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The critical care aspects of the gynecologic patient have many similarities to the care of the critically ill, adult patient with respect to physiologic and organ support. The critical care aspects of the pregnant patient, however, are substantially different from that of the nonpregnant patient. In part, this is because treatment considerations that are geared toward optimization and individualization of a single patient in the adult ICU setting must be approached with greater flexibility to consider the needs of each of the individual components of the maternal–fetal unit complex. Pregnant patients in the critical care unit can present with diseases and conditions that are specific to pregnancy, but must also be viewed in the light of specific differences in physiology between the gravid and nongravid patient, as well as the gravid patient and the fetus [1–13]. Maternal–fetal medicine was first recognized as a distinct obstetric subspecialty by the American Board of Obstetrics and Gynecology in 1972 [14] and the need for specialty resource units to care for the obstetric patient soon followed. In the current era of cost containment few hospitals can devote specialized resources to maternal–fetal ICUs. Many critical care practitioners may not have had exposure to the special needs of a pregnant patient during their training or within their practice settings [15,16]. An issue which further complicates consideration of this topic is that many of the published reviews of this area were written from a purely obstetric viewpoint [17–19]. This article, which focuses primarily upon the obstetric patient, summarizes for the nonobstetric critical care practitioner the scope of the problem, the physiologic changes in pregnancy, the fetal concerns, the most common clinical illnesses that result in ICU care for the pregnant patient, and common conditions whose management may be altered in the situation of pregnancy or gynecologic surgery.

Incidence and prevalence of gestations requiring ICU care

The overall prevalence of obstetric patients who may require critical care during their pregnancy ranges from 1 to 9 in 1000 gestations [1,20]. The mortality of critically ill, obstetric patients ranges from 12% to 20% in most series [5]. The number of patients who require admission to an ICU during their pregnancy, or die because of their illness, is difficult to estimate because substantial reporting variability exists in the literature. All investigators reported numbers of obstetric patients who were admitted to the ICU as a constant figure, but the numbers against which a comparison can be made are not constant. The obstetric patients who were admitted to the ICU were compared with total hospital admissions [20], total ICU admissions [6,8], or total delieveries [1]. Many investigators failed to report any number to serve as a foil for comparision [2,4,9–12]. Additional factors that affected the reporting of exact numbers included interinstitution variability. Marked differences in values are reported between high obstetric volume centers versus low obstetric volume centers, the overall volume of hospital admissions, and the degree of specialization of services offered by the hospital. There are marked differences reported between tertiary referral centers compared with primary referral centers, as well as the location of the hospital (eg, developed versus nondeveloped countries). It was suggested that ICU admission rates for women who become pregnant over age 40, who have had multiple previous pregnancies [21,22], who are carrying multiple gestations [23], and perhaps those with antepartum depressive symptoms [24,25], may be higher than those numbers reported for younger women, women with singleton gestations, and without evidence of depression. The same factors that account for higher ICU admissions affect the rates of maternal and fetal morbidity and mortality It is difficult to completely isolate the effect of advancing age on pregnancy and ascribe a critical age of pregnancy. As the demographics of the workplace change and more women enter the workforce, there has been a trend towards delaying pregnancy until after the establishment of a profession. The contribution of pregnancies in older women who require ICU care compared with the overall number of pregnancies which require ICU care is difficult to evaluate. Within the limits of database reporting error there were more than 4 million live births recorded in the United States in 1992. During this same period, only 2000 live births were recorded in women over age 45. A distinction is beginning to appear in the obstetric literature of “pregnancy”, “pregnancy at advanced maternal age” (≥ 35 years at delivery), and “pregnancy at very advanced maternal age” (≥ 45 years at delivery) [26]. To further blur the distinction between classic definitions of morbidity and mortality, the obstetric literature is unique in its use of the term “near miss maternal mortality”, which was popularized in reporting statistics within the United Kingdom [27]. This is defined as a serious morbidity during or up to 42 days after pregnancy, which requires intensive care unit admission [1,27]. Overall rates of maternal, fetal, or infant mortality and morbidity are similarly difficult to estimate. In developed countries, maternal death during or related to pregnancy has stabilized over the past 20 years to 5 to 10 per 100,000

pregnancies [13,28–30]. Not surprisingly, similar to the reports for overall ICU utilization rates, markedly different rates of maternal mortality are reported in developed countries compared with underdeveloped countries. These numbers have been under recent scrutiny; new international estimates of maternal death are not in agreement with previously reported figures. Estimates of maternal death range from 190 per 100,000 live births in developed countries to more than 1000 per 100,000 live births in underdeveloped countries (eg, subSaharan regions of Africa) [13]. New initiatives that are directed at lowering overall mortality are being sponsored by the World Health Organization and the United Nations Children's Fund [31]. Additional information from the analysis of scoring system data supports the idea that Acute Physiology and Chronic Health Evaluation (APACHE II), Simplified Acute Physiology Score (SAPS II), and Mortality Prediction Model (MPM II) are sufficiently robust to allow their use as scoring systems for critically ill, obstetric patients, provided that errors in attributing primary diagnosis codes are avoided [32].

Physiologic changes in pregnancy

Pregnancy induces changes in every organ system of a woman. Each of the changes is intended to provide the best outcome for the mother and the fetus. It is helpful to globally consider the changes as part of an adaptive process, the goal of which is to provide maximal support for the fetus and minimize the stress for the mother. The processes that support the fetus, are, in large part, designed to maintain placental blood flow, which, in turn, promotes delivery of oxygen and nutrients to the fetus. The processes that minimize the stress on the mother are designed predominantly to expand the maternal blood volume. This provides a buffer to offset the inevitable blood loss that is associated with even an uncomplicated vaginal delivery. The expanded blood volume is a major factor that increases cardiac output and flow through the placental vessels [33].

Weight gain during pregnancy is closely related to the preconception, nutritional status. As a rule, thin women gain more weight during pregnancies than obese women. The majority of the weight gain is from increases in maternal body fat and protein stores, as well as from the expansion of maternal blood volume. The increased nutrient stores provide a reserve from which the future caloric requirements of the fetus in pregnancy and lactation may be drawn. As with any patient with increased metabolic demands, the pregnant patient should have specific attention directed toward maintenance of nutritional stores. In cases of altered intake, a more aggressive approach in the early application of nutritional support is warranted [33,34].

Cardiovascular physiology is affected by changes that are necessary to support the demands of the enlarging placenta and fetus, increased uterine blood flow, and in response to increased pelvic, venous capacitance. The maternal blood volume and extracellular fluid increases progressively during pregnancy. An average increase in the blood volume of approximately 2 liters, or 30% to 50% more than in the nongravid state, occurs. Estrogen is the mediator of these changes, and it predominantly affects uterine blood flow. At term, uterine blood flow may approach 600 mL/min. A portion of the total volume increase is manifested in the accumulation of 1 to 5 liters of extracellular fluid, which may be manifested in near-term, normal pregnancies as peripheral edema. The maternal red cell mass increases only 20% to 30%. The disproportionate rates of increase of maternal red cell mass and blood volume produces a state of progressive hemodilution; the overall, expected effect on oxygen delivery is offset by a simultaneous increase in cardiac output to maintain overall, maternal oxygen delivery. The increased cardiac output is mediated by the circulatory effects of the uteroplacental vessels, which are themselves influenced by the vasodilatory effects of progesterone and prostaglandins E2 and I2. The uteroplacental vessels function essentially as a low pressure, left-to-right shunt, which by nature of increased, left ventricular ejection fraction results in increased cardiac output. The net effect is to maintain the overall maternal oxygen carrying capacity. The fetus has characteristics that enhance its oxygen extraction capacity. Factors which increase the ability of the fetus to extract oxygen are the presence of a higher hemoglobin concentration (15 g/dL) compared with the mother, as well as the oxygen hemoglobin disassociation characteristics of fetal hemoglobin (Hgb F). Hgb F is 80% to 90% saturated at a PO2 of 30 to 35 mm Hg compared with the predominant form of adult hemoglobin, Hemoglobin A (Hgb A- p50 = 27mm Hg) which is only saturated 50% to 55% at this partial pressure of oxygen. Finally, the presence of a ductus arteriosis provides the fetus with essentially two ventricles to supply systemic circulation. These effects, the offloading advantage of oxygen from more dilute maternal hemoglobin to higher fetal hemoglobin concentrations, the differences in oxygen affinity between Hgb A and Hgb F, and the concurrent exchange mechanism of the placenta to fetal interface enhance maternal-to-fetus oxygen transport [5,8,35,36]. Although the fetus is relatively hypoxic when compared with the mother, without the mechanisms described above, sufficient oxygen delivery to the fetus would be impaired [16,36,37]. It is of cardinal importance to keep in mind the effects of maternal position and uterine contractions on hemodynamics. As the uterus enlarges after 20 weeks' of gestation, a

supine position may allow the uterus to compress the vena cava and the aorta which effectively decreases venous return and increases cardiac afterload. Either effect may result in a decrement of up to 30% in ejection fractions [38,39]. Uterine contractions can cause sudden and marked changes in stroke volume and cardiac output related to transient increases in preload that is caused by the abrupt diminution of the uteroplacental capacitance capacity. This contraction-to-contraction variation in stroke volume and cardiac output preserves flow to maternal organs during the stress of delivery. Cardiac arrest in the pregnant patient and resuscitation demands attention to elevation of the uterus to the left, either manually or by elevating the right hip/flank to avoid compression of the inferior vena cava by the gravid uterus. Supportive data exist for the consideration of an emergency bedside cesarean section if 4 to 5 minutes of CPR are ineffective in restoring a maternal rate, rhythm, and sufficient blood pressure [3,5,8].

Respiratory physiology changes are manifested by the need to provide increasing amounts of oxygen and eliminate increasing amount of carbon dioxide as the fetus matures. The predominant changes are a decrement of approximately 18% in the functional residual capacity (FRC) from limitation of diaphragmatic excursion. The diaphragm may be elevated by as much as 4 centimeters above its normal position near term. A major respiratory change in pregnancy is an increase in minute ventilation of up to 45% that is attributable to an increase in tidal volume and a more substantial increase in respiratory rate. The increased minute ventilation leads to a mild respiratory alkalosis and a left shift of the maternal oxygen hemoglobin disassociation curve; this further augments the maternal-to-fetus transfer of oxygen that is already favored by hemoglobin composition and concentration differences between mother and fetus [5,8,33,39]. The changes in alveolar ventilation and decreases in FRC have important implications for the use of inhaled anesthetics. Generally lower minimum alveolar concentrations of inhaled agents are needed to achieve general anesthesia. Lower doses of agents that are used for regional as well as neuraxial (epidural and spinal) anesthesia to achieve analgesia were reported [40].

Renal physiology changes in pregnancy are manifested by increases in glomerular filtration rate and creatinine clearance (CrCl) to 45% above baseline compared with nongravid levels. Urinary volumes exhibit an increase that is similar to those exhibited by GFR and CrCl. These changes occur throughout pregnancy and are in response to the increased excretory loads that are imposed on the mother by the enlarging fetus and increases in maternal metabolism [5,8,33]. The significance of these increases is a lowering of the serum creatinine values (< 0.9 mg/dL nl in pregnancy), blood urea nitrogen values (< 15 mg/dL nl in pregnancy), and uric acid levels (< 6 mg/dL nl in pregnancy). An increase in urine glucose occurs. These changes require caution in the interpretation of serum values that are reflective of renal function in the pregnant female. A pregnant female with serum creatinine levels that would be considered normal in a nonpregnant condition already has significant renal impairment [5,8,33]. The GFR-mediated glucosuria makes urine glucose unreliable in the late term patient. As pregnancy progresses, decreases in plasma osmolarity, and increases in aldosterone, estrogen, and plasma renin activity are offset by reduced sensitivity to these endogenous vasopressors; these are clearly responsible for the characteristic decreases in systolic and diastolic blood pressures and widening of the pulse pressure that are seen in the late second and early third trimesters. Failure of this response may be genetically linked to eclamptic conditions. If the clinician fails to recognize the cardiovascular and renal-mediated changes of pregnancy then hypertensive states, or response to critical illness and blood loss conditions requiring transfusion therapy may not be appropriately treated [33,39].

Gastrointestinal physiology is similarly affected by the changes in maternal hormonal status which mediate many of the other organ physiology changes during pregnancy. Within the GI tract, most of the changes are caused by a progesterone-related alteration in smooth muscle relaxation. The increasing size of the uterus and associated pelvic vasculature causes displacement of gastrointestinal organs in cephalad, lateral, and posterior directions. Although many common conditions that require surgery in the pregnant patient occur with the same frequency in age- matched, nonpregnant women, the displacement of viscera may lead to difficulty in the interpretation of physical signs that are characteristic of common illnesses. McBurney's point tenderness and Murphy's sign assume decreasing significance as pregnancy progresses. Gastrointestinal effects that are ascribed to the hormonal fluxes of pregnancy include decreased lower esophageal sphincter tone, which may exacerbate signs of GERD, and a generalized hypomotility of the gastrointestinal tract. The hypomotility is thought to allow more complete adsorption of nutrients from the GI tract and maximize maternal and fetal nutrition. It is associated with increased complaints of obstipation or constipation during pregnancy [33].

The hematologic changes that occur during pregnancy are not manifested by symmetric parallel changes in maternal blood volume, circulating red cell mass, stroke volume, and cardiac output. The end result is to maintain maternal oxygen-carrying capacity and promote the transfer of oxygen from mother to fetus (see earlier discussion) [5,8,33,39]. These changes act in concert to provide blood of lower viscosity. From a teleological standpoint, this would allow for the increases in cardiac output at the lowest expenditure of pumping energy. Recent trends caused a reappraisal of the notion of "physiologic anemia of pregnancy." The replacement of iron, folate, and B vitamins should have as their goal the support of hematopoeisis, not the restoration of prepregnancy hematologic values. As pregnancy progresses, increases in peripheral white blood cell counts occur; most of the increases are noted in the leukocyte fraction. This is especially evident in the T3 helper cell fractions. Similarly, increases in fibrinogen, and Factors VII and X occur, and more than offset the decreases in concentrations of Factors XI and XIII, and platelets. Although levels of Factor II (prothrombin) decrease, standard measures of coagulation, including prothrombin time and partial thromboplastin time are unchanged. The net effect is that pregnancy should be considered as a hypercoagulable state [33,41].

Organ-specific alterations in anatomy

Ophthalmic changes may be manifested as alterations in vision, generally myopia, that is thought to occur from the generalized increase in extracellular fluid and resultant mild corneal edema. This may affect contact lens users. Occasionally, modest bitemporal hemianopsia can occur that is related to volumetric increases in pituitary gland size and optic chiasm compression [33].

Otorhinolaryngologic changes can also be ascribed to progesterone-mediated hyperemia and edema of mucosa surfaces. Epistaxis, periodontal disease, and middle ear symptoms from decreased patency of the eustachian tubes may be exacerbated during pregnancy. Knowledge of (ears, nose, throat) ENT-related changes, coupled with pregnancy-associated obesity, altered dentition, and recognition of anatomic variants, such as a short neck or receding mandible, is essential to the practioners who may be called upon to insert nasogastric or endotracheal tubes. A higher than expected rate of epistaxis may occur or downsizing of endotracheal tubes may be required [4,33,40,42].

Endocrine changes are not limited to estrogen- and progesterone-mediated effects. As discussed earlier, increases in the size of the pituitary gland during pregnancy may contribute to visual field disturbances or may be associated with ischemia-induced necrosis in the puerperium, which is manifested as hypothalamic dysfunction (ie, Sheehan's syndrome). This is most commonly caused by excessive blood loss during delivery which leads to hypotension. As pregnancy progresses, corticosteroid-binding globulin levels increase which are manifested by the elevation of serum cortisol levels. Deoxycorticosterone levels are elevated secondary to steroidogenesis of the fetal-placental unit, the production of which is stimulated by human chorionic gonadotropin, a hormonal product of the placenta. Overall, the relative ratio between bound and free cortisol does not change and diurnal variability is preserved. Despite levels that could seem to be cushinoid in the nonpregnant patient, the pregnant patient remains largely asymptomatic. The overall, elevated levels of gucocorticoids may, in part, be an explanation for the reported observations of improvement in autoimmune diseases, such as rheumatoid arthritis, that are seen during pregnancy. As discussed earlier, levels of aldosterone are increased during pregnancy. This may be a compensatory mechanism to maintain sodium balance in the face of increased losses that are associated with the elevations of GFR from increased intravascular volume, cardiac output, and dilatation of the renal vasculature. Attributable to the continuous transplacental transfer of nutrients, ketosis may appear more quickly in the pregnant female than in the nonpregnant female. The nutritional intake of pregnant females must be carefully monitored [41].

Orthopedic changes are mediated by the effects of relaxin and progesterone. Increased ligamentous laxity serves to increase the mobility of the pelvic girdle, which is thought to enhance vaginal delivery. Unfortunately, the progressive ligamentous laxity may lead to pelvic discomfort and gait disturbances. These disturbances may exacerbate pre-existing lumbarsacral disc disease or be associated with an increased risk of falls with resultant trauma. Increases in extracellular fluid may cause more complaints from nerve entrapment syndromes, especially carpal tunnel syndrome. In addition to the increased frequency of nerve entrapment syndromes, pregnant women have an increased frequency of complaints that are associated with overall cerebral vascular disease, including headache and cerebral vascular accidents. Large studies with high statistical power to address this, however, are lacking [74].

Psychiatric changes in pregnancy are generally limited to anxiety disorders. Most are related to the fear of fetal abnormalities, injury from labor or undue pain, or even feelings of inadequacy that stem from ambivalence toward the pregnancy, or performance issues that are related to the future role as a mother and caregiver. There is an association between antepartum depression and an increase in instrument and operative-assisted deliveries, infant admissions to the neonatal intensive care unit, intrauterine growth retardation, prematurity, and an increased use of pain control adjuncts during vaginal delivery [24,25].

Critical illness not specific to pregnancy

Acute respiratory failure in the pregnant patient is produced by conditions that are similar to those seen in the nonpregnant patient. The leading causes of respiratory failure include asthma, pulmonary edema from intrinsic cardiac disease, amniotic fluid embolism, beta-adrenergic tocolytic therapy, infectious pneumonias, aspiration pneumonia, pulmonary embolism, and, rarely, pulmonary hypertension. Chronic lung disease is an uncommon cause of acute respiratory failure in the pregnant patient [38,39,43–45]. Asthma is the most common cause of pulmonary complaints in the general and pregnant population [45]. No definite findings exist to support the notion that pregnancy and its associated hormonal changes, many of which diminish smooth muscle contractility, increases the severity of asthma. Current recommendations for the treatment of asthma, including the use of peak flowmeters to monitor endpoints of therapy, apply to the pregnant patient. The possible teterogenic effects of all medications that are used in pregnancy should be evaluated on a case-by-case basis and frank discussions shoud be held with patients about the benefits and risks of therapy. Generally, either inhaled or systemic steroids, cromolyn sodium, and inhaled beta agonists are acceptable therapeutic options [5,8]. Pulmonary edema in pregnant patients was most commonly ascribed to mitral stenosis. It is the most common valvular heart disease that is encountered in pregnant patients and is often complicated by atrial fibrillation. Treatment options include digitalis therapy, which is considered safe during all phases of pregnancy. The teteragenocity of warfarin precludes its use. Heparin is the preferred drug if anticoagulation is needed to treat or prevent pulmonary embolism in the pregnant patient. Consideration can also be given to insertion of a venal caval filter [5,39,45]. Amniotic fluid embolism will be discussed later. The use of tocolytics to retard the progression of labor was associated with pulmonary edema in from 0.3% to 9% of all pregnancies during which it was required. The overall use of tocolytic therapy to retard the progression of labor is decreasing as available evidence suggests that neither oral nor parenteral tocolytic therapy reduces the incidence of maternal–fetal complications. Additionally, pulmonary edema which is resistant to conventional forms of therapy was associated with the use of tocolytics. The current indications of tocolytic therapy are to inhibit uterine contractions in fetal distress and to assist external cephalic inversion [8]. Pneumonias in pregnant patients, similar to pneumonias in the nonpregnant patient, may result from typical or atypical organisms that are acquired in the patient's community or nosocomially. Aspiration is a common cause of pneumonia in the obstetric patient because of hormone-mediated effects on the lower esophageal sphincter, coupled with increased transdiaphragmatic pressures from the gravid uterus. Considerations in the diagnosis and treatment of pneumonias in the pregnant patient should include knowledge of potential adverse effects of antibiotic therapy [46] and diagnostic radiation on the fetus. Thoughtful consideration should be given to the use of the least toxic, antibiotic choice coupled with limitation of unnecessary, radiation exposure. Initiation of preterm labor occured during severe pneumonias as well as from many other conditions in which the systemic inflammatory response system is activated for an extended time. As in the general population, mortality rates of pregnant patients who progress to severe lung injury and adult respiratory distress syndrome remains high. Although pregnant patients are generally younger than patients who are admitted to general ICUs, there is no survival advantage conferred by their younger age [8,23,47–52]. Ventilator management of the pregnant patient is similar to management in the nonpregnant patient. Attention should be directed to the avoidance of respiratory alkalosis, the use of the lowest possible oxygen concentrations, and avoidance of high inflation pressures (measured at peak and plateau phases). In later stages of pregnancy, the increase in intra-abdominal volume and its effect on decreasing chest wall compliance may dictate somewhat higher inflation pressures to maintain acceptable levels of ventilation [5]. The clinician must remain cognizant of the spectrum of pregnancy-mediated changes in common physiologic measurements that are used to base judgments affecting therapy.

Cardiac disease is a complicating factor in approximately 1% to 4% of all pregnancies; rheumatic valvular disease is the most common cause. Bacterial endocarditis seems to be increasing, as does the presentation of females of reproductive age who have had correction of congenital cardiac anomalies; in the past, congenital cardiac anomalies would have precluded their survival to childbearing age. Valvular replacement, commissurotomy, coronary artery bypass grafting, removal or correction of intracardiac lesions, and repairs to great vessels have been performed with an overall 1.7% to 3% maternal death rate and 17% to 19% fetal

death rate. Although no prospective data exist, major considerations that are related to the performance of cardiac surgery during pregnancy include: (1) avoidance of open heart procedures if possible in the first trimester; (2) the use of high flow, high pressure normothermic bypass presents the lowest risk to the mother–fetal unit; (3) adequate placental perfusion should be checked with fetal monitoring of heart rate and uterine tone to guide pharmacologic interventions; and (4) elective, pre term cesarean section as guided by measures of fetal lung maturity may be considered. Novel approaches to the treatment of cardiomyopathy of the postpartum period, including continuous veno-venous hemofiltration, were reported [53–55].

Acute renal failure (ARF) may occur from many of the same initiating factors which cause renal failure in the nonpregnant patient. These are commonly hypovolemia from dehydration and acute blood loss, infectious etiologies that are systemic and confined to the urinary system, progression of pre-existing renal disease, and drug toxicities. Causes of ARF that are related to obstetric complications include sepsis from illegally performed abortions, acute fatty liver of pregnancy (AFLP) preeclampsia, obstetric causes of hemorrhagic shock, the hemolysis elevated liver enzymes low platelets (HELLP) syndrome, and hemolytic uremic syndrome (HUS). The patterns of the renal failure and clinical courses vary with respect to the initiating cause. Bilateral renal necrosis occurs in patients with renal failure from septic abortions, pre-eclampsia, and acute pregnancy-associated hemorrhagic shock and is associated with irreversibility in as many as 26% of patients. This is compared with an irreversibility rate of 12% from all other causes of ARF in pregnant patients [5,8].

Autoimmune diseases may be quiescent or exacerbated during pregnancy. Diseases that may improve during pregnancy, include rheumatoid arthritis, polymyositis, and polyarteritis. This improvement presumably results from increases in overall levels of glucocorticoids reported in pregnancy. Myasthenia gravis may present with a remission or an exacerbation. It may also be associated with the appearance of a transient neonatal myasthenia from direct transplacental passage of acetylcholine antibodies. The most severe autoimmune diseases that are encountered during pregnancy are systemic lupus erythematosus and antiphospholipid ayndrome. (APS). The preponderance of literature associates these diseases with increased rates of abortion, fetal loss, and maternal death. Additionally, the risk of morbidity and mortality for patients with APS extends beyond the period of pregnancy well into the puerperium, as demonstrated by reported increases in maternal and infant morbidities [8,56]. The traditionally held ideas that advise against pregnancy in patients with autoimmune diseases such as SLE and APS have recently come under criticism. The reported numbers of affected patients are small. The relatively low prevalence of pregnant patients with severe autoimmune disorders such as SLE and APS may be secondary to many clinicians' unfamiliarity with autoimmune disease entities. The complete clinical course in afflicted patients remains somewhat unclear. APS was first officially described by Harris in 1987 [57]. Encouraging work in the early detection of affected individuals by review of patient databases for those who present with recurrent miscarriages, led to treatments. Aspirin alone or a combination of aspirin and heparin seems to hold promise. In a recently published study, women who had APS failed to exhibit increased rates of preterm delivery, intrauterine growth restriction, or infant developmental abnormalities when compared with historical controls. Although the overall numbers are small this may hold promise for future developments [58].

Endocrine diseases that occur during pregnancy are rare. Despite the fluxes in many hormone levels, including cortisol, pregnancy-associated weight gain, changes in cardiovascular, renal and gastrointestinal physiology, and the commonly observed deterioration of glycemic control of the insulin-dependant diabetic during pregnancy, frank ketoacidosis is rare. There are reports of an incidence rate of only 9%. Recent evidence challenges the historical viewpoint that pregnancy accelerates the course of nephropathy in patients with pre-existing insulin-dependant diabetes mellitus and moderate renal impairment [59]. Gestational diabetes (GDM), defined as reduced glucose tolerance initially diagnosed during pregnancy, is also infrequent and occur in 1% to 3% of all pregnancies. GDM was associated with lower gestational age at delivery, which is unexplained by a corresponding increase in premature deliveries. It is also associated with increased infant morbidity and mortality as well as the use of neonatal ICU services. Recent evidence suggested that if GDM appeared before 20 weeks' of gestation, then it is correlated with a higher likelihood of insulin-use during pregnancy and a higher risk of the later, non-GDM–associated development of overt diabetes [60]. Thyrotoxicosis and pheochromocytomas rarely occur in conjunction with pregnancy. Symptomatology that includes tachycardia, palpitations, thermal intolerance, facial flushing, and headache may be erroneously attributed to normal physiologic adaptations of pregnancy. Many commonly use antithyroid drugs cross the placenta so their dosages should be monitored and kept as low as possible to prevent the development of fetal hypothyroidism. The use of propylthiouracil seems to have the least trans-placental transfer secondary to its extensive binding to serum proteins [61].

Trauma occurs in as many as 6% to 7% of all pregnant patients. The cause is generally motor vehicle crashes, other decelerational type injuries such as falls, burns, and penetrating injuries [5,8,47,62–67]. The pregnant patient is not excluded from trauma and can incur all forms of traumatic injuries that are reported in nonpregnant patients. Trauma may range from relatively minor trauma to the most devastating neurologic injuries; there were several reported cases of the successful delivery of an intact infant after prolonged life support of a severely brain-injured mother [68–70]. Recent studies, many of which may be criticized for methodologic flaws, such as the Neyman bias (incidence-prevalence bias) or the volunteer bias (increased incidence reported by voluntary populations) have focused attention on the role of physical abuse of pregnant women. This is most commonly reported in the form of domestic violence. The incidence of physical abuse during pregnancy has been reported to be as high as 41% when survey based instruments of data collection were reviewed [66,71,72]. Uniformity exists within the trauma literature with respect to maternal–fetal outcomes, and similar to the characterization of all other trauma outcomes, maternal-fetal outcomes are closely linked to the severity of the initial presenting injuries. Maternal or fetal death is increased in the presence of shock, pelvic fractures, severe head injury, and hypoxemia, a situation not unlike other trauma victims. Pregnant women do not have higher mortality or morbidity rates when compared with matched, nonpregnant controls. The physiologic alterations found in a normal pregnancy may make diagnosis and management more difficult. Generally acceptable clinical endpoints are altered by pregnancy-induced physiology changes. Therapeutic decisions based upon the patient alone, without knowledge of the pregnancy-induced changes in physiology, may account for the high ratio of fetal to maternal deaths (3:1 to 9:1) that are ascribed to trauma in the literature [63–65,73,74]. Anatomic changes of pregnancy may either mask the presentation of certain injuries or increase the possibility of other injuries. As pregnancy progresses, the enlarged, pregnant uterus assumes a more intra-abdominal position, as does the urinary bladder. Both are more prone to rupture from blunt trauma as pregnancy progresses. Uterine rupture from blunt trauma is an injury unique to the pregnant female and carries a fetal mortality rate of 100%. Maternal survivability is further hindered by other injuries that are associated with this condition, such as amniotic fluid embolus. The displacement of organs by the enlarging uterus, makes them more prone to injury during blunt and penetrating trauma, and may cause the patient to present with unusual injury patterns. The dilatation of retroperitoneal veins in late pregnancy may increase the propensity for retroperitoneal hemorrhage after pelvic fracture [39,47,63,75–81]. Most maternal–fetal mortalities are caused by the failure to recognize the potential for the presence of trauma-induced hypovolemia from visceral, vascular, or uterine injury, or pregnancy-specific conditions, such as placental abruption, or feto-maternal hemorrhage. Feto-maternal hemorrhage is a condition in which blood is lost directly from the fetus into the maternal system. It may cause a spectrum of symptoms that range from minor fetal anemia, to fetal heart rate abnormalities, to fetal death. It is a condition of maternal isoimmunization from the transfusion of Rh positive fetal blood to an Rh negative mother. It may occur with minor trauma or even normal delivery and can complicate future pregnancies. A Kleihauer-Bettke analysis should be obtained to detect the presence and percentage of fetal red blood cells in the maternal circulation. Any positive results should prompt prolonged fetal monitoring. Commercially available anti-D immunoglobulin should be administered in the situation of an Rh-positive fetus carried by an Rh Negative mother. Suggested dosages are 300 micrograms for every 15 mL of fetal blood that is present in the maternal system. Monitoring assists in the quantification of ongoing fetal well being and ongoing fetal–maternal transfusion [39]. Well-established guidelines for the management of trauma with specific applicability for use in the pregnant patient exist [82]. In each incident of trauma to a pregnant patient, the assistance of a surgeon whose practice includes the care of the acutely injured patient as well as the assistance of an obstetrician for assistance in the application of monitoring techniques such as cardiotocography, continuous fetal heart rate monitoring, fetal scalp blood sampling, ultrasound, and the judicious use of radiographs should be considered standard care [5,8,39,65,75,76,81,86].

Surgical emergencies that require operative intervention occur in pregnant patients at rates that are similar to those reported in the nonpregnant, general population. Appendicitis, acute cholecystitis, intestinal obstruction, biliary colic, pancreatitis, peptic ulcer disease, inflammatory bowel disease, and adenexal pathology, including torsion and masses, have been reported. Pregnancy-specific conditions that require surgery, such as renal rupture, esophageal rupture, acute hepatic rupture (most commonly associated with AFLP), rupture of visceral aneurysm, and vaginal bleeding related to alterations in placental function or implantation are well described. Their association with pregnancy presents an initial diagnostic challenge followed by a surgical challenge. ICU care, when warranted, follows generally acceptable standards and has no disease-specific differences from that of the general population. In general, surgical emergencies that require acute, operative intervention, are generally rare and occur in only 1% to 2% of pregnancies. General rules of maintenance of blood and plasma volume, avoidance of caval compression by the gravid uterus,

and deep venous thrombosis prophylaxis are applicable to all cases. Appendicitis is the most common, nonobstetric/gynecologic condition of pregnancy that requires surgery and has a reported incidence of 0.5 to 1 in 1000 pregnancies. In confirmed cases, the risk of conservative treatment far outweighs the risk of operative intervention at all stages of pregnancy. Ultrasound is useful in cases with an unclear or atypical presentation. Cephalad displacement of the cecum that is caused by the progressive enlargement of the uterus may alter the location of somatic pain and the location of the incision, which as a rule, should be over the point of maximal tenderness [83,84]. The role of laparoscopic appendectomy may be best relegated to cases that present in earlier trimesters, as abdominal domain issues that are related to the enlarging uterus increase the difficulty of laparoscopic visualization near term. Improvements in diagnostic accuracy, perioperative care, and fetal monitoring led to a reduction in fetal and maternal mortality over the past 50 years [85]. Cholecystitis is the second most common condition that requires surgical intervention in the pregnant patient. It is thought to occur in 1 in 1600 pregnancies. Medical management with intravenous antibiotics that are safe for use in pregnancy, intravenous fluids, and pain control are recommended as the initial approach in the first and third trimesters. Surgery or medical management may be used in cases that present during the second trimester. Intestinal obstruction afflicts an estimated 1 in 2500 to 3500 pregnancies. As in the nonpregnant population, the most common cause is adhesions from previous surgery. Sigmoid or cecal volvulus, pseudo-obstruction (Olgivie's syndrome), and intussusception have been reported. Presentation of these conditions must be distinguished from complaints that are related to the gastrointestinal dysmotility of pregnancy. Standard therapy that includes gastrointestinal decompression, intravenous fluid management and laboratory analysis of blood values, which should be interpreted in the light of the hematologic changes of pregnancy, are applicable during the period of diagnosis confirmation. Surgery is indicated only after the failure of medical management has occurred. After confirmation of intestinal obstruction that is resistant to medical therapy has been made, surgery is mandated, without regard to the stage of the pregnancy. A perforated duodenal ulcer is exceeding rare but must be considered an emergency that mandates surgery. Biliary colic, pancreatitis, peptic ulcer disease, and inflammatory bowel disease are generally treated for relief of their symptoms. Surgery is reserved for complications of these conditions. Fortunately, adenexal pathology, including torsion and masses, are rare. No data from large series are available, and the true incidence of these diseases is unknown. An observed increase in prevalence may be the result of increased skill, familiarity, and the more liberal application of ultrasound. Other general surgical gynecologic conditions such as breast masses, mastitis, vulvar or cervical disease present infrequently as emergencies during pregnancy and seldom require the utilization of ICU services [83,85,86].

Critical illness considerations specific to pregnancy occur in patients who can present with preeclampsia, Eclampsia, HUS, HEELP AFLP, amniotic fluid embolus, tocolytic-induced pulmonary edema, or severe obstetric infections and sepsis. Distinction between these syndromes may be difficult because of similarities and overlap in their presentations, variability in reporting and lack of standard nomenclature to allow distinction between syndromes. Preeclampsia, eclampsia, HUS, HEELP and AFLP all have hypertension as a component of their presentation. The relationship between pregnancy and hypertension has been recognized since the time of the ancient Greeks; hypertension complicates approximately 5% of all pregnancies. It is the second most common cause of maternal mortality in the United States [87]. The cause remains unknown. The American College of Obstetricians and Gynecologists classifies hypertension that occurs in pregnancy into four groups: (1) preeclampsia, (2) chronic hypertension, (3) chronic hypertension with superimposed preeclampsia, also known as gestational hypertension, and (4) eclampsia [88–90]. Each entity exhibits microangiopathic changes in the vascular endothelium associated with thrombosis and vasospasm. Many theories exist about the causes, but current attention is focusing on the role of mediators of the microangiopathic changes. Putative agents that are currently being investigated include angiotensin II, catecholamines, prostaglandins, abnormal trophoblastic invasion, insufficiency in the production of blocking antibodies, and genetic predisposition [91–93]. As a result of the microangiopathic changes, dysfunction may occur in renal, hepatic, hematologic, neurologic, and uterine–placental beds. The diseases associated with accelerated hypertension and microangiopathic changes may present as a progressive spectrum of illness. Severity of presentation ranges from mild cases, to cases associated with multiple organ dysfunction syndrome (MODS), multi-system organ failure (MSOF), or even death [90].

Preeclampsia is a disease state that was previously known as pregnancy induced hypertension, gestational proteinuric hypertension, or toxemia of pregnancy. It is more common in nulliparous women and was initially labeled as a disease of first pregnancies. Paradoxically, it may also be associated with older multiparas who have changed sexual partners, or women who are carrying multiple gestations [87–90]. Other risk factors include obesity, insulin resistance, diabetes, chronic hypertension, renal disease, dyslipidemias, smoking,

molar pregnancy, hydrops fetalis, and collagen vascular diseases, such as APS [8,88,89]. The disease most commonly presents after the 20th gestational week but it may present at any time; postpartum development was reported. The syndrome invariably includes rapid weight gain, nondependent edema proteinuria (> 300 mg/24 h), and hypertension (≥ 140/90 after the 20th week of gestation). The presentation is unpredictable and can be mild, resolve with delivery, or become more severe and progress to a spectrum of illnesses. Treatment hinges upon recognition of the entity and remains largely supportive. The only known definitive treatment is termination of the pregnancy. While near term, effectively terminating the pregnancy is seldom a problematic choice; the management of earlier term pregnancies requires a careful evaluation of risks to mother and fetus. If signs of severe eclampsia are present, then the risk to the mother may outweigh the risk to the fetus.

Eclampsia is a more severe form of preeclampsia and has, in addition to the aforementioned hallmarks of preeclampsia, the manifestation of MODS or MSOF. Signs of progression of preeclampsia from severe pre-eclampsia to frank eclampsia include: (1) progressive hypertension (≥ 160 systolic or ≥ 110 diastolic); (2) worsening proteinuria, oliguria, or rising creatinine levels; (3) abdominal pain (especially in the right upper quadrant or epigastric area); (4) neurologic changes (including headache, visual disturbances, papilledema, altered level of consciousness or seizures); (5) pulmonary edema; (6) a new third heart sound; (7) petechia; (8) ecchymoses; or (9) elevated liver functions. To optimize outcomes, ICU management that uses a team of obstetricians and intensivists was suggested [5,48,94–100]. Temporizing measures that are commonly used to treat hypertension and prevent the neurologic complications, such as seizures, are used. After the establishment of secure intravenous access, volume expansion that is directed at the correction of any pre-existing deficits is initiated. This reduces the risk of vasodilator-induced hypotension and preserves the remaining renal function. Volume expansion is begun with crystalloids at maintenance rates of at least 100 to 125 cc/h. Level I evidence exists for the use of magnesium sulfate in the therapy of eclamptic seizures and many RCTs have demonstrated its superiority over other prophylactic agents, such as phenytoin and diazepam. It is given as a 6 gram loading dose intravenously over 20 minutes followed by titration of a continuous infusion of 2 to 3 g/hour that is sufficient to maintain a serum magnesium level in the range of 4.8 to 8.4 mg/dL (2–3.5 mmol/L). Magnesium administration should continue until 24 hours postpartum or 24 hours after the last known seizure. The IV administration of magnesium sulfate is favorable to therapies that use IM injections secondary to assured administration of drug and avoidance of injection site complications. The goal of therapy is to prevent seizures and avoid respiratory paralysis. All patients who are on high-dose magnesium therapy should have serial monitoring of deep tendon reflexes. Loss of patellar reflexes will occur at levels > 12 mg/dL. Serum magnesium levels may climb precipitously in the face of worsening renal function. Overdosage is characterized by muscular paralysis and respiratory arrest; this occurs at serum levels of 15–17 mg/dL. It is treated with intubation for respiratory support, cessation of the magnesium infusion, and infusion of calcium gluconate. Therapy with additional prophylactic anticonvulsants does not seem to add additional protective effect or demonstrated superiority to the use magnesium alone. Frank seizures that last longer than the usual 60 to 90 seconds that is reported to be the duration of the majority of eclamptic seizures, require emergency therapy. This includes: (1) airway control and oxygen therapy; (2) the determination of the need for mechanical ventilation; (3) diagnosis and initial steps to correct metabolic causes for seizures that may have been overlooked (including determination of arterial blood gases, CBC with differential, glucose, electrolytes, BUN/creatinine levels, toxicology screen, and levels of antiepileptic drugs if indicated by history; (4) intravenous thiamine 100 mg and 25 grams of glucose (50 mLs of D 50 % solution); (5) benzodiazepine therapy and phenytoin loading; and (6) immediate preparation for delivery [88,94,99–101]. Antihypertensive therapy is offered in the hope of preventing maternal hypertensive vascular damage. Agents that are used in this situation include alpha-methyldopa, labetalol, atenolol, metoprolol, hydralazine, and nicardipine. Nitroprusside or nitroglycerin should be avoided as both molecules may cross the placenta. Large trials of their use are lacking and adverse outcomes, especially an increased risk of refractory pulmonary edema, were reported when used concomitantly with tocolytics [102]. Although antihypertensive therapy seems to offer benefit no reversal or delay in the progression of maternal organ damage complications or increased fetal salvage was reported with their use. Other questions were raised about the wisdom of employing a therapy that may hinder uteroplacental flow. At the present time, after a review of available literature in maternal and neonatal physiology support, the historical view supporting the use of antihypertensive agents to lower blood pressure in the hopes of reducing hypertensive-associated neurologic injuries, maternal end organ damage, and permit prolongation of a pregnancy to delivery, must be questioned in all but the highest volume, tertiary care centers [95,97,100–106]. Insufficient data exist to promote the routine application of technologically-based measurements of cerebral and placental regional blood flow and hemodynamics as surrogate measures for the prediction of global maternal–fetal well being until large-scale, randomized, controlled trials exist [50,91,95,107].

HUS, AFLP, HELLP, and EELP are currently considered complications or variants of the basic preeclampsia-eclamptic state. Distinction between the entities may be difficult. HUS was thought to occur only in children before its description and categorization as a disease entity that is separate from thrombotic thrombocytopenic purpura. HUS was also thought to be a variant of TTP. TTP is a disease process that is manifested by a pentad of fever, neurologic symptoms, thrombocytopenia, a microangiopathic hemolysis, and renal impairment (all signs may not manifest, however), that occurs after the presentation of a disease state associated with initial thrombocytopenia and hemolysis. After the initial report of Moschowitz in 1925, other reports noted a female predominance in patients with TTP [52]. TTP was later divided into primary or secondary classes, with primary cases ascribed to idiopathic causes. Secondary TTP was associated with pregnancy, the puerperium, verotoxins, and metastatic carcinoma, infectious agents (bacterial and viral), as well as with many drugs. Other cases were described in which the renal involvement, thrombocytopenia, and uremia was not associated with neurologic changes; this was labeled HUS. The occurrence of uremia and thrombocytopenia, without neurologic changes, that occurs in pregnancy or the puerperium was classified as secondary HUS [49,52,90,96,108]. Little is known about the incidence of the disease and a genetic predisposition was suggested. Total numbers of involved patients are small. The highest survival rates were in patients whose therapy was targeted at stabilization and reversal of the endothelial injury which is thought to lead to distortion of the subendothelial space, deposition of thrombi, and microvascular and occlusion of affected organs. Biochemically, serum lactic dehydrogenase levels are in the range that reflect hemolysis, usually higher than 1000 U/L. Haptoglobin levels are decreased and the direct Coombs test is negative. Elevation of the patient's WBC is characteristic. Coagulation studies, fibrinogen, PT, PTT, and ATIII levels are normal.

The mainstays of current treatment are plasma transfusions. Initially, 30 to 40 mL/kg of plasma is infused, with reduction of the infusion to 15 to 20 mL/kg after the process is stabilized. Plasmapheresis is added, unless a prompt response can be demonstrated to plasma transfusions [92]. No delay in the institution of plasmapheresis should occur if improvement is not seen. Corticosteroids, aspirin, dipyridamole, immunoglobulins, and immunosuppressive drugs were tried with limited success. Unless massive hemorrhage occurs, the administration of platelet transfusions should be balanced against the risk of intravascular thrombosis. AFLP, is estimated to have an incidence of 1 in 13000 pregnancies [96,109–113]. It is associated with a maternal survival rate of 10% in untreated cases and microvesicular fatty infiltration of the central zone of the liver. An association with deficiencies in long-chain 3-hydroxyacyl-coenzyme A dehydrogenase was described. Hepatic dysfunction that is demonstrated by impaired gluconeogenesis (hypoglycemia), decreased production of plasma proteins (hypofibrinogenemia, elevated PT and PTT), increased serum ammonia levels, and hyperbilirubinemia characterize the laboratory abnormalities. The differences in coagulation studies are helpful in distinguishing AFLP from HUS; therapy is supportive [96,110].

HELLP syndrome was thought to be an atypical or more severe variant of the pre-eclampsia–eclampsia disease process until the end of the nineteenth century. The acronym HELLP was first suggested by Weinstein in 1982 to describe the syndrome of a microangiopathic hemolytic anemia, elevated liver enzymes, and low platelets in women with preeclampsia–eclampsia [52]. Since that time additional characterization of the disease produced the elevated liver enzymes and low platelet counts syndrome (ELLP) that was coined by Redmam in 1991 [114] as well as further description of a three-class system that is based upon the lowest observed value for platelet counts. The classification ranges from Class I HELLP (platelet counts ≤ 50,000/É L) to Class II HELLP (> 50,000 but ≤ 100,000 platelets/É L) and Class III HELLP (> 100,000 but ≤ 150,000 platelets/É L). Serum levels of lactic dehydrogenase (LDH) as well as hyaluronic levels are elevated. Serum haptoglobin levels are decreased and liver function studies, including transminases and bilirubin, are elevated. Anemia is inevitably present in HELLP as it is in patients with AFLP and HUS, but a key component to distinguish this disease from HUS is the presence of depressed levels of ATIII. Hypoglycemia and decreased fibrinogen levels may help in the distinction of AFLP from HUS and HELLP [49]. HELLP is a disease that commonly affects older women. This is a further distinction from the general association of pre-eclampsia with the young nulliparous female. The diagnosis is made by maintaining a high index of suspicion in patients who present with epigastric pain, nausea, emesis, eclampsia, severe hypertension, and abnormal bleeding, such as from placental abruption. After HELLP is diagnosed, immediate steps should be taken to assess the gestational age of the fetus. If the fetus is less than 34 weeks' of gestational age then corticosteriods, usually dexamethasone 10 mg IV every 12 hours, should be administered to assist in fetal lung maturation and for possible amelioration of symptoms [49]. The steroids are continued into the puerperium. As with other pre-eclampsia–eclampsia states, seizure

prophylaxis treatment is initiated with magnesium sulfate IV, blood pressure is controlled, and careful management of fluids and electrolytes is performed. After fetal lung maturation has been evaluated (ideally it is greater than 34 weeks'), or 24 to 48 hours of steroid therapy has elapsed in the case of progression of disease that is not responsive to therapy, then delivery should be considered. Although a trial of vaginal delivery is warranted, patients with HELLP syndrome demonstrated a cesarean section rate as high as 68% in selected series. Platelets are administered if the platelet counts are < 40,000/É L and a surgical delivery is planned. Efforts should be made to maintain the platelet count at > 20,000/É L for 24 hours after vaginal delivery and > 50,000/É L following surgically-assisted delivery. Modifications of the cesarean section technique using a vertical, rather than a Pfannenstiel, incision were suggested. A short course of antibiotics is given postoperatively and the mother should be maintained in an intensive care environment to monitor improvements in renal function and platelet counts. Decreased hemolysis will be evidenced by decreasing LDH levels and normalization of blood pressure. With optimal conditions, maternal mortality rates that ranged from 2% to 60% were reported. Higher mortality rates occurred in patients with the more severe forms of the syndrome or in patients whose disease was complicated by associated conditions, such as liver hemorrhage or rupture [5,8,49,98,115].

Amniotic fluid embolus was first described in 1926 and was not listed as a distinct heading in causes of maternal mortality until 1957 when it was labeled as obstetric shock. The overall incidence is unknown but is estimated to affect 1 in 8000 to 80,000 deliveries. It is a disease that spares the fetus. Maternal mortality rates from 61% to 85%, compared with neonatal mortalities of 21%, were reported. Recent reviews that were assisted by the maintenance of countrywide databases were helpful in outlining the incidence of this disease. Data from the United Kingdom reveal that compared with the decline in overall maternal deaths reported from 1970 until 1996 from all causes, maternal death ascribed to amniotic fluid embolus remained relatively constant at about 9%. Although the overall percentage is low, it remains the third leading cause of maternal deaths in the United Kingdom after thromboembolism and hypertensive disorders of pregnancy. It is associated with increased maternal age, multiparity, large gestations, chemically-induced labor, tumultuous or protracted labor, placental disruption, or signs of fetal distress, such as merconium-stained amniotic fluid. It may occur in vaginal or caesarean sections. Overall, the number of cases are small, which does not lead to a clear distinction of risk factors. The most common presenting sign is a sudden deterioration of maternal or fetal condition. This may be manifested by acute dyspnea, cyanosis, hypotension, bleeding, seizures, disseminated intravascular coagulation, or cardiopulmonary arrest. The pathophysiology is unknown and is thought to be mediated by leukotrienes or other arachidonic acid metabolites. It has similarities to anaphylactic shock. The diagnosis is largely one of suspicion; no confirmatory test exists. Treatment is supportive and includes CPR, fluid and pressor support of circulatory collapse, and treatment of coagulopathy. Prompt surgical delivery should be considered in cases of cardiopulmonary arrest [51,116–118].

Summary

The critical care aspects of obstetrics and pregnancy are varied and demand that critical care practitioners have a thorough knowledge of fetal and maternal changes in physiology as pregnancy progresses. Pregnancy can affect every organ system; and organ-specific conditions as well as syndromes that span multiple organ systems were described. Care of the critically ill, pregnant patient requires a true multidisciplinary approach for optimal outcomes. A review of the current concepts and suggestions for therapy were presented.