stockings

Initial studies evaluating antiembolic stockings proved inconclusive and relied on several different methods to diagnose VTE, which compounded the uncertainty. Sigel and colleagues designed a compression thromboembolism deterrent (TED) hose with graduated pressures of 18, 14, 12, 10, and 8 mm Hg from the ankle to the upper thigh. Scurr and associates evaluated TED hose in a study of 70 patients older than 40 years undergoing major abdominal surgery in which only one leg had TED hose applied. Using I^{125} fibrinogen scanning as the diagnostic tool, 19 patients had DVTs in the control leg, and only 1 had a DVT in the TED hose leg. A subsequent similar study by Inada and colleagues found a DVT frequency of 14.5% in the control leg and only 3.6% in the TED leg. Malignancy is a powerful predisposition to VTE secondary to stasis and tissue factor production by the tumor. In a study by Allan and associates assessing the efficacy of TED stocking in patients undergoing abdominal surgery for malignant and benign diseases, the incidence of DVT in the benign disease group was 24.5% in the control limb and 6.1% in the TED limb. However, in the malignant disease group, the incidence of DVT was only slightly increased in the control limb at 27.9%, but the incidence of DVT in the TED limb was significantly higher at 11.5%, clearly amplifying the impact of the tumor on the DVT risk. The Sixth ACCP Consensus Conference suggested that TED hose with early ambulation was an acceptable and effective means of VTE prophylaxis in the low-risk gynecology surgery patient.

External Intermittent Pneumatic Compression

These techniques also promote increased blood flow in the lower extremities that is due to decreased stasis and improved fibrinolysis. Nicolaides and colleagues compared intermittent sequential pneumatic compression, nonsequential (one chamber) pneumatic compression, and UFH in the prevention of DVT. Using pressures of 35, 30, and 20 mm Hg sequentially for 12 seconds at the ankle, calf, and thigh, respectively, they observed a 240% increase in peak blood velocity. In contrast, using the singlechamber device at 35 mm Hg, the increase was only 180%. The intermittent sequential device was more effective than was the single-chamber device and was as effective as 5,000 U of UFH every 12 hours in preventing DVT. In addition, the intermittent sequential device increased the time interval for clot formation proximal to the calf compared with UFH. In another study, the same authors compared electrical calf stimulus, low-dose UFH, intermittent sequential compression, and TED hose in 150 patients older than 30 years undergoing major abdominal surgery. The incidence of proven DVT was 18%, 9%, and 4%, respectively.

In a similar study in patients undergoing surgery for gynecologic malignancy comparing no thromboprophylaxis to nonsequential external compression, the control group had a VTE frequency of 34.6%. In the compression treatment group, the VTE incidence was reduced to 12.7%. Diagnostic tools for VTE were IPG and I^{131}.

Treatment of Venous Thrombosis

The initial treatment of VTE in most hospitals still involves the use of intravenous UFH, although LMWHs are approved for treatment of VTE. Given the potential variability of the hypercoagulable state in these patients, constant intravenous infusion UFH still remains the easiest drug to use. Most patients are now treated using a weight-based heparin nomogram and are given a loading dose of 80 U/kg to a maximum of 10,000 U. They are maintained on a constant infusion of 18 U/kg, and the first APTT or factor Xa assay should be done at 4 to 6 hours after the initiation of therapy. Because of the large thrombus burden these patients may have, they can clear or use UFH at an accelerated rate; thus, they are often undertreated. Patients with gynecologic malignancies will typically require higher-than-average doses until the cancer has been surgically removed or treated. Care must be taken to reduce the infusion, or these patients are susceptible to bleeding that is due to heparin overdose. There is little place for intermittent bolus treatment with UFH in modern anticoagulation practice.

If the APTT is used to monitor efficacy of UFH therapy, it should be used in conjunction with the laboratory heparin monitoring nomogram. Because APTT reagents can vary widely between institutions, the old concept of using a ratio of 1.5 to 2 times some poorly defined control APTT value is completely outmoded and can lead to erroneous and inadequate anticoagulation therapy. Any modern coagulation lab should have a heparin treatment weight-based nomogram specific for their reagent and APTT instrument combination.

Standard clinical practices—such as leg elevation to minimize or treat leg edema after a DVT—are still appropriate. Patients should probably not be aggressively mobilized as long as they have significant leg edema. Most patients will require 5 to 7 days of UFH or LMWH treatment, and the modern trend is to rapidly introduce oral anticoagulation, usually within 24 to 48 hours after initiation of heparin therapy. This will not always be possible in this population, but any UFH or LMWH treatment should be continued until the international normalized ratio is in the therapeutic range of 2 to 3 for several days. The new oral anticoagulants (Xarelto) can be initiated in place of Coumadin anticoagulation immediately after

surgery and do not require any time to become therapeutic. Similar to Coumadin, they need to be continued for 3 to 6 months or longer, depending on the circumstances and any other complicating factors (i.e., prior VTE or congenital thrombophilia).

The patient who is found to have an asymptomatic DVT of the lower extremity poses a dilemma for some physicians. Some feel that this is not a significant risk and should not be treated, but the risk of thrombus extension into the proximal and popliteal veins remains high and is a possibility. Although this is still a small risk for most patients, the longer-term complications of postphlebitic syndrome from the damaged valves in those veins can produce significant morbidity for such patients, leading to chronic leg edema and venous stasis ulceration. For that reason, most practitioners would elect to anticoagulate women with asymptomatic DVT. With the onset of widespread DVT prophylaxis in many hospitals, the problem of whether to treat asymptomatic DVT is becoming moot.

POSTOPERATIVE PULMONARY COMPLICATIONS

Postoperative pulmonary complications (PPCs) after abdominal surgery remain an important cause of increased morbidity, mortality, and resource use. Atelectasis, pneumonia, and pulmonary thromboembolic disease following abdominal surgery continue to occur frequently despite continuing advances in anesthetic, surgical, and postoperative treatment. The incidence of PPCs has surpassed that of postoperative cardiac complications, and PPCs have a greater impact on postoperative outcomes. Gynecologic surgery is increasingly performed in patients with advanced age, multiple comorbid conditions, and increased risk for the development of PPCs. Risk factors

for PPCs in patients undergoing a gynecologic surgery procedure vary among studies but are consistent with other patient groups undergoing abdominal surgical procedures (**Table 9.9**). The most important PPCs in terms of incidence, morbidity, mortality, and resource use are atelectasis, pneumonia, respiratory failure, and pulmonary thromboembolic disease.

TABLE 9.9 Risk Factors for Postoperative Pulmonary Complications in Gynecologic Surgery Patients

Age >60 y
Cancer
Congestive heart failure
Smoking within 8 wk of surgery
Upper abdominal incision
Vertical incision
Incision length >20 cm

An understanding of the physiology that predisposes to PPCs, the risk factors for their development, and the preventive and therapeutic measures to minimize and treat them are of critical importance to gynecologic surgeons.

Perioperative Respiratory Physiology

Effects of Anesthesia

General anesthesia results in important alterations in respiratory physiology (**Table 9.10**). Anesthetic agents influence not only the ventilatory response to oxygen and carbon dioxide but also the pattern of respiration. Inhalational agents and intravenous agents both result in a reduction of the ventilatory response but differ in their effects on respiratory pattern. The classic breathing pattern produced by inhalational anesthetics is a rhythmic, rapid, and shallow pattern of respiration with no intermittent sighs (large breaths), whereas intravenous anesthesia is associated with slow, deep respirations. Little metabolism of inhalational anesthetics occurs during surgery, with most of the anesthetic agents stored in the tissues, such as muscle and fat.

At the conclusion of anesthesia, most of the stored anesthetic agent is eliminated via the lungs. As a result of tissue stores, significant concentrations of the anesthetic agent may be present well into the recovery phase, particularly after high anesthetic doses, long anesthetic times, or the presence of cardiopulmonary disease. This prolonged anesthetic effect can lead to clinically significant respiratory depression in the postoperative period.

The number of functional alveolar units participating actively in gas exchange is directly related to the functional residual capacity (FRC). General anesthesia is associated with a reduction in FRC by approximately 16%, irrespective of the anesthetic techniques used. The cause of the reduction in FRC is multifactorial and includes cranial movement of the diaphragm, chest wall relaxation with reduction in thoracic volume, reduction in respiratory compliance, and shift of central blood volume from the thorax into the abdomen. Reduction in FRC can have marked adverse effects on perioperative gas exchange, especially the development of hypoxemia.

TABLE 9.10 Effects of Anesthesia on Respiratory Physiology

Reduced ventilatory response to oxygen and carbon dioxide
Rhythmic rapid shallow breathing pattern
Reduced functional residual capacity
Diaphragmatic dysfunction
Atelectasis
Ventilation-perfusion mismatching
Blunting of hypoxic pulmonary vasoconstriction
Impairment in mucociliary clearance

Atelectasis is defined as the absence of gas from a part or the whole of the lungs that is due to the failure of expansion or resorption of gas from the alveoli. It occurs in the dependent areas of the lungs within 5 minutes of anesthetic induction in a patient with healthy lungs and leads to shunt physiology. Atelectasis may be caused by compression, gas resorption, or surfactant impairment. Compression occurs when the distending pressure in the alveolus is reduced to a level that causes the alveolus to collapse. In the setting of general anesthesia, compression occurs mainly as result of impairments in diaphragmatic position and function. In addition to the cephalad movement of the diaphragm that is due to relaxation from anesthesia as described above, increased intra-abdominal pressure from bowel edema, peritoneal fluid, and hematoma forces the diaphragm cephalad and contributes substantially to compressive atelectasis. The contractile function of the diaphragm is not altered as a result of an effect of anesthesia itself because diaphragmatic dysfunction, which almost always presents after upper abdominal surgery, does not arise after lower abdominal surgery.

Other factors contributing to postanesthesia pulmonary complications include reabsorption hypoxic pulmonary vasoconstriction and ventilation/perfusion abnormalities.

Although most gynecologic surgery is done in the pelvis, upper abdominal operations are sometimes done by gynecologic oncologists, and extension of the surgical incision and operative procedure into the upper abdomen produce increased respiratory effects. Respiratory muscle dysfunction is the major effect of upper abdominal surgery on respiratory physiology (**Table 9.11**). Tidal breathing depends on inspiratory muscle function, especially the function of the diaphragm. Other accessory muscles of respiration are recruited when work of breathing increases, such as when diaphragmatic dysfunction is present, during states of increased oxygen consumption, and in the presence of cardiopulmonary disease. In addition to breathing, respiratory muscles play an integral role in the generation of cough and act as stabilizers of the thorax and abdomen. Upper abdominal surgery may affect each of these respiratory muscle functions by several different mechanisms.

An important change following upper abdominal surgery is a shift in respiratory pump function from the diaphragm to accessory inspiratory and expiratory muscles of respiration. This results in a rapid shallow breathing pattern of respiration. The contractile function of the diaphragm is impaired by inhibition of phrenic nerve output by stimulation of visceral and somatic nerve pathways during manipulation of the abdominal

viscera and the peritoneum. The less efficient accessory muscles of inspiration, such as the intercostals and neck muscles, assume an increased share of the respiratory effort. Tonic and phasic contraction of the expiratory abdominal muscles also occurs. The net effect on respiratory mechanics is a reduction in lung volumes, including the FRC (which leads to atelectasis), V/Q abnormalities, and hypoxemia. These changes may be aggravated by hypoventilation that is due to the residua of general anesthesia, postoperative sedative-hypnotic therapy, and pain. In addition to a submaximal voluntary activation of inspiratory muscles, pain may also have a direct effect, through unknown mechanisms, on inspiratory muscle function.

TABLE 9.11 Effects of Upper Abdominal Surgery on Respiratory Physiology

Reduction in lung volumes: residual volume, total lung capacity, functional residual capacity and vital capacity
Reflex inhibition of phrenic nerve activity resulting in decreased diaphragmatic function
Increased neck and intercostal inspiratory accessory muscle use
Tonic and phasic contraction of abdominal expiratory muscles

Atelectasis

Clinically significant atelectasis occurs in 15% to 20% of patients undergoing abdominal surgery. The pathophysiologic effects of atelectasis include decreased respiratory compliance, increased pulmonary vascular resistance, predisposition to acute lung injury, and hypoxemia. Atelectasis may also be a precursor to more serious PPCs, such as postoperative pneumonia. The definition of atelectasis is not uniform across clinical studies, with most investigations incorporating a global definition of a PPC that includes atelectasis. However, generally accepted criteria for the diagnosis of atelectasis include impaired oxygenation in a clinical setting where atelectasis is likely, unexplained temperature of greater than 38°C, and chest radiographic evidence of volume loss or new airspace opacity. Risk factors implicated in the development of atelectasis after abdominal surgery include advanced age, obesity, intraperitoneal sepsis, prolonged anesthesia time, nasogastric tube placement, and smoking.

The risk of atelectasis may be reduced by a number of interventions (**Table 9.12**). Preoperative smoking cessation is effective if it is started well in advance of surgery (6 to 8 weeks before operation). If smoking cessation is attempted in close proximity to a planned surgical procedure, the improvement in mucociliary clearance in combination with reduced cough may lead to a secretion burden that paradoxically increases the risk of PPCs. Atelectasis is effectively prevented and treated by deep breathing exercises and mobilization. Voluntary lung inflation exercises enable redistribution of gas into areas of low compliance. Mechanical aids, such as incentive spirometry, have not been shown to be superior to properly performed deep breathing maneuvers. Effective deep breathing exercises require the patient to be conscious and cooperative. Chest physiotherapy is extremely labor intensive and has potential disadvantages in that it may exhaust the patient, cause pain, induce bronchospasm, and cause transient hypoxemia. Because chest physiotherapy has never been shown to be superior to deep breathing exercises in preventing or treating atelectasis, it is not recommended after abdominal surgery. Continuous positive airway pressure reduces the incidence of significant postoperative hypoxemia and may reduce rates of pneumonia and intubation. However, the cost, complexity, and potential complications of continuous positive airway pressure limit the practicality of routine application of this technique to patients undergoing abdominal surgery.

TABLE 9.12 Prevention and Treatment of Atelectasis

Smoking cessation 8 wk before elective surgery
Laparoscopic procedure
Deep breathing exercises
Mobilization
Adequate analgesia (epidural or patient-controlled analgesia preferred)
Selective gastric decompression

Although evidence suggests that effective postoperative pain control reduces PPC, there is inconclusive evidence as to whether postoperative epidural analgesia is superior to patientcontrolled analgesia. Both modalities, however, appear to be superior to on-demand narcotic analgesia in reducing PPCs. Laparoscopic procedures reduce postoperative pain scores, as well as have less adverse effect on postoperative respiratory muscle function. Through these mechanisms, atelectasis is reduced with a laparoscopic as opposed to

open abdominal surgical procedure. Irrespective of the procedure used, postoperative gastric decompression should be used selectively rather than routinely for postoperative nausea, symptomatic abdominal distention, or inability to tolerate oral intake. Routine nasogastric tube use significantly increases rates of atelectasis without reducing risk of aspiration in comparison with selective decompression.

Postoperative Pneumonia

Hospital-acquired pneumonia (HAP) is defined as pneumonia that develops 48 hours or more after hospital admission because of an organism that was not incubating at the time of hospitalization. Hospital-acquired pneumonia after abdominal surgery has a high attributable mortality, increases hospital length of stay and cost, and has lasting effects on patient-centered outcomes, including increased hospital length of stay of approximately 11 days and increased hospital charges by 75%. Hospital-acquired pneumonia is also associated with a fourfold increase in risk of discharge to a skilled nursing facility. Women have a risk of developing HAP after abdominal surgery that is twice that of men.

Hospital-acquired pneumonia is caused by a wide spectrum of bacterial pathogens and is occasionally due to viral or fungal pathogens in immunocompetent patients (**Table 9.13**). Common pathogens include the aerobic gram-negative bacilli and *Staphylococcus aureus* species. Early-onset HAP, defined as occurring within the first 4 days of hospitalization, is usually associated with a better prognosis and is more likely to be caused by antibiotic-susceptible pathogens. Late-onset HAP (occurring on or after 5 days of hospitalization) is more likely to be caused by multidrug-resistant (MDR) pathogens, which are associated with increased morbidity and mortality. The risk of HAP from MDR pathogens is related to characteristics of the patient, the health care environment, and the prescribed medical treatment (**Table 9.14**).

TABLE 9.13 Common Pathogens Causing Hospital-Acquired Pneumonia

Early onset (≤4 d)
Streptococcus pneumoniae
Methicillin-sensitive *Staphylococcus aureus*
Methicillin-resistant *S. aureus*
Haemophilus influenzae
Escherichia coli
Klebsiella pneumoniae
Enterobacter species
Proteus species
Serratia marcescens
Late onset (≥5 d)
All of the above plus:
Pseudomonas aeruginosa
Multidrug-resistant *Klebsiella pneumoniae*
Acinetobacter species

TABLE 9.14 Risk Factors for Multidrug-Resistant Pathogens Causing Hospital-Acquired Pneumonia

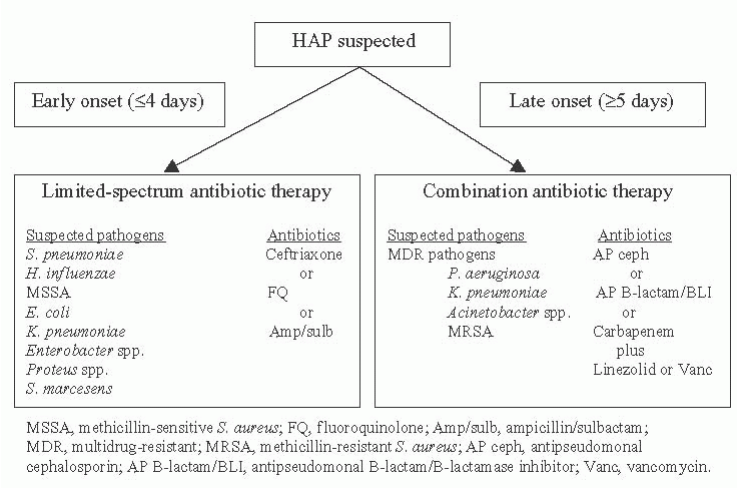
Immunosuppressive disease or therapy
Home infusion therapy
Chronic dialysis
Home wound care
Residence in a nursing home or extended care facility Antimicrobial therapy in the preceding 90 d
Current hospitalization of 5 d or more
Hospitalization for 2 d or more in the preceding 90 d
High frequency of antibiotic resistance in the community, hospital, or patient care unit

Bacterial colonization of the lower respiratory tract under conditions that promote bacterial invasion is necessary for HAP to develop. Sources of colonization include air, water, equipment, fomites, and direct transfer from health care providers. Among conditions that promote invasion are severity of underlying illness, comorbid conditions, prior exposure to antimicrobials, and exposure to invasive devices, such as nasogastric and endotracheal tubes. Risk of colonization may be modified by several means (**Table 9.15**). Infection control

procedures, including guidelines for alcohol-based hand disinfection and appropriate barrier precautions, should strictly be observed. Early removal of invasive devices, in particular nasogastric tubes, and avoidance of endotracheal intubation when noninvasive ventilation is feasible, will reduce the risk of HAP. As one of the mechanisms for initial colonization of the lower respiratory tract includes microaspiration of gastrically residing bacteria, measures to minimize aspiration and to reduce gastric bacterial overgrowth are important preventive measures. All patients should be maintained in at least a semirecumbent position with the head elevated to 30 to 45 degrees. Restriction of the use of stress ulcer prophylaxis to patients who meet criteria (i.e., receiving mechanical ventilation or coagulopathy/therapeutic anticoagulation) is critical in controlling a possible risk factor for HAP. Implementation of a restrictive red blood cell transfusion strategy in patients without evidence of active bleeding is proven to reduce infectious complications of all kinds, including HAP. Adherence to such a strategy not only reduces infectious complications but reduces overall in-hospital mortality and may reduce recurrence of malignancy. In most patients, a hemoglobin target of 7.0 should be adopted, with a hemoglobin target of 9.0 reserved for patients with active cardiac ischemia or hemodynamic instability. Strict control of hyperglycemia has been widely adopted as a means of reducing morbidity and mortality, including the risk of infection. Although the optimal glucose target and the means of achieving that target remain controversial, an attempt to limit capillary blood glucose to less than 150 mg/dL in both diabetic and nondiabetic patients appears warranted.

TABLE 9.15 Interventions to Decrease Risk for Hospital-Acquired Pneumonia

Strict adherence to infection control procedures
Early removal of invasive devices
Semirecumbent positioning of the patient
Early mobilization of the patient
Restriction of acid suppression therapy
Restrictive red blood cell transfusion strategy
Strict control of hyperglycemia



MSSA, methicillin-sensitive *S. aureus*; FQ, fluoroquinolone; Amp/sulb, ampicillin/sulbactam; MDR, multidrug-resistant; MRSA, methicillin-resistant *S. aureus*; AP ceph, antipseudomonal cephalosporin; AP B-lactam/BLI, antipseudomonal B-lactam/B-lactamase inhibitor; Vanc, vancomycin.

FIGURE 9.3 Algorithm for initiating empiric antibiotic therapy for hospital-acquired pneumonia (HAP).

The clinical definition of HAP includes a new opacity on chest radiograph (posterior-anterior and lateral views preferred) plus two of the following: fever greater than 38°C, leukocytosis or leukopenia, and purulent respiratory secretions. The diagnosis of HAP should be supported by sampling of lower respiratory tract secretions, with either an endotracheal aspirate or a bronchoscopic specimen, before the initiation of empiric antibiotic therapy. Importantly, antibiotics should not be excessively delayed while awaiting obtaining these samples in patients who are critically ill. The initial step in choosing antibiotic therapy for suspected HAP is determination of the patient's risk of infection with an MDR pathogen (**Fig. 9.3**). Knowledge of local microbiologic data including pathogens and sensitivity will assist with proper selection of antimicrobials. One of the consequences of increasing antimicrobial resistance is an increased probability of inappropriate initial empiric treatment. Inappropriate or delayed empiric treatment results in a substantial excessive attributable mortality

and hospital length of stay. Prompt institution of appropriate empiric therapy based on risk stratification for the presence of a possible MDR pathogen is crucial to improving patient outcome in HAP.

Initial therapy should be administered intravenously, with a switch to the enteral route of administration in selected patients with a good clinical response and a functional gastrointestinal tract. Combination therapy should be used initially if patients are at high risk of being infected with an MDR pathogen. Monotherapy is appropriate for those patients deemed to be low risk. Duration of therapy should be based on clinical response and may often be safely terminated after 8 days, provided the etiologic pathogen is not *Pseudomonas aeruginosa*, which requires a longer course of 15 days. Clinical improvement usually takes 48 to 72 hours, and therapy should not be changed during this time unless there is a rapid clinical decline. The responding patient should have therapy tailored to the most focused regimen possible on the basis of microbiologic studies. The nonresponding patient should be evaluated for drug-resistant organisms, complications of pneumonia (e.g., parapneumonic effusion or empyema), extrapulmonary sites of infection, or noninfectious causes of symptoms and signs of pneumonia (e.g., drug fever with drug-induced lung injury).

Respiratory Failure

Respiratory failure denotes either the inability to maintain normal tissue oxygen transport or the normal excretion of carbon dioxide. Clinically, respiratory failure is usually diagnosed by levels of arterial PO_2 and PCO_2, although the levels that constitute the threshold for respiratory failure are arbitrary. An arterial PO_2 of less than 60 mm Hg or an arterial PCO_2 of greater than 45 mm Hg generally indicates significant respiratory compromise in patients without preexisting lung disease. The diagnostic and therapeutic approach to the patient with respiratory failure is dictated by the underlying mechanism of abnormal gas exchange (**Table 9.16**).

Five basic pathophysiologic mechanisms cause acute hypoxemic respiratory failure. These include hypoventilation, V/Q abnormalities, shunt, diffusion limitation, and low inspired fraction of oxygen. All of these causes of hypoxemia are responsive to supplemental oxygen except shunt. Hypoventilation is usually due to depression of respiratory drive at the

level of the central nervous system as a result of drug therapy or the residua of anesthesia as described above. V/Q abnormalities are common causes of hypoxemic respiratory failure and result from such conditions as airflow obstruction from asthma and chronic obstructive pulmonary disease and pulmonary thromboembolic disease. Shunt is frequently caused by pneumonia and atelectasis. Diffusion limitation and low inspired fraction of oxygen are uncommon causes of hypoxemia in the absence of chronic lung disease and high altitude, respectively.

TABLE 9.16 Pathophysiologic Mechanisms of Hypoxemic Respiratory Failure and Ventilatory Failure

HYPOXEMIC RESPIRATORY FAILURE	VENTILATORY FAILURE
Hypoventilation	Insufficient respiratory drive
V/Q abnormalities (shunt, dead space)	Excessive respiratory workload
Venous admixture	Respiratory pump dysfunction
Diffusion limitation	
Low inspired fraction of oxygen	

The spectrum of causes of hypoxemia often can be narrowed according to the appearance of the chest radiograph. A simplified approach to chest radiographic interpretation for other than the pulmonary or critical care medicine specialist involves classifying the pulmonary parenchyma as generally white or black (normal). The causes of a white chest radiograph include atelectasis, pneumonia, pulmonary edema, and acute lung injury. Further characterization of a white chest radiograph includes the description of the radiographic opacities as diffuse or localized. Diffuse infiltrates are commonly associated with hydrostatic pulmonary edema that is due to cardiac pump failure, nonhydrostatic pulmonary edema that is due to lung injury from a variety of causes (e.g., aspiration or pancreatitis), or atypical pneumonias that are rarely hospital acquired (e.g., influenza, *Mycoplasma*, or *Chlamydia pneumoniae*). Focal infiltrates are suggestive of atelectasis or pneumonia. The causes of a black or a normal chest radiograph include pulmonary thromboembolic disease, microatelectasis, exacerbation of underlying obstructive lung disease, intracardiac or pulmonary arteriovenous right-to-left shunt, and a low cardiac output state. In the case of a black or white chest radiograph, information obtained from the history and physical examination may be crucial for determining the nature of further diagnostic testing to pinpoint the etiology of respiratory failure.

Ventilatory failure, the inability to maintain an appropriate arterial PCO_2, is caused by three major mechanisms: insufficient respiratory drive, excessive respiratory workload including increased dead space, or respiratory pump dysfunction. Insufficient ventilatory drive is usually due to drug therapy or the residua of anesthesia as stated above but may also be due to a primary central nervous system disorder (such as stroke, intracranial hemorrhage, and obesity-hypoventilation syndrome) or due to toxic-metabolic encephalopathy as a result of a wide variety of underlying conditions. Increased ventilatory workload may result from increased CO_2 production, increased dead space, altered respiratory mechanics (increased airway resistance or decreased pulmonary compliance), or compensation for metabolic acidosis.

Increased CO_2 production is a by-product of overall increases in metabolism. Fever, delirium with marked agitation, severe sepsis, overfeeding, and hyperthyroidism are examples of clinical states associated with increased CO_2 production. Increased dead space is a hallmark of severe chronic obstructive pulmonary disease; in patients with normal lung function, increased dead space is usually due to either pulmonary thromboembolic disease or a change in respiratory pattern. Rapid shallow breathing as a result of the physiologic changes is induced by anesthesia, and upper abdominal surgery produces an increase in the proportion of ventilation to the anatomic dead space and a decrease in effective alveolar ventilation. In addition to anesthesia and upper abdominal surgery as causes of respiratory pump dysfunction in the postoperative patient, any other cause of respiratory muscle weakness may result in ventilatory failure. Endocrine disorders, such as myasthenia gravis or hypothyroidism, as well as electrolyte abnormalities, such as hypophosphatemia, are examples of conditions that could significantly contribute to respiratory muscle weakness and pump dysfunction.

Treatment of acute respiratory failure depends on the underlying cause and is best accomplished in an appropriately

monitored environment, such as the intensive care unit with the aid of a specialist in critical care medicine, as mechanical support of ventilation—via either conventional invasive mechanical ventilation through an endotracheal tube or noninvasively through a tight fitting nasal or facial mask—may be necessary (**Fig. 9.4**). Management of mechanical ventilation and its consequences has acquired a level of complexity that mandates subspecialist care and is beyond the scope of this chapter.

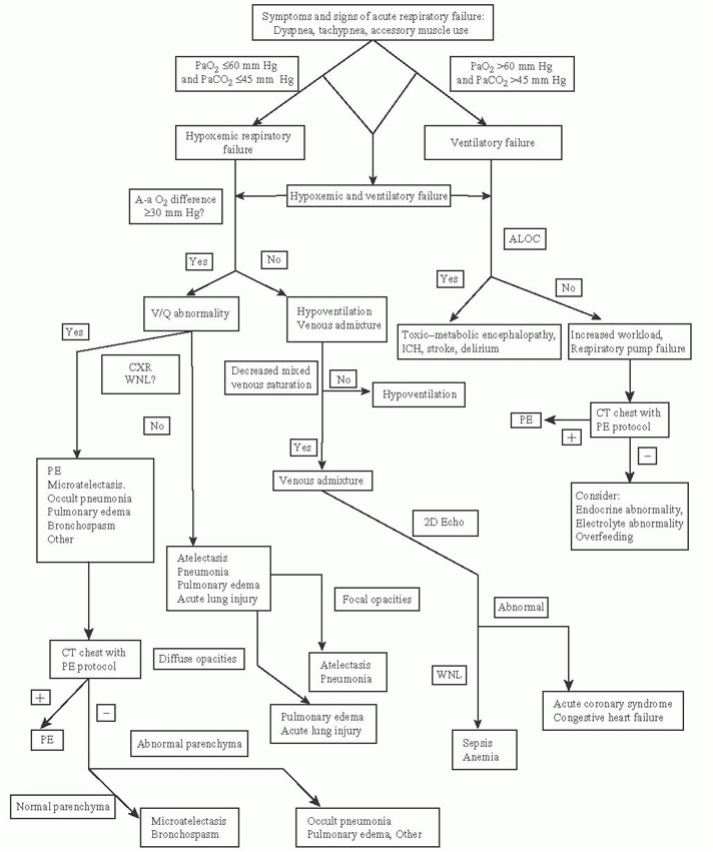


FIGURE 9.4 Algorithm for the evaluation of respiratory failure. ALOC, altered level of consciousness; CT, computed tomography; CXR, chest x-ray; ICH, intracranial hemorrhage; PE, pulmonary embolism; WNL, within normal limits.

POSTOPERATIVE CARE OF THE URINARY BLADDER

The most common postoperative problem in the female bladder is atony caused by overdistention and the reluctance of the patient to initiate the voluntary phase of voiding. After abdominal/pelvic surgery, the patient is often unwilling to contract the abdominal muscles to produce sufficient intraabdominal pressure against the dome of the bladder to initiate the voiding reflex. After anterior colporrhaphy, spasm, edema, and tenderness of the pubococcygeal muscles may obstruct the process of voiding. The operative trauma from plication of the pubovesicocervical fascia causes edema of the urethral wall and submucosa, especially at the urethrovesical junction, thus contributing to the urinary obstruction.

For spontaneous voiding to occur, the parasympathetic function of the bladder detrusor must be coordinated with the voluntary motor function of the abdominal wall and the levator muscles. In the past, it was customary to insert an indwelling urethral catheter for 5 or more days after vaginal plastic surgery. Although this technique is still used in many clinics, a suprapubic catheter has proved to be an effective alternative. The suprapubic technique was developed and introduced to the gynecologic literature in 1964. When inserted at the time of surgery, the suprapubic Silastic tube eliminates the necessity for repeated bladder catheterization until spontaneous voiding occurs. Although used preferentially after anterior vaginal colporrhaphy, suprapubic bladder catheterization also is useful when the need for prolonged bladder drainage is anticipated, such as after radical Wertheim hysterectomy. A suprapubic catheter also can be inserted when a Marshall-Marchetti-Krantz urethral suspension is performed.

The procedure for suprapubic bladder drainage is performed either before or after the operative procedure. Catheter placement consists of insertion of a 12F Silastic (silicone) catheter into the bladder through a needle trocar (Fig. 9.5). A 12F pigtail (Bonanno) Teflon catheter and other modifications also have been used by many surgeons. The bladder is filled with 300 mL of sterile water, and the needle trocar is inserted through the surgically cleaned anterior abdominal wall about 2 cm above the symphysis pubis. When the stylet is removed from the trocar, clear fluid should pass from the bladder under pressure. About 10 cm of the suprapubic catheter is threaded through the trocar, after which the trocar is removed by sliding it over the indwelling tube. The opposite end of the Silastic catheter is connected to a sterile 1-L drainage bottle or to a sterile closed drainage urinometer bag. The tubing should be filled with fluid at all times and should be anchored to the skin with silicone paste or sutured to the skin to avoid accidental removal. A two-way stopcock is inserted between the catheter and drainage tubing for easy opening and closing of the system. The system is not irrigated unless there is plugging of the bladder catheter and failure of drainage.

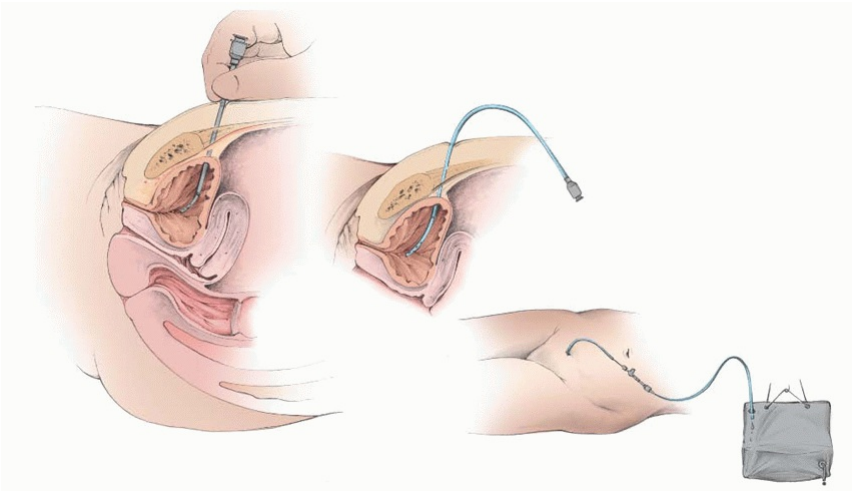


FIGURE 9.5 Method of inserting a suprapubic tube into the bladder through a needle, with resultant drainage into a bottle. Suprapubic catheterization avoids the trauma to the urethra caused by repeated catheterization of an indwelling catheter.

Alternatively, at the Emory University Hospital, a Foley catheter is placed through the abdominal wall. After placing a Kelly clamp through the urethra and elevating the dome of the bladder, an incision is made in the abdominal wall superior to the Kelly clamp. A 14F Foley catheter is then pulled into the bladder and connected to gravity drainage. If a suprapubic urethropexy is performed, a 2- to 3-cm opening is made in the dome of the bladder, and the bladder is inspected to ensure that no sutures penetrated the mucosa. The Foley catheter is placed in the bladder and sutured in place with a no. 2.0 absorbable purse-string suture. Using the preceding techniques for insertion of a suprapubic Foley catheter, we have decreased the frequency of catheter obstruction. Seven to ten days postoperatively, the suprapubic catheters are clamped and postvoid residuals are evaluated. Patients with more than 100 mL of residual urine after spontaneous voiding require an extended period of catheterization. If the urinary residual is less than

100 mL on two successive voidings of more than 200 mL each, the suprapubic catheter can be removed. In many patients who require prolonged catheterization following radical hysterectomy or pelvic support surgery, intermittent self-catheterization using a clean but not sterile technique has become favored by many gynecologists. We believe, as do other authors, that prophylactic antibiotics given during the use of an indwelling bladder catheter are ineffective in preventing urinary tract infection. Although urinary tract symptoms may be delayed with the use of prophylactic antibiotics, it is our experience that the incidence of infection is unchanged and that a subsequent urinary tract infection may result from resistant organisms that are more difficult to treat later. Therefore, we prefer to treat only patients who have significant bacteriuria and pyuria, which includes about 10% to 15% of the patients with suprapubic drainage.

POSTOPERATIVE GASTROINTESTINAL TRACT MANAGEMENT

Dysfunction of the gastrointestinal tract remains a challenge in postoperative management. Each patient should be treated as an individual and not placed on a standard protocol for advancing diets. Patients who have had uncomplicated surgery may be given a regular diet on the first postoperative day if bowel sounds are present, if abdominal examination reveals no distention, and if the patient is no longer nauseated from her anesthesia. Seriously ill or malnourished patients or patients requiring extensive bowel surgery may benefit from preoperative and postoperative parenteral nutrition.

It is important to differentiate between postoperative ileus and postoperative obstruction (**Table 9.17**) if proper therapy is to be initiated promptly with beneficial results. The distinction may be difficult. This is because partial bowel obstruction is often accompanied by a secondary ileus as part of the clinical picture. Only by close clinical monitoring of the bowel sounds, serial abdominal radiographic studies, and frequent white blood cell counts can one clearly separate these two postoperative complications. Adynamic ileus is the more common clinical entity, a fact that may mislead the surgeon into a false sense of security unless he or she remains acutely aware of the distinguishing features of intestinal obstruction. Serial monitoring of the white blood cell count and differential count is an important method for differentiating between bowel obstruction and paralytic ileus. A key feature of advancing bowel obstruction is necrosis of the bowel wall, which causes progressive leukocytosis, along with distention and peritonitis. The most common gynecologic disease process associated with both ileus and intestinal obstruction is severe pelvic inflammatory disease (PID). Notoriously acute exacerbation of PID or rupture of a pelvic abscess is associated with prolonged ileus. Occasionally, fibrous adhesions form, and secondary bowel obstruction occurs. Postoperative pelvic peritonitis from any cause, including cellulitis resulting from hematoma formation and secondary infection of the vaginal cuff, is often associated with ileus, whereas intestinal obstruction only rarely results from such a complication.

TABLE 9.17 Differential Diagnosis Between Postoperative Ileus and Postoperative Obstruction

CLINICAL FEATURE	POSTOPERATIVE ILEUS	POSTOPERATIVE OBSTRUCTION
Abdominal pain	Discomfort from distention but not cramping pains	Cramping progressively severe
Relation to previous surgery	Usually within 48-72 h of surgery	Usually delayed, may be 5-7 d for remote onset
Nausea and vomiting	Present	Present
Distention	Present	Present
Bowel sounds	Absent or hypoactive	Borborygmi with peristaltic rushes and highpitched tinkles
Fever	Only if related to associated peritonitis	Rarely present unless bowel becomes gangrenous
Abdominal radiographs	Distended loops of small and large bowels; gas usually present in the colon	Single or multiple loops of distended bowel (usually small bowel) with air-fluid levels
Treatment	Conservative with nasogastric suction, enemas, cholinergic stimulation	Conservative management with nasogastric decompression Surgical exploration

Total Parenteral Nutrition

Nutritional support has proved efficacious in patients undergoing major surgery and in patients with impaired bowel function, inadequate oral intake, or cancer. A few patients require total parenteral nutrition (TPN) for prolonged periods secondary to their inability to obtain adequate calories orally. Parenteral nutrition may be administered through a peripheral or central access, depending on the patient's initial nutritional status and the time required on TPN.

Hospitalized patients may require TPN for disease processes such as gastrointestinal tract obstruction, prolonged ileus, short-bowel syndrome, radiation enteritis, intra-abdominal abscess, pancreatitis, regional enteritis, and enterocutaneous fistula. A patient with any condition that prevents oral intake of adequate amounts of food for more than 7 to 10 days probably requires central parenteral nutrition. Because it is much easier to maintain an adequate nutritional state than to improve a poor one, the decision to use TPN should not be delayed.

TPN is not without complications. Many of these pertain to the need for central venous access. Catheter tip infection is one of the more frequently encountered problems. Meticulous aseptic technique when placing the central venous catheter and adherence

to aseptic technique when using the catheter minimizes the risk of infection. Antibiotic-coated central venous catheters have been around for more than a decade and are coming more into favor as compelling data surface suggesting decreased infection rates with their use. At the Emory University Hospital, central venous catheters coated with chlorhexidine and silver sulfasalazine are being used. Other potential problems with central venous catheters include catheter or air embolism and pneumothorax or hemothorax. Total parenteral nutrition itself can cause fluid overload, electrolyte abnormalities, or metabolic disturbances.

Patient Evaluation

A complete medical history and physical examination must be obtained before parenteral nutrition is initiated. Particular attention should be paid to identifying patients with cardiovascular or renal disease, hyperlipidemia, diabetes, and thyroid disease. Total parenteral nutrition modification can include decreasing or eliminating fat emulsion in patients with severe cardiovascular disease or hyperlipidemia, administering low-nitrogen TPN to patients with renal failure, and increasing the insulin dosage in patients with diabetes mellitus.

The patient's degree of malnutrition should be assessed by taking measurements of several physical indicators, such as actual body weight (ABW) and ideal body weight (IBW), usual body weight (UBW; preillness), creatinine-to-height index, triceps skin fold thickness (TSFT), and arm circumference (AC). The arm muscle circumference (AMC) is calculated and used as an index of nutritional status.

AMC = AC-(TSFT × 3.14)

Fat stores are reflected in the triceps skin fold measurement, whereas somatic proteins are evaluated by measuring muscle mass, such as the AMC. The Frisancho standards (1984) are used to interpret body weight (kilograms), triceps (millimeters), and bone-free arm muscle area (square centimeters). Patients found to be in the 5th to 10th percentiles are severely malnourished and require an anabolic environment. Weight loss is considered significant when the (UBW – ABW)/UBW × 100 is >10%. Weight loss during starvation occurs at a rate of 0.4 kg/d. Survival also is compromised when the ABW falls below the 70th percentile of the IBW. In addition to the preceding physical measurements, a thorough evaluation of chemical indicators is required (**Table 9.18**) before initiating TPN. Extensive monitoring is required while the patient is receiving TPN (**Table 9.19**).

TABLE 9.18 Pretreatment Screening

LABORATORY EVALUATION
Complete blood count with differential
Prothrombin time/partial thromboplastin time
Electrolytic panel
Chemistry panel
Albumin
Transferrin
Total lymphocyte count
Triglycerides
Magnesium
Phosphorus
Copper
Zinc
Selenium

TABLE 9.19 Treatment Monitoring

TEST	FREQUENCY
Electrolyte panel	Twice weekly
Chemistry panel	Weekly
Magnesium	Weekly
Transferrin	Weekly
Triglycerides	Monthly, or as needed
Zinc	Monthly, or as needed
Copper	Monthly, or as needed
Selenium	Monthly, or as needed

The physical and chemical measurements of malnutrition are subject to many influences during illness and should be treated as confounding variables. For example, albumin values less than 3.2 g/dL are frequently used to indicate malnutrition. Starker and colleagues observed that in hospitalized patients, albumin and body weight measurements in conjunction provided better indications of sodium balance and extracellular fluid volume. In addition, albumin serum levels are required for maintenance of the intravascular colloid oncotic

pressure and as a carrier protein.

The half-life of albumin is 20 days and thus reflects a depletion of visceral proteins of at least 3 weeks' duration. Transferrin, with a half-life of 8 to 9 days, provides the clinician with a measurement of recent protein status changes. Because transferrin is required to bind Fe^{2++}, its level is affected by intravascular iron status and can increase during pregnancy, in patients with hepatitis, and in patients receiving estrogen supplementation. Serum protein content can be reduced in protein-losing enteropathy, nephropathy, chronic infections, uremia, and during catabolism. Transferrin reflects recent losses and remains a better indicator of protein status and change than is albumin. Total lymphocyte counts of less than 1,500 mL are indicative of an immunocompromised patient. Immunologic skin testing for recall antigens and total lymphocyte counts has been correlated with both nutritional status and morbidity and mortality. Its usefulness in the assessment of nutritional status is limited to confounding variables such as cancer, side effects of cancer treatment protocols, stress of trauma or surgery, and infection. Phosphorus and the trace elements are thoroughly evaluated before initiating TPN and during TPN because they are often depleted with many disease states and are required when alimenting (**Tables 9.7**, **9.8**, and **9.18**,9.19,9.20 and **9.21**).

Nutritional Requirements

Total parenteral nutrition consists of six components: carbohydrates, fat, protein, electrolytes, vitamins, and trace elements. The Harris-Benedict basal energy expenditure (BEE) accounts for two thirds of the total daily energy requirements, with the remaining one third obtained from protein. Daily requirements for protein are between 1.5 and 2.5 g/kg/d. Patients receiving TPN who are severely malnourished and stressed require larger amounts of protein daily.

The BEE is calculated as follows:

BEE(kcal/d) = 655 + 9.56(wt) + 1.85(ht)-4.68(age)

TABLE 9.20 Design of Parenteral Nutrition Programs: Examples

Nonobese patient weighing 60 kg; assume basal Harris-Benedict equation estimate of daily caloric requirements of 1,250 kcal/d

Patient characteristics

1. Euvolemic, normal urine output, and no unusual gastrointestinal losses; therefore, appropriate initial estimate of daily fluid requirement is 30 mL/kg body weight
2. Moderately stressed with normal renal and hepatic function; therefore, appropriate to provide 1.2 g protein per kg body weight
3. Nonobese; therefore, appropriate to provide Harris-Benedict estimate plus 20% for calories, i.e., 1,250 kcal plus 20% = 1,500 kcal

Program design

1. Fluid requirement: 30 mL × body weight; 30 × 60 = 1,800 mL
2. Caloric requirement: Harris-Benedict estimate plus 20%; 1250 kcal + 250 kcal = 1,500 kcal
3. Protein requirement for moderately stressed patient: 1.2 g/kg body weight; 60 × 1.2 = ˜70 g protein. 70 g protein × 4 kcal/g protein = 280 kcal
4. Fat requirement: 30% of total calories; 30% × 1,500 kcal = 450 kcal
5. Carbohydrate requirement: caloric requirement minus the sum of protein and fat calories; 1,500 – (280 + 450 kcal) = 770 kcal. 770 kcal carbohydrate – kcal/g carbohydrate (3.4) = ˜225 g carbohydrate
6. Therefore, consider the following parenteral nutrition formula: 1.5 L amino acids, 5% dextrose, 15%; plus 250 mL of 20% fat emulsion, which provides 1,750 mL, 1565 kcal, 75 g protein, 225 g carbohydrate, and 500 fat calories. Note that 5% amino acids equals 50 g protein per liter
7. If institution uses three-in-one admixture (amino acids plus dextrose plus fat in one container) and stock solutions of 10% amino acids, 70% dextrose, and 20% lipid, a comparable parenteral nutrition program would be 1.5 L amino acids, 5%; dextrose, 15%; fat, 3.5%

Similar patient characteristics except patients volume-expanded

1. Consider the following fluid-restricted parenteral nutrition formula: 1 L of amino acids, 7%; dextrose, 20%; plus 250 mL 20% fat emulsion, which provides 1,250 mL, 1,460 calories, 70 g protein, 200 g carbohydrate, and 500 fat calories

Height is in centimeters; weight is in kilograms.

Once the patient has reached the estimated daily calorie goal, a 24-hour nitrogen balance study is performed by obtaining a 24-hour urine collection and an am electrolyte panel. If a large quantity of fluid from the nasogastric tube, ileostomy, fistula, or wound is present, this also should be collected and sent for nitrogen measurements.

N_2 (g) balance = N_2 (g) in – N_2 (g) out

Adding 4 to the N_2 out value accounts for nitrogenous losses in the stool and skin. This does not include an estimate of the losses from the gastrointestinal tract and wound, as previously described.

N_2 (g) balance = N_2 (g) in × [N_2 (g) out + 4]

N_2 (g) out = urine volume (mL) × urine urea N_2 (mg / dL)

N_2 (g) in = amino acids per day / 6.24

6.24 = g protein / g nitrogen

Patients with normal renal and liver function are started on standard total hyperalimentation solution (THAS) (**Table 9.18**). Each liter provides a total of 1,020 kcal, including 41 g of amino acids and 250 g of dextrose. The osmolality of this solution is 1,850 mOsm, which therefore necessitates a central venous access. The calories-to-nitrogen ratio of this solution is 157:1 and is optimal in nonstressed patients. The addition of lipids also is effective in promoting a positive nitrogen balance. The total daily sodium concentration should be equivalent to that of normal saline (150 mEq/L). This can be altered to accommodate patients who require sodium restriction or loading. **Table 9.22** outlines the recommendation for daily electrolyte requirements. It should be noted that acetate serves as a precursor to bicarbonate, because the latter is not compatible in the THAS solution. Multivitamins are added daily to 1 L of THAS, whereas trace elements are divided equally in the volume to be infused during a 24-hour period. The recommended daily allowances for both fat- and water-soluble vitamins are outlined in **Table 9.23**.

Blood glucose levels in patients receiving TPN should be between 100 and 200 mg/dL. A minimum of 10 IU should be added to each liter when required. This permits about 50% to

adhere to the plastic tubing. This can be supplemented with subcutaneous doses of regular insulin to obtain the desired blood glucose level. About one half to two thirds of the previous day's requirements are added in divided doses to the THAS solutions.

Intravenous lipids provide a nonprotein source of energy and serve as a source of essential fatty acids. Ten percent fat emulsions (550 kcal/500 mL) and 20% fat emulsions (1,000 kcal/500 mL) are commercially available. In patients receiving standard TPN, 500 mL of 10% fat emulsion are infused twice weekly at a rate of 42 mL/h. However, when fat emulsion is used with peripheral THAS, the patient requires 2 L of peripheral THAS and 1 L of 10% fat emulsion daily. Twenty percent fat emulsions also can be used for calories in patients with glucose intolerance or patients who require a decreased protein-calorie percentage.

TABLE 9.21 Trace Minerals

MINERALS AND LEVELS	DEFICIENCY		TOXICITY	
	SYMPTOMS	ETIOLOGY	SYMPTOMS	ETIOLOGY
Zinc, 55-150 mg/dL	Diarrhea, mental depression, alopecia, night blindness, dermatosis, impaired taste, hypogonadism, impaired wound healing	Gastrointestinal (failure of ingestion, absorption, retention) Large wounds Protein-energy malnutrition Cancer	Vomiting Diarrhea Neurologic damage ("zinc shakes") Metal fume fever	Increased ingestion from galvanized containers
Selenium, 90-150 µg/dL (synergism with vitamin E)	Myositis with muscle weakness Cardiomyopathy with arrhythmias and congestive heart failure	Unsupplemented TPN	Liver cirrhosis Alopecia Pathologic loss of nails Dermatitis	Increased ingestion (rare)
Chromium, 50-200 µg/d	Neuropathy Encephalopathy New insulin-dependent diabetes mellitus	Unsupplemented TPN Increased renal loss secondary to injury Gastrointestinal losses	Respiratory Lung cancer	Workers manufacturing products containing hexavalent chromium
Phosphorus, 3.0-4.5 mg/dL	Nausea, vomiting, anorexia, dysarthria, paresthesia, hemolytic anemia, peripheral neuropathy, respiratory depression, congestive heart failure, renal glycosuria	Gastrointestinal (failure of ingestion, absorption, retention) Cellular anabolism Respiratory or metabolic alkalosis $Al(OH)_3$ antacids Alcoholism	Neurotoxicity secondary to compensatory hypocalcemia	Renal failure Hypoparathyroidism
Magnesium, 136-145 mEq/L	Nausea, vomiting, muscle weakness, lethargy, tetany, muscle tremor, personality changes	Gastrointestinal (failure of ingestion, absorption, retention) Cellular catabolism, acidosis, K^+ depletion Glomerular dysfunction	Hyporeflexia, lethargy, respiratory depression, cardiac arrest	Magnesium supplementation in patients with renal compromise
Copper, 70-155 µg/dL	Hypochromic anemia not responsive to iron, neutropenia	THAS without copper or high amino acids Gastrointestinal (failure of ingestion, absorption, retention) Pregnancy, lactation (increased requirements) Renal losses	Jaundice-hepatic necrosis Intravascular hemolysis Gastric hemorrhage Tremors, choreoathetoid movements, dementia, rigidity, dysarthria	Iatrogenic Wilson disease Absorption of copper nitrate salves in burn patients

THAS, total hyperalimentation solution; TPN, total parenteral nutrition.

Patients deficient in fatty acids present with dermatitis, hemolytic anemia, thrombocytopenia, elevated liver enzymes, and poor wound healing.

To improve glucose tolerance, the first liter should be started at a rate of 42 mL/h. On the second day, the solution can be increased to 84 mL/h; on day three, it can be increased to 124 mL/h. If the patient is unable to tolerate this schedule, increments can be decreased to 21 mL/h each day. Treatment monitoring is outlined in **Table 9.19**. Total nitrogen balance should be recalculated if there is a marked change in the patient's condition or in the parenteral nutrition administered.

Recent data in the surgical literature suggest that supplementing TPN with glutamine dipeptides improves nitrogen balance, preserves intestinal permeability and absorption, and improves recovery of lymphocytes. These authors also demonstrated a shorter hospital stay in postoperative patients receiving glutamine dipeptide-enriched TPN compared with controls receiving TPN alone.

Initiating Total Parenteral Nutrition

Safe venous access is required for initiating TPN. A reliable intravenous catheter should be placed into a large central vein with the catheter tip located so that blood flow dilutes the highly concentrated nutritional fluids. The insertion site also should allow easy fixation of the catheter at the entrance site, minimum catheter movement during body

movements, and easy dressing changes. A subclavian vein approach satisfies the requirements for safe catheter placement, but neither internal jugular vein nor antecubital fossa placement is optimal. The internal jugular vein should be used only if the subclavian approach has failed. Movement of the head and neck results in an increased incidence of occluding venous access when the internal jugular vein has been cannulated.

TABLE 9.22 Daily Electrolyte Requirements for Parenteral Nutrition

ELECTROLYTE	DOSAGE (mEq/d)
Sodium	60-150
Potassium	60-240
Phosphate	30-45
Calcium	10-15
Magnesium	8-26
Acetate	80-120
Chloride	60-150

Anatomy of Infraclavicular Subclavian Vein

In 1952, Aubaniac, a French physician, was among the first to advocate use of the subclavian vein for intravenous infusions. Wilson and colleagues cannulated the superior vena cava through a percutaneous puncture of the subclavian vein. They reported a high percentage of successful cannulations and a low incidence of complications.

As Figure 9.6 shows, the subclavian vein is located within the costoclavicular-scalene triangle, which is bounded anteriorly by the medial end of the clavicle, posteriorly by the upper surface of the first rib, and laterally by the anterior scalene muscle. The anterior scalene muscle separates the subclavian vein from the subclavian artery, which lies beneath and along the lateral aspect of the muscle. The subclavian vein is covered by 5 cm of the clavicle medially and joins the internal jugular vein near the medial border of the anterior scalene muscle to form the innominate vein. The innominate vein descends behind the sternum and joins with the opposite innominate vein to form the superior vena cava. The subclavian vein, which is about 3 or 4 cm long, continues as the axillary vein below the clavicle en route to the axilla. Several other significant structures occupy this region. The phrenic nerve courses across the anterior surface of the anterior scalene muscle near its attachment to the first rib and courses medially to lie posterior to the subclavian vein. It can be injured if the posterior wall of the vessel is penetrated. The internal thoracic nerve and apical pleura are in contact with the posterior surface of the subclavian vein at its junction with the internal jugular vein. The roots of the brachial plexus formed by the fifth, sixth, seventh, and eighth cervical and first thoracic nerves lie lateral to the anterior scalene muscle on the lateral side of the subclavian artery. If a cannulating needle is directed too far laterally, the brachial nerve plexus could be injured or the subclavian artery could be punctured. The thoracic duct on the left side and the lymphatic duct on the right cross the anterior scalene muscle on either side of the thorax to enter the superior aspect of each subclavian vein near its junction with the internal jugular vein. These lymphatic vessels are rarely encroached on during subclavian catheterization.

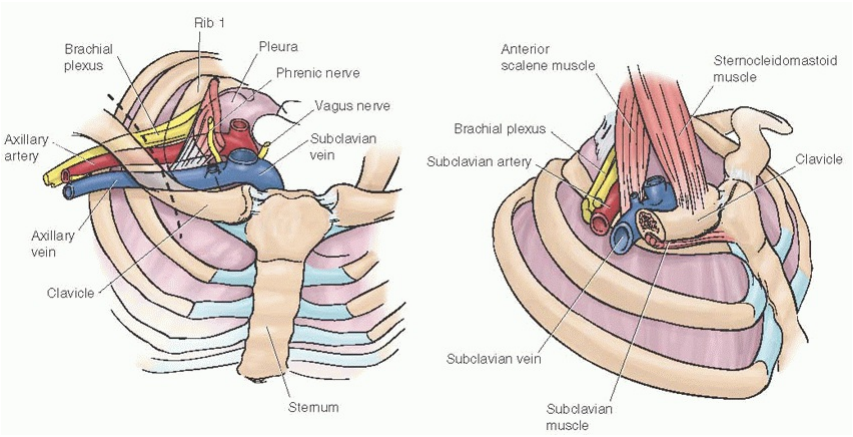


FIGURE 9.6 Anatomic relations of the subclavian vein. The broken line represents the location of the transverse section for lateral view.

Subclavian Catheter Placement

As illustrated in Figure 9.7A, the subclavian catheter is inserted with the patient in the supine position, with the foot of the bed elevated 6 to 12 inches to increase the pressure in the subclavian vein and produce venous distention. After meticulous

aseptic preparation of the skin with povidone-iodine (Betadine), the skin and subcutaneous tissues are infiltrated with a 1% solution of lidocaine (Xylocaine) if the patient is awake. The point of needle insertion is about 1 cm below the junction of the inner and middle third of the clavicle. Most central venous catheter units include an external introducer catheter (Teflon) and an internal (silicone) infusion catheter. The outer Teflon sheath accommodates a no. 12 needle, which fits snugly into and protrudes beyond the end of the Teflon catheter. The needle and sheath are introduced into the skin with the shaft of the needle held almost parallel with the anterior chest wall (Fig. 9.7A). The needle is directed medially and advanced along the undersurface of the clavicle. It is not necessary to scrape the posterior surface of the clavicle to ensure that the pleura is protected from puncture. By applying suction constantly, the needle passes beneath the skin and immediately aspirates dark red blood, which confirms entry into the vein. If the vein is not entered, the needle is withdrawn

and readvanced in a similar manner but in a slightly more cranial or caudal direction. Use of real-time sonography has been shown to increase the likelihood of successful subclavian catheter placement. As soon as a free flow of blood is obtained, the introduced Teflon sheath is advanced far enough to be certain that it is securely placed within the vein. The sheath is held in place by the connector, the finger is placed over the end of the needle to prevent air embolism, and the internal needle is replaced (Fig. 9.7B) by the silicone infusion catheter that accompanies the central venous pressure kit (Fig. 9.7C). A thin wire stylet inside the infusion catheter allows the catheter to be advanced easily; occasionally, the stylet must be withdrawn slightly to advance the catheter as far as possible into the innominate vein and superior vena cava. The silicone infusion catheter is advanced until the attached connector can be securely wedged into the connector of the Teflon sheath (Fig. 9.7C). After the infusion catheter is connected to an intravenous fluid line, the Teflon sheath is carefully withdrawn from the vein, remaining partially in the subcutaneous tissue while leaving an ample length of the infusion catheter in the vena cava (Fig. 9.7D). A suture sleeve on the introducer sheath is slid down to the puncture site and sutured to the skin (Fig. 9.7E). The tip of the catheter is preferably positioned in the superior vena cava and should not be advanced into the right atrium or ventricle, where it could cause accidental trauma to the heart wall or cardiac arrhythmias. To ensure its

continued sterility and proper function, the subclavian vein catheter should not be used to replace fluids or withdraw blood for laboratory studies, if it is at all possible to avoid these uses. A central venous line for hyperalimentation is an exception to this rule. The dressing should be changed daily and the catheterization site cleaned with povidone-iodine or a similar antimicrobial solution.

TABLE 9.23 Recommended Dietar

PATIENT PARAMETERS					FAT-SOLUBLE VITAMINS									
AGE (Y)	**WEIGHT[b] (KG)**	**(LB)**	**HEIGHT[b] (CM)**	**(IN)**	**PROTEIN, G**	**VITAMIN A, > MG RE[c]**	**VITAMIN D, IU[d]**	**VITAMIN E, IU[e]**	**VITAMIN K, >MG**	**ASCORBIC ACID (C), MG**	**THIAMINE (B_1), MG**	**RIBOFLAVIN (B_2), MG**	**NIACIN (B_3), MG**	**PYRI (B_6**
Females 11-14	46	101	157	62										
15-18	55	120	163	64	44	800	400	12	55	60	1.1	1.3	15	
19-24	58	128	164	65	46	800	400	12	60	60	1.1	1.3	15	
25-50	63	138	163	64	50	800	200	12	65	60	1.1	1.3	15	
51 +	65	143	160	63	50	800	200	12	65	60	1.0	1.2	13	

[a]The allowances, expressed as average daily intakes over time, are intended to provide for individual variations among most normal persons as they live in the United States unc have been less well defined.

[b]Weights and heights of reference adults are actual medians for the U.S. population of the designated age, as reported by National Health and Nutrition Examination Survey **II** (N these figures does not imply that the height-to-weight ratios are ideal.

[c]Retinol equivalents. 1 retinol equivalent = 1 mg retinol or *6* mg β-carotene.

[d]As cholecalciferol. 10 mg cholecalciferol = 400 IU of vitamin D.

[e]α-Tocopherol equivalents. 1 mg D-α-tocopherol = α-TE = 1.49 IU.

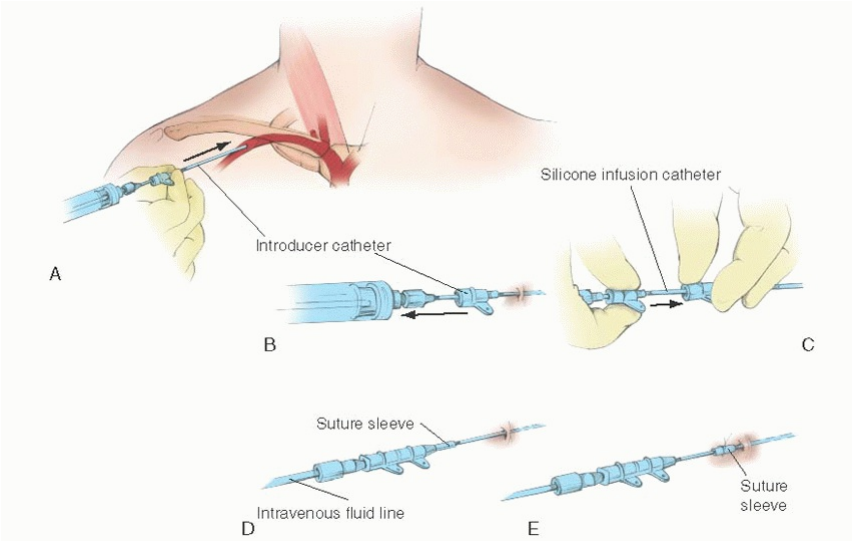


FIGURE 9.7 Insertion of subclavian catheter for monitoring central venous pressure. A: After locally anesthetizing the puncture site, the needle with overlying introducer catheter is directed medially between the first rib and the clavicle at the junction of the middle and inner third of the clavicle. The needle is held parallel to the anterior chest wall and advanced along the undersurface of the clavicle. Entry into the vein is evident with aspiration of blood in the attached syringe. B: The needle and syringe are removed from the Teflon sheath, and a finger is held over the end of the open catheter to prevent entry of air. C: The silicone infusion catheter is inserted through the introducer catheter until the two connectors meet and lock firmly. D: The intravenous fluid line is connected to the silicone infusion catheter. E: The suture sleeve is advanced to the skin surface, where the catheter is sutured firmly to the skin.

Cardiac and Respiratory Insufficiency

Patients with congestive heart failure require decreased sodium and decreased total fluid volume. The best solution can be prepared from the most concentrated solutions of glucose, amino acid, and fat available. Fluid-restricted solutions also may be beneficial for patients with respiratory failure, who should receive less total glucose in favor of more fat because the respiratory quotient (CO_2/O_2) of glucose (1.00) is greater than that of fat (0.70) and because excess glucose will increase the load of CO_2 the lungs must excrete. Excessive total caloric intake resulting in fat synthesis from glucose substrate may severely compromise respiratory function because large amounts of CO_2 are released (respiratory quotient 8.0).

Discontinuing Total Parenteral Nutrition

Before discontinuing TPN, the patient should tolerate an enteral diet that provides adequate calories. It is permissible to aliment patients with an enteral diet before decreasing the THAS solution. An abrupt discontinuation of central parenteral nutrition results in rebound hypoglycemia. Our recommendation is to decrease the THAS stepwise to 42 mL/h before discontinuation. Some institutions recommend that the patient receive 10% dextrose for an additional 12 hours once central parenteral nutrition has been discontinued.

Team Approach to Total Parenteral Nutrition

Total parenteral nutrition can now be safely administered to patients in many hospitals because of the existence of a team of physicians, nurses, and health care professionals. Although the composition and exact function of the team members vary between hospitals, most teams consist of a physician, nurse, pharmacist, and nutritionist. The role of the team varies in each institution from consultation to complete management of the patient's nutritional needs. The team approach by either method is highly beneficial because it provides a high concentration of personnel with knowledge, expertise, and interdisciplinary communication at the patient's bedside. Team members can provide continuing education on nutrition therapy, continuously audit and collect quality control data, and investigate ways to improve the safety and efficacy of TPN as a treatment modality. Most teams operate with a standardized protocol that covers patient assessment, catheter insertion techniques, solutions used, and monitoring functions performed.

Enteral Nutrition

Enteral nutrition is preferable to TPN. The old adage “if the gut works, use it,” applies for several reasons. Ease of administration, economic considerations, and decreased number of complications are all advantages of enteral feeding over parenteral nutrition. Several studies have shown TPN and

enteral nutrition equally efficacious in achieving nitrogen balance. Patients with good bowel function should receive enteral feedings. Relative contraindications to enteral feeding include gastrointestinal bleeding, diarrhea, and intestinal obstruction. Small-bore nasal feeding tubes are placed in the stomach, duodenum, or jejunum. An abdominal radiograph should be obtained to confirm placement. Failure to obtain appropriate studies may result in tube placement in the trachea. Alternatively, a gastrostomy or jejunostomy tube could be placed for long-term enteral nutrition (**Table 9.24**). Several products are commercially available (**Table 9.25**). Enteral tube feedings are routinely administered by pump, with either bolus feeds to the stomach or continuous feeds to the small bowel, and should be administered by pump at 25 to 30 mL/h with gastric residuals evaluated every 4 hours. **Table 9.26** shows the essential and nonessential amino acids.

TABLE 9.24 Protocol for Enteral Tube Feeding

Nasogastric route: Use small-bore, flexible tube (8F preferred); obtain radiograph after placement to confirm position.
Elevate the head of the bed at least 30 degrees.
Use feeding pump for continuous feeding.
Begin with full-strength formula at 25-30 mL/h and, if tolerated, increase by 25-30 mL/h at 12-h intervals until desired total volume is reached.[a]
Check gastric residuals every 4 h; if > 100 mL, hold feeding, and repeat at hourly intervals until residuals are <100 mL before resuming feeding.
Irrigate with 30-50 mL of water after each residual check or after any medications are given. (If the patient requires additional free water, use greater volumes of water for irrigation.)
If the patient experiences diarrhea or intestinal cramping, slow rate of feeding or decrease concentration of formula.

[a]When using hypertonic formulas or feeding into the jejunum with a nasojejunal or jejunostomy tube, diluting the formula to one-half or three-quarter strength may improve tolerance initially. The concentration then can be increased after the desired volume is reached.

TABLE 9.25 Commonly Used Adult Commercial Enteral Feeding Formulas[a]

CATEGORY FORMULATION				
POLYMERIC BALANCED	**1.0 KCAL/ML**	**1.2 KCAL/ML**	**1.5 KCAL/ML**	**2.0 KCAL/ML**
≤16% protein	Ensure, Resource, Isocal, Osmolite, Nutren 1.0		Nutren 1.5, Ensure Plus, Resource Plus, Boost Plus, Comply	Nutren 2.0, Deliver, Magnacal
17%-20% protein	Osmolite HN, Isocal HN, Ensure HN, Ultracel, Jevity		Ensure Plus HN	TwoCal HN
≥20% protein	Sustacal, Replete, Promote, Protein XL (22%), Isosource VHN (25%)		TraumaCal	
Modified conventional				
≤16% protein	Peptamen, Reabilan, Vivonex Plus, Criticare HN			
17%-20% protein	Vital HN, Reabilan HN, Alitra Q			
Peptide based	Peptamen (16%), Reabilan (12.5%), Criticare HN (14%), Peptamen VMP (25%), Reabilan HN (17.5%), Alitra Q (21%), Vital HN (16%), SandaSource Peptical (20%)		Crucial (25%)	
Elemental	Tolerex (8%), Vivonex T.E.N. (15%), Vivonex Plus (18%)			

Modified-disease specific (% protein)[b]				
Critical care	Immun-Aid (32%), Impact (22%), Impact/Fiber (22%)	Perative (20%)		
Glucose intolerance	Glytrol (18%), Choice dM (17%), Glucema (16.7%), Diabetasource (20%)			
Hepatic	Travasorb Hepatic (11%), Hepatic-Aid (15%)	Hepatic-Aid II (15%)	NutriHep (11%)	
Malabsorption renal			Lipisorb (17%), Travasorb Renal (7%)	Renal Cal *(6%-9%),* Amin-Aid (4%)
Pulmonary			Nutrivent (18%), Respalor (20%), Pulmocare (16.7%)	
Modular supplements				
Protein	Casec, ProMod			
Carbohydrate fat	Moducal, Polycose Microlipid, MCT oil			

[a]This table includes only a partial listing of commercial products.

[b]Manufacturers market these products as disease specific. The author's use of this designation is intended neither to endorse the manufacturer's claims of special efficacy in the diseases specified nor to deny that the polymeric-balanced or modified-conventional formulas might be appropriate or even superior in these conditions.
MCT, medium-chain triglyceride.

ROUTINE ORDERS

When the patient has fully recovered from the anesthetic and is ready for return to the nursing floor and routine postoperative care, we have found the basic postoperative orders shown in **Figure 9.8** to be useful. They are only a general outline. This list should be expanded to include the special needs of each postoperative patient.

It is imperative that each patient be evaluated before being transferred from the recovery room. If the patient is not ready for transfer, additional efforts are made to stabilize the patient or transfer her to an intensive care bed. On transferring, the frequency of physicians' rounds should be based on the severity of the patient's condition. All patients should be evaluated on the evening of surgery and appropriate documentation made in the chart. A thorough evaluation of the vital signs, catheter drainage (nasogastric, peritoneal, and Foley), and pulmonary status is required, and abdominal examination is performed. Each physician has a desired protocol for postoperative management. The routine orders outlined in this chapter provide

the clinician with a framework to design patient care plans that address the individual patient's requirements. Laboratory and radiographic evaluation of the postoperative patient also is tailored to the individual patient. Unfortunately, many physicians are predominantly concerned with quantitative test values. However, it is just as important to develop a close rapport with the patient, the patient's family, and the nursing staff. Only through good communication can the gynecologic surgeon deliver optimum medical care.

TABLE 9.26 Amino Acids

ESSENTIAL	NONESSENTIAL
Arginine	Alanine
Histidine	Asparagine
Isoleucine	Aspartic acid
Leucine	Cysteine
Lysine	Glutamic acid
Methionine	Glutamine
Phenylalanine	Glycine
Threonine	Proline
Tryptophan	Serine
Valine	Tyrosine

BEST SURGICAL PRACTICES

- Approximately 500,000 hospitalized patients develop DVT and approximately 200,000 PE.
- Ten percent of hospital deaths in the United States are secondary to a PE.
- Tumors up-regulate the production of TF and PAI-1, which promotes coagulation and VTE.
- Anti-factor Xa assay can be used to monitor patients who are anticoagulated with LMWHs.
- General anesthesia is associated with a reduction of FRC by approximately 16%.
- Atelectasis occurs in 15% to 20% of patients undergoing abdominal surgery.
- An arterial PO_2 of less than 60 mm Hg or an arterial PCO_2 of greater than 45 mm Hg indicates significant respiratory compromise in patients without preexisting lung disease.
- Increased CO_2 production is a by-product of increased metabolism (i.e., fever, marked agitation, severe sepsis, overfeeding, and hypothyroidism).
- During stress, urinary nitrogen levels may increase greater than or equal to 20 g in 24 hours, corresponding to a loss of 600 g of hydrated body protein.

BASIC POSTOPERATIVE ORDERS

Patient's Name: _____.

1. Admit to Unit #____
2. Diagnosis:
3. Allergies:
4. Condition:

5a. Vital signs:
- ____ q 15 minutes until stable
- ____ q 2 hours for 24 hours
- ____ q 8 hours, if stable

5b. Notify House Officer (H.O.) if
- BP < 90/60, > 160/100
- Pulse <60, >120
- Temp >38.0°C

6. Activity:
- ____Bed rest
- ____Ambulate
- ____ Other (specify)

7. Diet:
- ____NPO
- ____ Other (specify)

8. Intravenous fluids:
9. Incentive inspirometer q 2 hours while awake
10. Encourage deep breathing
11. Drains:

Type	Location	Drainage
____ Nasogastric	____ Stomach	Low/intermittent suction
____ Peritoneal	____ Pelvis	____ Bulb suction
____ Foley catheter	____ Bladder	____ Gravity

12a. Fluid intake and output chart.
12b. Notify H.O. if urine output <30 cc/h.
13. Pain medication: Specify
- (a) route of administration
- (b) dosage

14. Antiemetic medication: Specify
- (a) route of administration
- (b) dosage

15. Antibiotics
16. Venous thrombosis prophylaxis
17. Other medications
18. Catheterize q 6 hours, or sooner, if bladder is full and patient unable to void.

FIGURE 9.8 Sample of basic postoperative orders.

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