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Preventing deaths due to the hypertensive disorders of pregnancy

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In this chapter, taking a life cycle and both civil society and medically oriented approach, we will discuss the contribution of the hypertensive disorders of pregnancy (HDPs) to maternal, perinatal and newborn mortality and morbidity. Here we review various interventions and approaches to preventing deaths due to HDPs and discuss effectiveness, resource needs and long-term sustainability of the different approaches. Societal approaches, addressing sustainable development goals (SDGs) 2.2 (malnutrition), 3.7 (access to sexual and reproductive care), 3.8 (universal health coverage) and 3c (health workforce strengthening), are required to achieve SDGs 3.1 (maternal survival), 3.2 (perinatal survival) and 3.4 (reduced impact of non-communicable diseases (NCDs)). Medical solutions require greater clarity around the classification of the HDPs, increased frequency of effective antenatal visits, mandatory responses to the HDPs when encountered, prompt provision of life-saving interventions and sustained surveillance for NCD risk for women with a history of the HDPs.

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Introduction

The hypertensive disorders of pregnancy (HDPs), chronic (or pre-existing) hypertension, gestational hypertension and, especially, pre-eclampsia, remain leading causes of maternal and perinatal morbidity and mortality as well as identifying individuals at increased risk for premature cardiovascular disease [1,2].

Diagnosis and classification

Here we use the Canadian definitions and diagnostic criteria for, and classification of, the HDPs [3].

Blood pressure (BP) should be measured at each antenatal visit. Hypertension in pregnancy is defined by a systolic BP (sBP) $\geq$140 mmHg and/or diastolic BP (dBP) $\geq$90 mmHg [3]. Severe hypertension is defined as sBP$\geq$160 mmHg (instead of 170 mmHg) as that level of sBP reflects stroke risk [3–7]. Elevated BP should be confirmed by repeat measurement, at least 15 min apart, being measured three times, the first value being disregarded and the average of the second and third taken as the BP value for the visit [3].

We suggest screening with a urinary dipstick at each antenatal visit. Proteinuria diagnosis can be performed on random samples (by a urinary dipstick or protein: creatinine ratio (PrCr)) or timed urine collections (usually 24 h). Quantification of urinary protein by 24-h urine collection is often inaccurate [8] and has been replaced by spot urine samples outside pregnancy [9]. A dipstick value of $\geq$++ proteinuria and PrCr of $\geq$30 g/mol represent significant proteinuria of 0.3 g/d, the current if flawed gold standard (SOGC 32); a threshold up to 40 g/mol may be more appropriate in multiple pregnancies (SOGC 33,34). Proteinuria should be quantified (by PrCr or 24-h urine collection) if pre-eclampsia is suspected, as dipstick proteinuria <++ has a significant false negative rate.

Chronic hypertension is defined as hypertension that predates pregnancy, or appears before 20 weeks or persists more than 3 months postpartum. It can be complicated by superimposed pre-eclampsia [3].

Gestational hypertension appears at $\geq$20 weeks and resolves by 3 months postpartum [3]. Associated risks depend on the gestational age at presentation and progression to pre-eclampsia; gestational hypertension at <34 weeks is associated with a $\approx$35% risk of pre-eclampsia, which takes an average of 5 weeks to develop [3].

Pre-eclampsia is the HDP associated with the greatest risks, particularly when it is severe or present at <34 weeks [3,10,11]. The risk of small for gestational age (SGA) infants is primarily among women who present at <34 weeks, with macrosomia more common with the term pre-eclampsia [12]. Pre-eclampsia is most commonly defined by new-onset proteinuria and potentially other end-organ dysfunction. Table 1 outlines the end-organ dysfunction of pre-eclampsia: 'adverse conditions' and 'severe complications'. 'Adverse conditions' consist of maternal symptoms, signs, abnormal laboratory results and abnormal foetal monitoring results that may herald development of severe maternal or foetal complications (including stillbirth). The 'adverse conditions' are those for which we wait and to which we respond (e.g., low oxygen saturation) to avoid the severe complications that we wish to avoid entirely (e.g., pulmonary oedema). Remember that a significant minority of women will present with unheralded eclampsia [13]. That response could be more intensive maternal or foetal monitoring, specific treatment or delivery. In the Canadian rubric, 'severe complications' of pre-eclampsia warrant delivery, irrespective of the gestational age [3].

It must be recalled that delivery does not 'cure' pre-eclampsia; it only initiates the recovery from the disease – and is the only intervention that will do so. Especially with early-onset pre-eclampsia, that arising before 34 weeks of gestation, many women experience a transient deterioration in their clinical state, with nadirs in platelet count and renal function and maximal liver enzyme abnormalities, before their recovery. Frequently, this period of clinical vulnerability may last up to 72 h postpartum [1,2]. In more developed countries, those women who die due to the consequences of pre-eclampsia almost uniformly do so postnatally.