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A Historical Overview of Preeclampsia-Eclampsia

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Abstract

Preeclampsia is a hypertensive, multi-system disorder of pregnancy whose etiology remains unknown. Although management is evidence-based, preventative measures/screening tools are lacking, treatment remains symptomatic, and delivery remains the only cure. Past hypotheses/scientific contributions have influenced current understanding of preeclampsia pathophysiology and guided management strategies and classification criteria. To provide insight into how past hypotheses/scientific contributions have shaped current practice trends, this paper provides a historical overview of preeclampsia-eclampsia.

Keywords

Preeclampsia-eclampsia; historical overview; practice trends

Preeclampsia is a hypertensive, multi-system disorder of pregnancy that significantly contributes to maternal and fetal/neonatal morbidity and mortality (American College of Obstetricians and Gynecologists [ACOG], 2002; National Heart, Lung, and Blood Institute [NHLBI] National High Blood Pressure Education Program, 2000). At present, the etiology of preeclampsia remains unknown. As a result, preventative measures and screening tools are lacking, treatments are directed at the management of overt clinical manifestations, and delivery remains the only definitive cure (ACOG; NHLBI National High Blood Pressure Education Program; Norwitz & Repke, 2009).

Despite our inability to identify preeclampsia's etiology, hypotheses and scientific contributions throughout history have influenced our current understanding of preeclampsia pathophysiology. Such contributions are further reflected in current management strategies and classification criteria. To provide insight into how current practice trends have been shaped by past hypotheses and scientific contributions, this paper will provide a historical overview of preeclampsia-eclampsia from ancient times through present day. Although it is not the intent of this paper to provide an all-inclusive historical analysis of primary sources, attention will be directed toward an overview of theories on disease causation, treatments, and disease classifications extracted from available primary and reliable secondary sources.

Ancient Times

Theories on Disease Causation

In ancient Greece, between the late 5th and early 4th centuries before current era (BCE), the Hippocratics subscribed to the theory of the four humors to describe the cause of illness and disease. They believed that the body was made up of four humors (fluids) that included

blood, phlegm, yellow bile, and black bile. Health depended on a balance of the humors and any imbalance in the humors resulted in illness (Demand, 1994; Green, 1985).

The wet and dry theory was used to explain the vulnerability of female physiology to disease (Green, 1985). Women were considered wet while men were considered dry (Green; Trotula of Salerno, 1544/1940). Because a woman's flesh was porous and soft, she was at risk of drawing in too much moisture, resulting in an overabundance of fluids (humors) and subsequent illness (Green).

More specifically, many female ailments were attributed to the wandering womb. Central to this theory was that the uterus was able to physically uproot itself from its seat in the pelvis and travel anywhere within a woman's body in search of satisfaction. While Hippocrates believed that a dried up uterus wandered the body in search of moisture, Plato viewed the uterus as an animal that wandered because it was sexually unsatisfied and desired to make children (Thompson, 1999). Regardless of reason, as the uterus wandered the body, it was capable of wreaking havoc upon the liver, stomach, spleen, lungs (Thompson) and head (Veith, 1965), ultimately leading to disease.

Treatment

Because disease was believed to result from either an imbalance in the four humors, a woman's overly porous skin, or a wandering womb, treatments focused on the restoration and maintenance of internal balance and health. As a result, remedies to restore balance included altered diets, purging, and blood-letting. As for methods to maintain balance and health, the Hippocratics believed that a woman needed to be pregnant, lactating, or regularly menstruating. These methods were based on the belief that pregnancy, lactation, and menstruation provided mechanisms for the elimination of excess fluids. During pregnancy, extra blood was used to nourish the growing fetus. During lactation, extra blood was diverted to the breasts where it was converted to milk and eliminated through breast feeding. During menstruation, excess blood was eliminated through menstrual flow (Green, 1985).

Disease Classification

Preeclampsia-eclampsia was not formally classified as a disorder of pregnancy during Ancient times. Despite limited knowledge and technology, the writings of this period did demonstrate that the concept of eclampsia was recognized. For example, aphorism XXXI 507 in the Coan Prognosis states that a headache accompanied by heaviness and convulsions during pregnancy is considered bad (Hippocrates, 400 BCE/1950).

Middle Ages & the Renaissance

Theories on Disease Causation

During the Middle Ages, medical and scientific progress came to a standstill. Between 400 current era (CE) and 700 CE, Christianity greatly influenced such progress, for Christians were opposed to science and forbade human dissection (Cianfrani, 1960; Graham, 1951). Closing of medical schools at Athens and Alexandria by Byzantium Emperor Justinian in the 6th century further slowed medicine's progression (Cianfrani). Consequently, little original work was accomplished. Instead, individuals such as Oribasius, Aetius of Amida, and Paulus of Aegina, focused on the compilation and rewriting of the medical works of their predecessors (Cianfrani; Graham).

Between 700 CE and 1200 CE, Byzantium deteriorated, Christian influence began to decline, and Salernitan (Salerno, Italy) and Arabian influences increased. The first European medical school was opened in Salerno, Italy and a slightly altered four humors theory

emerged. Under this newer theory, it was believed that one humor dominated the other humors to form a new balance. Furthermore, the dominant humor determined an individual's physical and emotional characteristics (Cianfrani, 1960).

During the Renaissance, an acceleration in the progression of medicine resulted from the further waning influence of the church, freedom of the intellect, rearrangement of government and geography, and discovery of printing (Cianfrani, 1960). In 1537, Pope Clement VIII granted permission to teach anatomy by human dissection (Burton, 2005). With this new found freedom, anatomists and artists such as Jacopo Berengario da Capri (1460–1530), Nicolaus Massa (1499–1569), Leonardo da Vinci (1452–1519), Andreas Vesalius (1514–1564), and Fallopius (1523–1562) were instrumental in more clearly and accurately describing the female reproductive tract. For example, the first accurate description of the tubes and ovaries was provided by Fallopius. He is also credited with naming the placenta and indicating that it was only found in the uterus during pregnancy (Cianfrani).

In the seventeenth century, medicine continued to gain momentum and men began to enter the field of obstetrics (McMillen, 2003). Born in 1637, Frenchman Francois Mauriceau was one such man whose writings helped to establish obstetrics as a specialty (Speert, 1958). According to McMillen (2003), he was the first to systematically describe eclampsia and to note that primigravidas were at greater risk for convulsions compared to multigravidas. As for the causes of convulsions, Mauriceau attributed convulsions to either abnormalities in lochial flow or intrauterine fetal death. In the case of suppressed lochial flow, inflammation, pains in the head, convulsions, suffocation, and death could arise. In the case of intrauterine fetal death, a retained dead fetus gave off foul-smelling and cadaverous humours in the womb, predisposing a woman to convulsions (Mauriceau, 1668/1710).

Treatment

Treatment of disease during the Middle Ages was greatly influenced by Christian beliefs. Remedies prescribed by physicians in Ancient times were often replaced with charms, amulets, faith healing, miracles, and prayers (Cianfrani, 1960; Graham, 1951). However, as time passed and Christian influence waned, remedies similar to those used to treat disease in Ancient times became prominent again. For example, in an attempt to decrease cerebral congestion and prevent eclampsia, Mauriceau recommended two to three phlebotomies during pregnancy (Chesley, 1978; Chesley, 1984; McMillen, 2003).

Disease Classification

Near the end of the Renaissance, the classification of disease progressed. Gabelchoverus distinguished between four types of epilepsy in 1596, which included epilepsy resulting from the head, stomach, the pregnant uterus, and chilled extremities (Chesley, 1978). But it wasn't until 1619 that the word "eclampsia" first appeared in Varandaeus' treatise on gynecology (Ong, 2004).

18th Century through 19th Century

Theories on Disease Causation

In the 18th century, Boissier de Sauvages distinguished eclampsia from epilepsy. Along with the distinction he made in disease classification, de Sauvages offered his views on the cause of convulsions. He believed that convulsions resulted from nature trying to free the organism of any morbid element (Temkin, 1971).

Theories on disease causation continued to be proposed and thoroughly discussed in the writings of 19th century physicians. In his work entitled *Introduction to the Practice of Midwifery*, Dr. Thomas Denman (1821) focused much attention on the labors affected by convulsions. Although Denman attributed convulsions to certain customs and manners associated with living in large cities and towns, he noted that the greatest risk of convulsions came from the uterus. According to Denman, as the uterus expanded with pregnancy, greater pressure was placed upon the descending blood vessels. Such an increase in pressure led to the regurgitation of blood in the head and resulted in an overload of the cerebral vessels and subsequent convulsions (Denman, 1821).

In his 1849 work, *Parturition and the Principles and Practice of Obstetrics*, Dr. William Tyler Smith challenged the theory of cerebral congestion, for he believed that pregnancy was a state of increased fullness in circulation. Given that contractions during the second stage of labor normally interfered with the circulation of blood, he believed that more cases of convulsions would be observed if such congestion caused convulsions. In contrast, Smith attributed puerperal convulsions to several other causes: (1) any mechanical or emotional stimulus applied in excess to the spinal centre; (2) bloodletting; (3) variations in the wind, temperature, and other atmospheric changes; (4) irritation of the uterus, uterine passages, intestinal canal, and the stomach; and (5) “toxic” elements. As for Smith’s theory on “toxic” elements, he believed that preservation of health during pregnancy depended on the exponential increase in the elimination of wastes (e.g., secretions of the bowels) and debris from the maternal and fetal systems. Failure to do so resulted in a “toxemia” in which morbid elements accumulated in the blood causing irritation to the nervous center (Smith, 1849).

Treatment

Blood-letting remained a staple in the prevention and treatment of preeclampsia-eclampsia during the early 1800’s. The amount and frequency of blood-letting depended on the strength of the patient and symptom severity. Bleeding from the arm was attempted initially, but if convulsions continued, bleeding was repeated. In some cases the jugular vein or temporal artery were opened in an attempt to stop convulsions (Denman, 1821).

The use of opiates, warm baths, splashing of the face with cold water, and hastening of delivery were also recommended by Denman. Opiates were used to decrease irritability of the female constitution. If blood-letting and opiates failed, splashing of cold water to the patient’s face or placement of the patient in a warm bath were attempted. In cases where all treatments were unsuccessful, the physician had to choose between either hastening delivery or allowing natural labor to ensue. Per Denman, hastening of delivery was only to be attempted when a woman was physiologically ready (completely dilated, ruptured membranes, and fetus descended) because intervention in the early stage of labor increased maternal mortality (Denman, 1821).

When the theory of disease causation shifted to the toxin theory in the late 1800’s, treatments were targeted at the elimination of overabundant toxins. Those who believed that preeclampsia-eclampsia was caused by meat toxins restricted the consumption of meat and prescribed diets of fruits, vegetables, and milk products (Chesley, 1978). With the recognition of the pre-eclamptic state, women with headaches and edema of the superior extremities were admitted to lying-in hospitals where they underwent treatments such as bleeding and purging to prevent convulsions (Johns, 1843).

Disease Classification

After the introduction of the word “eclampsia,” Bossier de Sauvages (1739) differentiated eclampsia from epilepsy (Chesley, 1978; Friedlander, 2001). Eclampsia was acute in nature because convulsions resolved once the precipitating event was removed. Epilepsy was chronic in nature because convulsions recurred over time (Friedlander). Furthermore, eclampsia was not restricted to pregnancy. Severe hemorrhage, various sources of pain, vermicular infestations, and eclampsia associated with pregnancy were several species of eclampsia noted by de Sauvages (Chesley, 1978).

At the end of the 18th century and through the 19th century, the classification of pre-eclampsia-eclampsia continued to become more refined as the classic signs and symptoms of preeclampsia-eclampsia became more readily recognized. In 1797, Demanet noted a connection between edematous women and eclampsia (Chesley, 1978) while John Lever discovered albumin in the urine of eclamptic women in 1843 (Thomas, 1935). The connection between premonitory symptoms during the later months of pregnancy and the development of puerperal convulsions was also recognized in 1843 by Dr. Robert Johns. These premonitory symptoms included headache, temporary loss of vision, severe pain in the stomach, and edema of the hands, arms, neck, and face (Johns, 1843). In 1897, Vaquez and Nobecourt were credited with the discovery of eclamptic hypertension (Chesley, 1978). As a result of these contributions, the concept of the preeclamptic state was recognized. Physicians were now aware that the presence of edema, proteinuria, and headaches should raise concern about the possibility of convulsions (Sinclair & Johnston, 1858).

20th Century

Theories on Disease Causation

Although researchers in the 20th century failed to uncover the etiology of preeclampsia, much progress was made in the understanding of pathophysiological changes associated with its development. In the 1960's, several groups described dramatic differences in placental physiology between placentas from pregnancies affected by preeclampsia versus placentas from pregnancies unaffected by preeclampsia. Through the examination of placental bed biopsies, it was discovered that placental trophoblast cells failed to adequately invade maternal spiral arteries and convert the arteries from small muscular vessels into large, low resistant vessels in preeclampsia. With the lack of spiral artery conversion, arterial lumen diameter and distensibility was limited, resulting in restricted blood flow to the placenta and growing fetus (Brosens, Robertson, & Dixon, 1967; Brosens, Robertson, & Dixon, 1972; Gerretson, Huisjes, & Elema, 1981; Kong, De Wolf, Robertson, & Brosens, 1986).

Although these findings were instrumental in laying the groundwork for the current understanding of preeclampsia-eclampsia, not all theories or scientific discoveries have readily been accepted by the scientific community. Published in the American Journal of Obstetrics and Gynecology in 1983, the Hydatosi lualba (parasitic worm) theory of preeclampsia was one such theory quickly refuted by the scientific community. Under this theory, it was posited that the development of preeclampsia-eclampsia may be associated with the presence of a worm-like organism. Specimens collected from women with preeclampsia-eclampsia, including peripheral circulating blood, bloody fluid on the maternal surface of the placenta, and umbilical cord blood, were found to be positive for Hydatosi lualba (Lueck, Brewer, Aladjem, & Novotny, 1983). However, several other research groups demonstrated that starch powder from gloves, cellulose debris from common laboratory paper products, and alterations in staining technique produced the same characteristic worm-like organisms (Papoutsis, Irwin, Curry, & Zuspan, 1983; Sibai & Spinnato, 1983), which lead to refutation of the theory.

Unlike the parasitic worm theory, the theory posited by Roberts and colleagues in 1989 continues to guide research related to preeclampsia-eclampsia etiology. Dr. Roberts and colleagues posited that preeclampsia represented an endothelial disorder. Drawing on past work that associated preeclampsia with shallow trophoblast invasion and subsequent reduction in placental perfusion, they hypothesized that the ischemic placenta released a damaging factor(s) into the maternal circulation. Although factor identity was unknown, the circulating factor was hypothesized to have caused endothelial dysfunction and would lead to activation of the coagulation cascade, blood pressure abnormalities, and loss of fluid from the intravascular space (e.g., proteinuria) (Roberts et al., 1989).

Treatment

At the end of the 19th century and into the beginning of the 20th century, two very diverse approaches were used in the treatment of eclampsia. According to Chesley, physicians in Germany and the Netherlands advocated for aggressive management (e.g., prompt abdominal or vaginal cesarean section), but the associated maternal mortality rates were extremely high. As a result, a more conservative management gained popularity and was widely used up until the 1930's (Chesley, 1978; Chesley, 1984). Physician pioneers of this conservative method included Tweedy of Dublin and Stroganoff of Russia (Speert, 1958).

Tweedy's rationale for conservative management (as cited in Speert, 1958) was rooted in the belief that hastening of labor and delivery increased the occurrence of convulsions through the induction of reflex stimulation. Physicians were to abstain from vaginal examinations, abdominal palpation, massage of the kidneys, cold blasts of air, and the dilatation of the cervix in an effort to mitigate the risk of reflex stimulation. In contrast, his management plan revolved around patient sedation and included large doses of morphine. However, if a patient went into labor, he believed that the application of forceps was permissible given that the os would safely permit their application (Speert).

Because convulsions disrupted the functions of the heart, lungs, kidneys, and liver, Stroganoff's main objective was to eliminate convulsions (Speert, 1958). He treated the eclampsia, ignored the pregnancy, and waited for natural onset of labor. All examinations and treatments were performed under light anesthesia and sensory stimuli were reduced by keeping the patient's room dark and quiet (Chesley, 1984). Morphine and chloral hydrate were administered to keep patients sedated and to decrease frequency of convulsions. To restore respiratory function, oxygen was administered. To restore cardiac function, digitalis was administered if the pulse was found to be rapid and weak after a seizure (Chesley, 1984; Speert). Labor was to progress naturally and once a woman's cervix had dilated to six centimeters, the membranes were artificially ruptured (Chesley, 1984).

In addition to the diverse approaches to manage preeclampsia-eclampsia in the 20th century, the use of magnesium sulfate was introduced. Although a mainstay of current treatment, it was not until 1906 that Horn first used magnesium sulfate to manage preeclampsia-eclampsia (Chesley, 1984). During the 1920's, the parenteral use of magnesium sulfate in the treatment of preeclampsia-eclampsia was popularized by Lazard and Dorsett (Chesley, 1984), for Dr. Lazard's work (as cited in Gabbe, 1996) demonstrated that treatment with intravenous magnesium sulfate was both efficacious and safe.

Since the 1960's, few alterations have been made in the management of preeclampsia. Such consistency can be observed by studying the obstetrical textbooks used to educate students within the healthcare fields. Routine prenatal care (e.g., routine blood pressure measurement, urinalysis, maternal weight) remained a hallmark surveillance method since early signs and symptoms of preeclampsia may not be readily recognized by pregnant women. Once diagnosed with preeclampsia, management consistently included

hospitalization, frequent blood pressure measurement and weighing, urinalysis, bed rest, fetal surveillance, and assessment of maternal headache, blurry vision, and epigastric pain. With fulminating preeclampsia or the development of eclampsia, magnesium sulfate and antihypertensives were administered over the later part of the 20th century to prophylactically prevent or manage convulsions and acute hypertension, respectively. Ultimately, the decision to proceed with a vaginal delivery or cesarean section depended on a myriad of factors, including gestational age, condition of the cervix, and maternal and fetal condition (Eastman & Hellman, 1966; Hibbard, 1988; Pritchard & MacDonald, 1976).

Disease Classification

As with the treatment of preeclampsia, review of obstetrical textbooks provides insight into the changes made in preeclampsia-eclampsia disease classification throughout the 20th century (Table 1). According to Chesley (1978), the “pre-eclamptic” state was not included in textbooks until 1903. Furthermore, restriction of preeclampsia-eclampsia to the obstetric definition was not observed until 1961 (Chesley, 1978).

In the 13th edition of Williams Obstetrics (1966), preeclampsia-eclampsia fell under the category of the toxemias of pregnancy. According to the classification set forth by the American Committee on Maternal Welfare, toxemias of pregnancy included acute toxemia of pregnancy (preeclampsia and eclampsia), chronic hypertensive disease with pregnancy, and unclassified toxemia. Criteria for the diagnosis of preeclampsia included the presence of hypertension, edema, or proteinuria after the 24th week of gestation. Moreover, this classification required that a woman must meet only one of the three criteria to be diagnosed with preeclampsia (Eastman & Hellman, 1966).

In the 15th edition of Williams Obstetrics (1976), the term toxemia of pregnancy was replaced with hypertensive disorders of pregnancy. The Committee on Terminology of the American College of Obstetricians and Gynecologists recommended new classifications. The new classification of preeclampsia included the development of hypertension with proteinuria, edema, or both commencing after 20 weeks gestation (Pritchard & MacDonald, 1976).

In Hibbard’s 1988 text titled Principles of Obstetrics, the classification of preeclampsia underwent yet another revision. Although preeclampsia fell under the hypertensive disorders of pregnancy classification, it was further grouped under the term pregnancy induced hypertension. In addition to preeclampsia, hypertension that developed during pregnancy excluding the features of preeclampsia was also grouped under the term pregnancy induced hypertension. Mild to moderate preeclampsia was classified as the presence of hypertension and edema while severe preeclampsia was classified as the presence of hypertension and proteinuria with or without edema or cerebral or visual disturbances (Hibbard, 1988).

21st Century

Theories on Disease Causation

At present, the scientific community has failed to uncover the etiologic mechanisms responsible for the development of preeclampsia-eclampsia. As evidenced by the many review articles published in the scientific literature, the theories on disease causation are numerous and diverse. Such theories are related to mechanisms involving oxidative stress, immunologic intolerance between the fetoplacental unit and maternal tissue, and angiogenic imbalance (Leeman & Fontaine, 2008). For example, the endoglin protein, which is involved in regulation of placental trophoblast differentiation/invasion of the uterus (Caniggia, Taylor, Ritchie, Lye, & Letarte, 1997) and maintenance of vascular tone (Jerkic et al., 2004; Toporsian et al., 2005), represents an anti-angiogenic factor potentially involved

in preeclampsia development given that placental and blood pressure abnormalities are observed in preeclampsia.

Regardless of the mechanism, a two stage model of preeclampsia has been developed to provide a guiding framework for scientists in their search of disease causation (Hladunewich, Karumanchi, & Lafayette, 2007; Roberts & Gammill, 2005; Roberts & Hubel, 2009). For an in-depth review, consultation of the latest model iteration presented by Roberts and Hubel (2009) is recommended. Briefly, the model proposes that reduced placental perfusion (stage 1), secondary to abnormal implantation and subsequent vascular remodeling, interacts with maternal constitutional factors (genetic, behavioral, and environmental) to produce the maternal syndrome (stage 2) of preeclampsia. The systemic maternal syndrome is characterized by reduced perfusion brought about by vasospasm and activation of the coagulation cascade with the formation of occlusive microthrombi. This leads to reduced perfusion to multiple organs, hypertension, proteinuria, and loss of fluid from the intravascular space (Roberts & Gammill). Although it was initially thought that maternal factors only interacted with reduced placental perfusion to produce the maternal syndrome (stage 2), it is now believed that maternal factors may be involved in the genesis of reduced placental perfusion (stage 1). It has been further hypothesized that the linkage between stage one and two may involve multiple factors, whose constitution may vary from individual to individual. For example, recent suggestions indicate that the placentally derived “toxins” (e.g., cytokines, antiangiogenic factors, and syncytiotrophoblast microparticles) thought to link stage one and two may not be pathogenic. In contrast, it has been proposed that placental factors are appropriately released by the fetal/placental unit to increase nutrient availability, but are not tolerated by some women who develop preeclampsia (Roberts & Hubel).

Treatment

In an era of evidenced-based practice, the standardized care of women affected by preeclampsia-eclampsia is guided by the best available evidence. Based on the National High Blood Pressure Education Program Working Group on High Blood Pressure report (2000), the American College of Obstetricians and Gynecologists’ (ACOG, 2002) most recent practice bulletin indicates that current management of preeclampsia-eclampsia is reflective of past treatments. Although ACOG’s bulletin was published eight years ago, a more current review of evidence-based information on the management of preeclampsia further demonstrates that the mainstay of treatment has remained consistent (Norwitz & Repke, 2009). Despite consistent, evidenced-based management strategies, the etiology of preeclampsia remains unknown. As a result, effective preventative measures and screening tools are lacking, treatments remain directed at the management of overt clinical signs and symptoms, and the only definitive cure remains delivery (ACOG, 2002; NHLBI National High Blood Pressure Education Program, 2000; Norwitz & Repke). However, it is likely that our current evidence-based practices will continue to evolve as we gain a more comprehensive understanding of preeclampsia-eclampsia.

Diagnosis of preeclampsia continues to be based on prenatal blood pressure and urinary protein measurements and initial disease severity is evaluated with laboratory testing. Fetal well-being is monitored via fetal movement counts, non-stress tests, and biophysical profiles. Blood pressure and urine protein measurements, follow-up laboratory testing, and assessment of additional signs/symptoms suggestive of preeclampsia (headache, blurred vision, right upper quadrant or epigastric pain) are used to monitor maternal wellbeing. The timing and type of delivery ultimately depends on gestational age, maternal and fetal conditions, and the severity of preeclampsia. As for pharmacologic management, magnesium sulfate is administered during labor, delivery, and postpartum to prevent convulsions in women with preeclampsia or to deter recurrent convulsions in women with

eclampsia. In addition, antihypertensive therapy (e.g., hydralazine or labetalol) is administered to treat acute hypertensive episodes (NHLBI National High Blood Pressure Education Program, 2000; Norwitz & Repke, 2009).

Disease Classification

In 2000, the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy published a report with revisions to preeclampsia-eclampsia classification criteria. Preeclampsia is currently classified as a pregnancy-specific syndrome characterized by the presence of new-onset hypertension in a previously normotensive woman after 20 weeks gestation with proteinuria. Blood pressure (BP) criteria include a systolic BP >140 mm Hg or a diastolic BP >90 mm Hg. Proteinuria is defined as urinary excretion of ≥ 0.3 grams of protein in a 24-hour specimen, which correlates with a random $\geq 1+$ urine dipstick in the absence of a urinary tract infection. The presence of edema was dropped from the diagnostic criteria because many pregnant women with normal pregnancies develop edema. Furthermore, eclampsia is classified as the presence of seizures, non-attributable to other causes, in a woman diagnosed with preeclampsia. For additional information on classification of other hypertensive disorders of pregnancy (e.g., gestational hypertension or chronic hypertension, and preeclampsia superimposed on chronic hypertension), a review of the Report of the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy is recommended (NHLBI National High Blood Pressure Education Program, 2000).

Conclusion: Nursing Implications

This historical overview provides present day nurses with a broadened perspective of preeclampsia-eclampsia related to theories on disease causation, evolution of treatments, and refinement of disease classification. With such a perspective, nurses gain insight into how past hypotheses and scientific contributions have influenced and shaped current practices. Although the etiology of preeclampsia remains unknown, ongoing research has vastly improved our understanding of preeclampsia over the years and continues to guide evidence-based management of women with preeclampsia and refine its classification. However, as we continue to gain a more comprehensive understanding of preeclampsia, it is likely that the current practices utilized to care for women with preeclampsia-eclampsia will also evolve to reflect the most up-to-date scientific evidence related to preeclampsia etiology and treatment.

As in the past, the current role of nurses in the management of preeclampsia-eclampsia continues to revolve around the protection of maternal/fetal wellbeing and optimization of positive health outcomes. Given that effective preventative measures and screening tools are presently lacking, routine nursing assessments of the signs/symptoms indicative of preeclampsia-eclampsia remains critical to the detection, monitoring, and effective management of preeclampsia-eclampsia. Nurse-led patient education and the provision of a supportive environment are also essential to the optimal management of preeclampsia-eclampsia. Active participation in one's care can be promoted through nurse-led education related to self monitoring of fetal activity and maternal symptoms (e.g., headaches, blurred vision, epigastric pain). Furthermore, review of the rationale behind all tests (e.g., laboratory analysis, non-stress test) and treatments (e.g., magnesium sulfate, antihypertensive) keeps patients informed and may help to alleviate stress and anxiety during an emotionally and physically trying time. Ultimately, individually tailored and compassionate nursing care of women with preeclampsia-eclampsia will serve to enhance the wellbeing of mother and baby.

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Table 1Progression of preeclampsia-eclampsia classification during the 20th Century

Year & Citation	Milestone	
1903 Chesley (1978)	"pre-eclamptic state" included in textbooks	
1961 Chesley (1978)	preeclampsia-eclampsia restricted to the obstetric definition	

Obstetrical Textbook Publication Year & Citation	Terminology	Classification Description
1966 Eastman & Hellman (1966)	toxemias of pregnancy	A. acute toxemia of pregnancy (pre- eclampsia and eclampsia); chronic hypertensive disease with pregnancy; unclassified toxemia B. preeclampsia diagnostic criteria: presence of hypertension, edema, or proteinuria after 24 weeks gestation
1976 Pritchard & McDonald (1976)	hypertensive disorders of pregnancy	A. "toxemias of pregnancy" replaced with "hypertensive disorders of pregnancy" B. preeclampsia diagnostic criteria: development of hypertension with proteinuria, edema, or both commencing after 20 weeks gestation
1988 Hibbard (1988)	pregnancy induced hypertension	A. under the classification of hypertensive disorders of pregnancy, preeclampsia was further grouped under "pregnancy induced hypertension," which also included hypertension that developed during pregnancy excluding the features of preeclampsia B. preeclampsia diagnostic criteria: mild to moderate preeclampsia- presence of hypertension and edema; severe preeclampsia- presence of hypertension and proteinuria with or without edema or cerebral or visual disturbances after 20–24 weeks gestation