NARCHI BULLETIN

SJH, Issue 2, November 2018

REACHING THE UNREACHED

NARCHI

Delhi Branch

Dedicated to "Dyspnea in Pregnancy"

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Dr Jyotsna Suri & Dr Rekha Bharti on behalf of National Association for Reproductive & Child Health of India, Delhi Branch.

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President's Message

I welcome you all to the second issue of our NARCHI bulletin; hope you all had a wonderful and sparkling festival season. With all the celebrations, also come respiratory problems for all, especially the vulnerable ones, pregnant women and the children. To deal with this problem our first CME after Diwali, held at Safdarjung Hospital was on 'Obstetric Dyspnoea: Management Approach'. It was attended by delegates from all over Delhi, and was well appreciated.

In the midst of the celebration, we have another great news which I take pride in announcing that Delhi is the host for the **NARCHI World Congress 2020.** To make this a success and a memorable event for people all over India, I need whole hearted participation and support from my esteemed members.

A reminder to all the members for our upcoming **25th Annual Conference** on 23rd and 24th February 2019 at Hotel Eros, Delhi, themed **"3Ps: Predict, Prevent and Practice"**. I request you all to register for the conference and invite others for the same, as it is going to be an academic feast as well as a fun filled bonanza.

Keeping the theme of conference in mind we are planning two more events: A **'Walkathon for Thalassemia Prevention'** on 2nd December 2018 at Inner Circle, Connaught Place and a **'Cultural Program and Dinner'** for Thalassemia Welfare on 23rd February at Hotel Eros, Delhi.

Hope to see you all joining for the cause!

For now happy reading...

Dr Achla Batra

Nominations are invited for the President and Vice-President, NARCHI Delhi, 2020-2022
The applications may be sent to NARCHI secretariat, Safdarjung Hospital

Secretary's Message

Greetings from NARCHI Delhi Branch!

On behalf of the Department of Obstetrics & Gynecology, VMMC and Safdarjung College it gives me great pleasure to bring forth this second quarterly issue of NARCHI Delhi bulletin dedicated to 'Dyspnoea in Pregnancy'. I congratulate the editorial team for their immense effort in conceptualizing and shaping up this wonderful issue.

Thank you all for appreciating and providing feedback on our first issue. The current issue also deals with an important issue in Obstetrics. It emphasizes the importance of providing optimal health care services, multi-disciplinary approach to maternal care to enhance diagnosis and management of undiagnosed preexisting medical and obstetric complications leading to dyspnoea in pregnancy.

Our NARCHI team has been doing all efforts in direction of fulfilling our motto of 'Reaching the Unreached' by organizing various health camps, awareness campaign and numerous CMEs on various aspects of maternal and reproductive health. We have the arduous task of keeping forth the momentum going. With a dynamic team of vibrant and energetic office bearers in NARCHI, we have our endevours focused on promoting maternal, child and reproductive health through our educational programs.

Continuing with the momentum we are preparing full-fledged for the 25th year celebrations of NARCHI Delhi which start with a "*Walk for Thalassemia Awareness*" in December first week and later the Annual Conference of NARCHI on theme of "*Optimizing Maternal & Child Health: Prevention, Prediction & Practice*". We hope that the NARCHI members will participate in large numbers and benefit from the elaborate scientific sessions and targeted workshops which are being meticulously prepared to cover a wide range of topics.

I also take this opportunity to congratulate Dr Achla Batra, our President for winning the prestigious bid for NARCHI World Congress in 2020. I humbly seek suggestions, cooperation and involvement of all the NARCHI members to make this endevour successful.

Happy Reading to all NARCHI Members!

Dr Monika Gupta

Editor's Message

Greetings to everyone on behalf of the editorial team! We are extremely grateful for the immense positive feedback on our first edition. The encouragement from all of you motivates us to work hard and give our best. The NARCHI team hopes that you like this edition.

Pregnant women often complain of breathlessness that can cause anxiety to both woman and the treating obstetrician. At one end too many unnecessary physician and cardiology referrals can be distressing; however missing on important warning signs can cause severe morbidity and mortality. We understand that dyspnea in pregnancy can be a physiological condition but it may be a symptom of an underlying life threatening disease, so it is important for the obstetricians to be vigilant when pregnant women complain of breathlessness. Keeping this in mind we have decided to dedicate this issue to the topic- **'Dyspnea in Pregnancy'.**

Our aim is to equip obstetricians with the information about this condition so that they are able to do the basic workup and initiate multidisciplinary approach when required. Along with providing an overview of dyspnea in pregnancy, this issue covers a wide range of topics like cardiogenic pulmonary edema that can be with or without hypertension, management of hypertensive disorders in pregnancy and emergent treatment of acute severe hypertension. The issue also includes basics in interpretation of ABG as it is important for obstetricians to be aware of the normal alterations in ABG during pregnancy so that they can pick up the pathology in time, because what is normal for non pregnant women may not be normal for pregnant women.

Look out for the Quiz and mail your answers to **narchidelhisjh@gmail.com.**

We hope that you all enjoy reading this issue and benefit from it in your daily practice. Happy reading!

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Overview of Dyspnea in Pregnancy

Jyotsna Suri[1], Rekha Bharti[2]
[1]Professor, [2]Asssociate Professor, Department of Obstetrics & Gynaecology, VMMC & Safdarjung Hospital

Dyspnoea in pregnancy is a very common symptom which affects 60-70% of the gravid women. It is often a dilemma for the treating obstetrician to determine whether this symptom is due to the physiological changes of pregnancy or whether it indicates a serious underlying pathology.

Knowledge of the physiological changes which take place in pregnancy is important to appreciate the difference between dyspnea due to pregnancy as compared to that of pathological origin. The physiological changes which affect the respiratory system and may be responsible for dyspnea of pregnancy are shown below:

Physiological changes of respiratory system in pregnancy

The respiratory tract undergoes many changes during pregnancy, mediated initially by changes in the endocrine system and later by the enlarging uterus, in order to provide oxygen for increased maternal demands and for fetal physiology. These changes act to lower maternal PCO2 to half that of the fetus, thereby facilitating more effective gas exchange. Oxygen consumption in pregnancy increases by 30–50 mL/min, two-thirds of which covers additional maternal requirements and one-third is for the developing fetus**.**

Under the effect of progesterone there is an increase in the depth of respiration though the rate remains fairly constant which gives rise to the clinical picture of dyspnoea. *The minute volume which is product of respiratory rate and tidal volume and is defined as the amount of air moved into and out of the lungs in 1 minute is increased in pregnancy because of increase in the tidal volume by 40%*. The minute volume also increases by 40%, from 7.5 L/min in nonpregnant to 10.5 L/min in pregnant women. This results in a fall in the arterial pCO2 from a normal of 40mmHg to a level of 30mmHg in pregnancy. To compensate for this respiratory alkalosis there is an increased excretion of the bicarbonate by the kidneys, leading to a fall of serum bicarbonate to about 20 mEq/L from a normal of 24 mEq/L. *This compensated respiratory alkalosis can result in a decreased buffering capacity for further metabolic acidosis (as in sepsis)*.

Besides these changes there are other changes, such as decrease in the expiratory reserve volume and residual volume, leading to decrease in functional residual capacity by about 500ml. However it is the 20% fall in residual volume which further increases alveolar ventilation. *The decrease in functional residual capacity can result in rapid fall in oxygen in response to respiratory insults.* Vital capacity, which is the maximum volume of air, expired after maximum inspiration however remains unchanged in pregnancy.

There are several causes of dyspnea in pregnancy. They can be divided into pulmonary, cardiac, metabolic, miscellaneous and causes specific to pregnancy. The causes are shown in Table 1

Evaluation of pregnant patient with dyspnea

History and examination

Duration of symptoms- Physiological dyspnea is gradual in onset and does not worsen with increasing gestational age. Dyspnea due to heart failure may also be gradual in onset but worsens progressively and may present in the third trimester as acute respiratory failure. Pulmonary embolism, acute asthma and pneumothorax are always acute in onset. Acute asthma may be associated with other IgE mediated conditions like urticaria, flushing, itching and angioedema. Pulmonary embolism is mostly accompanied by pleuritic chest pain and others symptoms like cough and hemoptysis.

Gestational age at time of symptoms- The physiological dyspnea normally appears in the first or second trimester whereas Peripartum cardiomyopathy normally manifests around 36 weeks. Heart failure due to rheumatic heart disease becomes symptomatic in late second trimester and peaks around 32 weeks.

Presence of cough and wheezing- Cough and wheezing are not present in physiological dyspnea. Presence of these symptoms goes in favour of asthma or cardiac dyspnea. Acute cough is often indicative of respiratory tract infection.

Findings on auscultation of the chest- The lungs are

Table 1: Causes of dyspnea in pregnancy

Pulmonary	Cardiac	Metabolic	Miscellaneous	Specific to pregnancy
COPD	Rheumatic heart disease	Sepsis	Anxiety	Preeclampsia
Asthma	cardiomyopathy	Diabetic ketoacidosis	Hyperventilation	Eclampsia
Pneumonia	High output failure	Anaemia	Anaphylaxis	Peripartum cardiomyopathy
ARDS	Arrhythmias	Salicylate, CO and organophosphorus poisoning	Ascites/effusion	Tocolytic induced pulmo- ary edema
Pulmonary embolism	Cardiac tamponade		Stroke	Amniotic fluid embolism
Pneumothorax	Acute coronary syndrome		Massive obesity	Pregnancy induced physiological dyspnea
Pulmonary contusion	Decompensated heart failure			

clear on examination in physiological dyspnea. Fine crackles are suggestive of abnormalities affecting the distal lung parenchyma, such as interstitial pulmonary edema from left ventricular failure or a variety of forms of interstitial lung disease. Wheezing represents airway obstruction and is seen in acute asthma and bronchitis.

Investigations

- Chest Radiography after abdominal shielding
- Pulmonary function tests especially in cases which are suspected to be due to asthma
- ABG for quantifying severity of problem & monitoring ventilation (target PCO2 is 30-32 mmHg)
- ECG to differentiate the dyspnea of respiratory origin from that of cardiac origin.
- ECHO to differentiate the dyspnea of respiratory origin from that of cardiac origin.
- Bedside USG of lung to look for pulmonary edema (B lines) and inferior vena cava collapsibility to assess fluid status of patient
- Other investigations according to differential diagnosis□ radionuclide scan (V/Q perfusion) in cases suspected to have pulmonary embolism; brain natriuretic peptide (BNP levels may be useful in the patient with suspected ventricular dysfunction as the cause of dyspnea

Important causes of acute respiratory failure in pregnancy

Acute respiratory failure in pregnancy presents as sudden onset shortness of breath and can be due to life threatening conditions like

- Pulmonary edema
- Community-acquired pneumonia
- Aspiration
- Pulmonary embolism
- Asthma exacerbation
- Peripartum cardiomyopathy
- Amniotic fluid embolism
- Venous air embolism

Signs of acute respiratory failure

- Rapid shallow breathing- RR > 25/min
- Use of accessory muscles
- Confusion, agitation, somnolence
- Crackles, wheeze, whispering- pectoriloquy, bronchophony
- SpO2 < 90%
- PaO2 < 60 mm Hg

Initial management of patient with acute respiratory failure

Initial management of all cases with respiratory failure is same with the aim to stabilize the patient. Only after the patient is stabilized is the definite management instituted according to the diagnosis. The steps of initial management are

- Oxygenate – nasal prongs/face mask/non rebreather mask (GOAL SPO_2 >95% and PaO_2 >70mm Hg- when fetus in utero)
- Early decision for intubation and ventilation if oxygenation not possible with oxygen devices or the patient has a low Glasgow Coma Scale.
- Secure IV access and maintain circulation of the patient

Some important clinical situations which are causes of acute respiratory failure and acute dyspnea are discussed below:

Acute pulmonary edema

Pulmonary edema represents a final common pathway of several complications of pregnancy and the peripartum period. Acute pulmonary edema (APE) in pregnancy is an important cause of morbidity and mortality and is a frequent reason for admission to ICU. The pregnant women are at an increased risk of APE due to physiological and sometimes pathological changes of pregnancy leading to reduced cardio-respiratory and metabolic reserve. The common causes of APE in pregnancy are preeclampsia, cardiomyopathy and inappropriate fluid administration. Early identification of high risk pregnant women and management with highly skilled multi-disciplinary team is essential. During management special consideration should be given to altered physiological targets of pregnancy, difficult airway, risk of aspiration, presence of fetus and prevention aorto-caval compression during resuscitation. A multidisciplinary team is required for the safe planning of the fetal delivery.

Management of acute pulmonary edema

- Oxygenate- 8-10 L/min with face mask
- Prop up the patient
- Furosemide 20-40 mg over 2 minutes
- Repeat dose 40-60 mg after 30 minutes if no response

(CAUTION: pre-eclamptics tolerate fluid depletion very poorly)

- Fluid restriction
- Positive pressure ventilation
  - Non Invasive in selected cases
  - Invasive mechanical ventilation

Acute respiratory distress syndrome (ARDS)

Acute respiratory distress syndrome (ARDS) is an acute, diffuse, inflammatory lung injury, characterized by increased pulmonary vascular permeability; increased lung weight; loss of aerated tissue.

Clinical hallmarks of ARDS are hypoxemia and bilateral radiographic opacities*;* and pathological hallmark is diffuse alveolar damage. **The diagnosis according to the Berlin criteria** requires the presence of all of the following:

- Respiratory symptoms within a week of clinical insult.
- Bilateral opacities consistent with pulmonary edema in X-Ray chest or CT Scan
- Cardiac failure and fluid overload to be ruled out
- Moderate to severe impairment of oxygenation defined as ratio of arterial O2 tension to fraction of inspired air (PaO2/FiO2)- Moderate <200; Severe <100

The etiology of ARDS in pregnancy may be classified into 3 categories

1. **Unaffected by pregnancy:** *Direct/ pulmonary causes*- Bacterial pneumonia, Ventilator associated lung injury (VALI), inhalational injury, chemical pneumonitis, near drowning, pulmonary contusion, fat embolism; *Indirect /extra-pulmonary causes*- sepsis, trauma, burns, acute pancreatitis, transfusion-related acute lung injury (TRALI)
2. **Modified by pregnancy:** *Direct*- aspiration of gastric contents, viral pneumonitis (H1N1), blastomycosis, coccidomycosis, listerosis, venous air embolism; *Indirect*- pyelonephritis, malaria, dengue fever
3. **Unique to pregnancy***: Direct*- tocolytic associated pulmonary edema, amniotic fluid embolism, trophoblastic embolism; *Indirect*- preeclampsia-eclampsia, HELLP, AFLP, chorioamnionitis, endometritis

Clinical features of ARDS

Normally appear within 6 to 72 hours of an inciting event, with rapid deterioration. The presenting features are typically, dyspnea, cyanosis (ie, hypoxemia), and diffuse crackles. There is evidence of respiratory distress, with presence of tachypnea, tachycardia, diaphoresis and accessory muscles use. Cough and chest pain may be present. Clinical findings related to the precipitant may be present for example in a case of puerperal sepsis there may be high grade fever and foul smelling purulent lochia.

Management of ARDS

The principles of management are:

- Supportive care
- Identify the cause of ARDS
- Treat the cause
- Formulate a plan for fetal monitoring/delivery

Dilutional anemia and decreased plasma oncotic pressure in pregnancy increase the risk for pulmonary edema, which along with increased cardiac output and supine hypotension make management of ARDS more challenging.

To prevent ventilator associated lung injury, ventilator parameters recommended by NIH-ARDS network protocol are summarized in Table 2.

Table 2: Recommended Ventilatory Parameters

Ventilatory Parameters	NIH-ARDS network protocol
Tidal volume	≤6 ml/kg
Plateau pressure	≤30 cm H_2O
Ventilation set rate/pH goal	6-35/min to achieve pH ≥7.30
Inspiratory flows, I:E ratio	Flow to achieve I:E ratio 1:1-1:3
Oxygenation goal	PaO_2 ≥70 mmHg, SPO_2 ≥95%
FiO_2/PEEP	0.3/5, 0.4/5-8, 0.5/8-10, 0.6/10
Weaning	When FiO2/PEEP 0.4/8, with PSV

Pulmonary embolism

Pregnant women are at risk of pulmonary embolism by 5-6 times than the general population. The patient presents with dyspnea, pleuritic chest pain, leg pain or swelling, tachypnea, tachycardia, and hypoxemia. Circulatory collapse can occur, but it is uncommon.

The diagnosis is confirmed by imaging- Computed tomography pulmonary angiography (CT-PA) and low-dose ventilation perfusion (V/Q) scanning are good imaging modalities, but conventional pulmonary angiography should be avoided.

Anticoagulation is the mainstay therapy and should be started with a high index of clinical suspicion considering the life threatening nature of the condition. Adjunctive therapies include supplemental oxygen and, if necessary, ventilatory assistance. Thrombolysis has been performed in the setting of circulatory collapse, but the risk of bleeding and its sequelae are high.

Conclusion

Dyspnea in pregnancy can be either physiological or pathological. Physiological dyspnea is due to changes in the respiratory tract and does not require any treatment; however it is very important to differentiate from pathological dyspnea by taking a good history, performing a complete respiratory and cardiovascular examination; and by conducting the relevant investigations like echocardiography and pulmonary function tests.

Suggested reading

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Hospitals are great places, and you can learn from them, but you don't necessarily need to go in anytime you get the sniffles. And maybe you shouldn't treat pregnancy as a disease. **Sarah Wayne Callie**

Hypertensive Disorder in Pregnancy

Harsha Gaikwad
Professor, Vardhman Mahavir Medical College & Safdarjung Hospital, New Delhi

Hypertensive disorders are the most common and yet serious conditions seen in obstetrics. These disorders complicate 5 to 10 percent of all pregnancies, and together they are one of the deadly triads, along with haemorrhage and infection, that contributes greatly to maternal morbidity and mortality rates.

In the World Health Organization (WHO) systematic reviews of maternal mortality worldwide, and in developed countries, 16 percent of maternal deaths were attributed to hypertensive disorders.

Of hypertensive disorders, the preeclampsia syndrome, either alone or superimposed on chronic hypertension, is the most dangerous. Preeclampsia is identified in 4 to 5 percent of all pregnancies. Also, new-onset hypertension during pregnancy known as gestational hypertension, is followed almost half the time by signs and symptoms of preeclampsia.

Terminology and classification of hypertensive disorders

To update and codify the terminology and classification of hypertensive disorders of pregnancy, a Task Force of the American College of Obstetricians and Gynaecologists (2013) has provided evidence-based recommendations for clinical practice. The previous basic classification was retained and describes four types of hypertensive disease:

1. Preeclampsia and eclampsia syndrome
2. Chronic hypertension of any aetiology
3. Preeclampsia superimposed on chronic hypertension
4. Gestational hypertension- definitive evidence for the preeclampsia syndrome does not develop and hypertension resolves by 12 weeks postpartum.

Diagnosis of hypertensive disorders

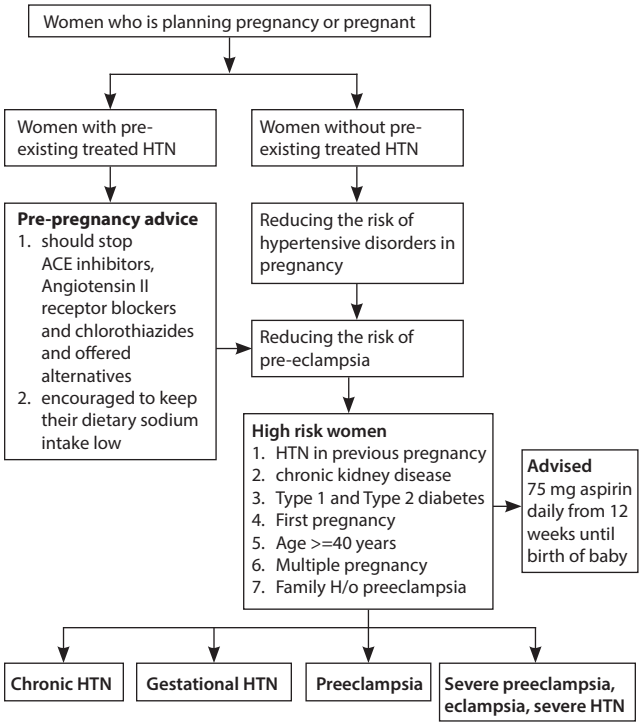
Hypertension is diagnosed empirically when appropriately taken blood pressure exceeds 140 mm Hg systolic or 90 mm Hg diastolic. Korotkoff phase V is used to define diastolic pressure. However, if the sounds are audible with the cuff deflated, Korotkoff IV sound should be used for diastolic blood pressure. Blood pressure is obtained after five minutes of rest, in the sitting position, feet touching the ground, legs uncrossed. If taken in semi-Fowler position back should be supported. Arm should be supported and kept at heart level. Blood pressure can also be measured in the left arm in left lateral position. Cuff used should be of appropriate size with width of bladder 40 percent of arm circumference and should encircle 80 percent of the upper arm.

Condition	**Criteria required**		
Gestational hypertension*	a) BP> 140/90 mmHg after 20 weeks in previously normotensive patient b) No proteinuria c) Return of BP to normal within 12 weeks after delivery		
Preeclampsia	New onset hypertension plus a) New onset proteinuria: - 300 mg or more in 24 hours or - urine protein: creatinine ratio ≥0.3, or - dipstick 1+ persistent b) thrombocytopenia: platelets <100,000/mm3 c) renal insufficiency: serum creatinine (>1.1 mg/dl) d) liver involvement: Elevated liver enzyme, especially AST or ALT e) cerebral involvement: headache, visual disturbances and convulsion Depending on the markers listed above preeclampsia can be 'severe' and 'without severe features'.		
Severity of hypertensive disorders	**Abnormality**	**Preeclampsia without severe features**	**Severe preeclampsia**
	Systolic BP	<160 mmHg	≥160 mmHg

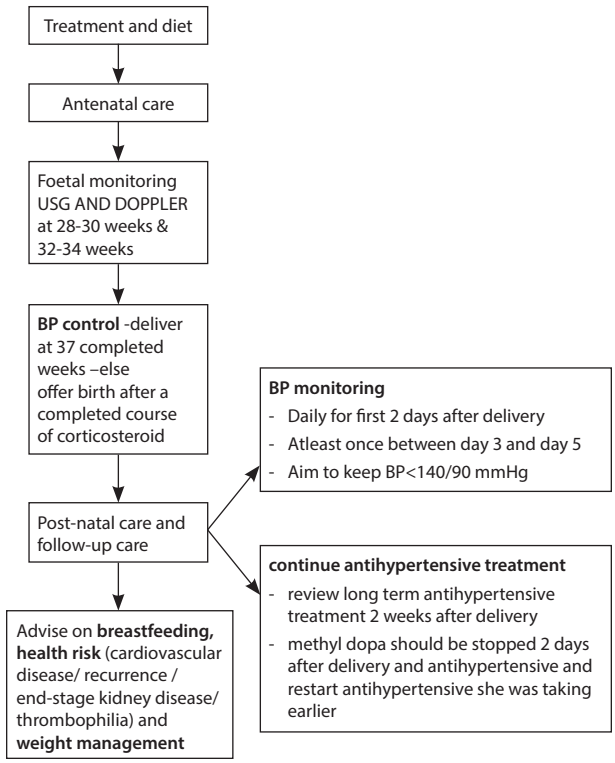
	Diastolic BP	<110 mmHg	≥110 mm Hg
	Proteinuria	None to positive	None to positive
	Headache	Absent	Present
	Visual disturbances	Absent	Present
	Upper abdominal pain**	Absent	Present
	Oliguria	Absent	Present
	Serum creatinine	Normal	>1.1 mg/dl
	Platelet count	≥100.000/µl	<100,000/µl
	Serum transaminase elevation	Minimal	≥twice normal concentration
	Pulmonary oedema	Absent	Present
Eclampsia	Development of convulsion in case of pre-eclampsia, in absence of other causes of convulsions		
Chronic hypertension	a) A known hypertensive woman becoming pregnant or BP 140/90mmHg or more diagnosed before 20 weeks of pregnancy in absence of gestational trophoblastic disease. b) Hypertension first diagnosed after 20 weeks but persisting 12 weeks after delivery		
Superimposed Pre-eclampsia	a) Onset of proteinuria of 300 mg/24 hours or more in a woman who had hypertension but no proteinuria before 20 weeks of gestation b) Development of sudden increase in proteinuria, hypertension or thrombocytopenia in woman who had hypertension or proteinuria before 20 weeks of gestation.		

* The final diagnosis is made after 3 months of delivery. If BP remains high after 3 months postpartum, diagnosis is changed to chronic hypertension.
**Unresponsive to medication and/or not accounted for by alternative diagnosis.

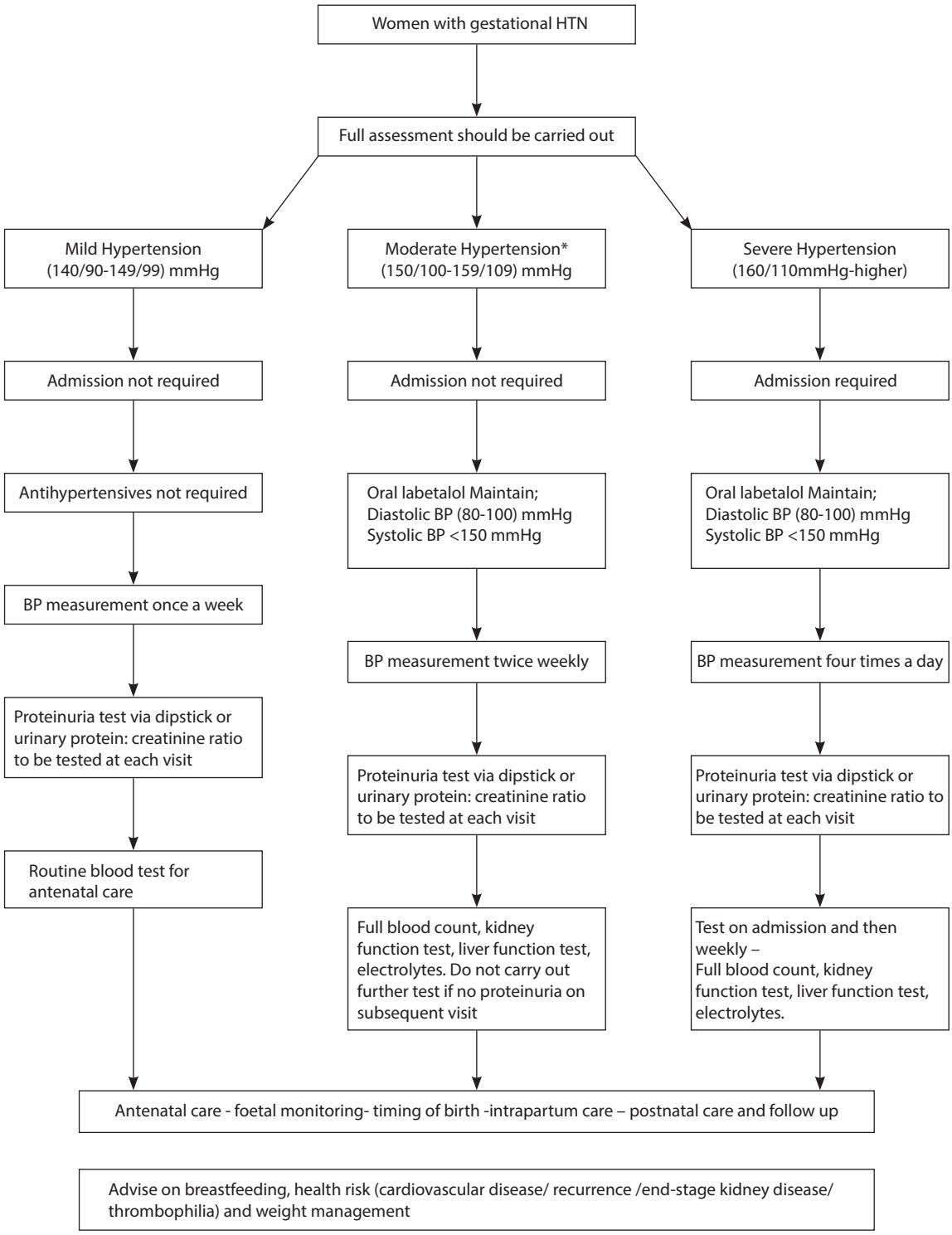
An overview of hypertensive disorders in pregnancy



Management of Chronic Hypertension



Management of Gestational Hypertension



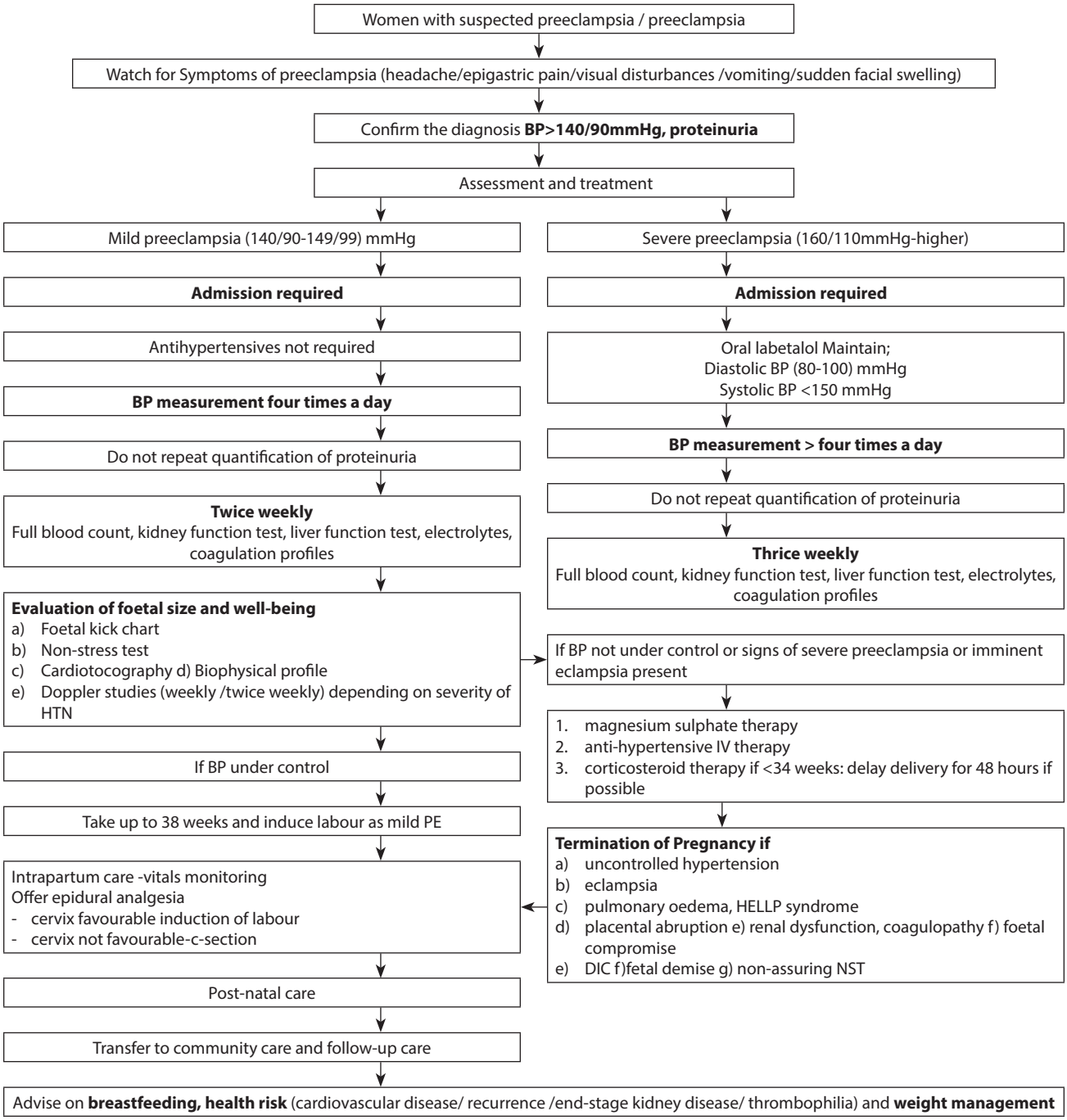
*US Task force for hypertension in pregnancy does not recommend antihypertensive medication in women with gestational hypertension or preeclampsia with a persistent BP of less than 160 mmHg systolic or 110 mmHg diastolic.

Management of Preeclampsia

Diagnosis of preeclampsia should be confirmed before managing it. The management depends upon the following factors:

a) Severity of preeclampsia as determined by the various factors mentioned above
b) Gestational age
c) Condition of the cervix (Bishop's score)
d) Foetal condition

Task Force (2013) recommends more frequent prenatal visits if preeclampsia is "suspected." Heightened surveillance permits more prompt recognition of ominous changes in blood pressure, critical laboratory findings, and clinical signs and symptoms (Macdonald-Wallis 2015)

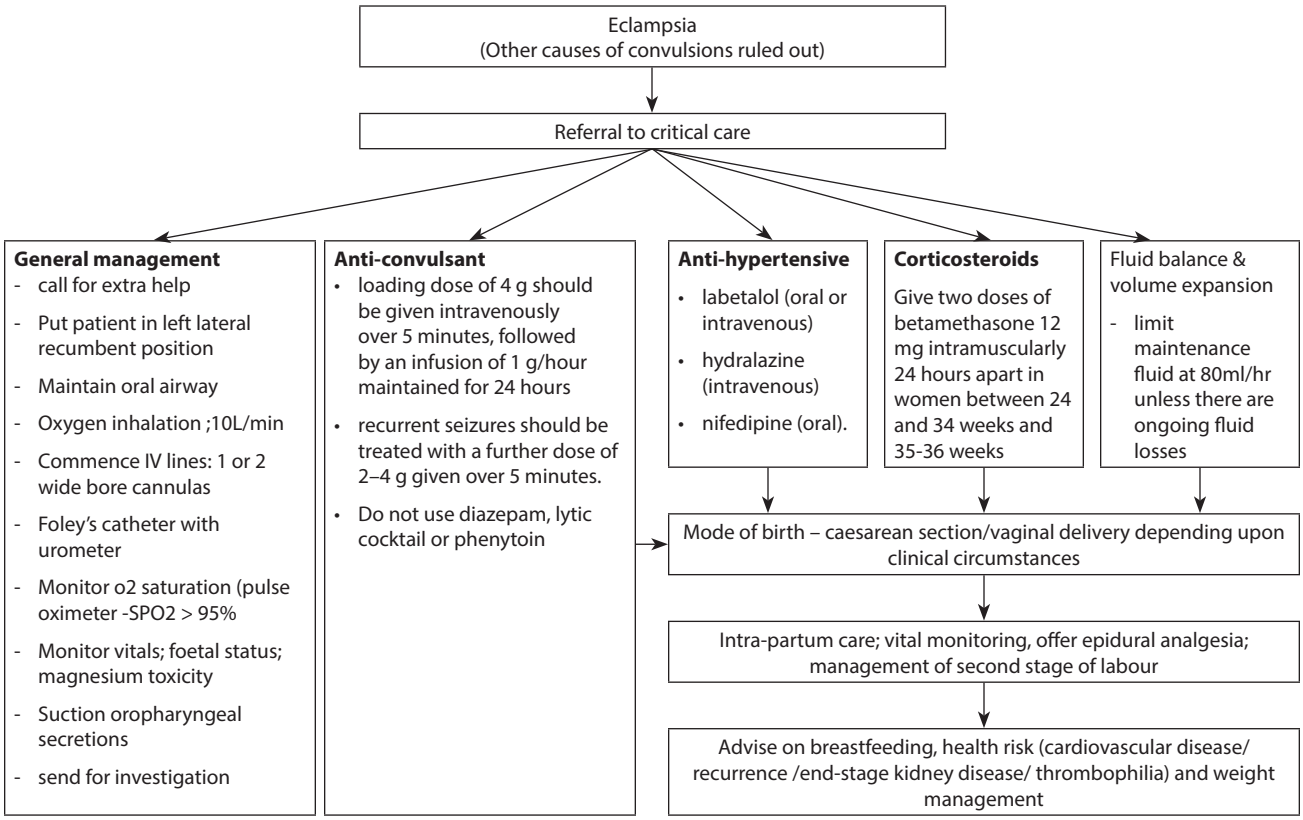


Management of Eclampsia

Eclampsia is when preeclampsia complicated by generalized tonic-clonic convulsions. Principles of management includes: -

1. General management
2. Control of seizures
3. Control of blood pressure
4. Obstetric management

Early detection and timely and appropriate management of complications



Suggested Reading

1. Khan KS, Wojdyla D, Say L, et al: WHO analysis of causes of maternal death: a systematic review. Lancet 367:1066, 2006
2. American College of Obstetricians and Gynecologists: Chronic hypertension in pregnancy and superimposed preeclampsia. In: Hypertension in Pregnancy. Report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. 2013
3. American College of Obstetricians and Gynecologists: Hypertension in pregnancy. Report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. Obstet Gynecol 122:1122, 2013
4. Macdonald-Wallis C, Silberwood RJ, de Stavola BL, et al: Antenatal blood pressure for prediction of preeclampsia, preterm birth, and small for gestational age babies: development and validation in two general population cohorts. BMJ 351:h5948, 2015
5. The NICE guidance that was used to create this part of the interactive flowchart.Hypertension in pregnancy: diagnosis and management (2010 updated 2011) NICE guideline CG107

The experience of pregnancy- it depends on your situation, but it can be quite isolating. **Cobie Smulders**

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Organised by

Department of Obstetrics & Gynaecology
VMMC & Safdarjung Hospital, New Delhi

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Sunday, 24 Feb 2019

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Events Held Under Aegis of NARCHI

1. A CME on Breastfeeding for Nurses was conducted on 7th August at Safdarjung Hospital. Around 60 nursing staffs were educated about the importance of breastfeeding.
2. ISCCP & NARCHI DELHI organized an interactive session on Screening for Common Cancers in women, for Safdarjung Hospital Nursing students on 9th August, 2018.
3. An Awareness programme on Postpartum Hemorrhage for nursing students was conducted by Dr Shivani Aggarwal, at Kasturba hospital on 10th August, 2018.
4. A CME on "Rational use of blood and blood products" and "Basics to Advances in Breast Cancer" was conducted by FOGsd on 14th August. It was followed by Independence Day celebrations.
5. A talk on "Menstrual Health and Hygiene" was delivered by Dr Mamta Mittal on 29th August 2018, at Fortis Hospital, Shalimar Bagh.
6. An awareness camp on cervical cancer prevention was organized on 30th August 2018 for paramedics under the aegis of NARCHI. This was followed by another awareness camp on breast cancer screening and awareness of breast self examination, in association with Brahmakumaris of Kingsway Camp area.
7. Health awareness camp was organized on 31st August 2018, by Maternal and child welfare center Baprola, Near Lal convent school, Baprola vihar.
8. 2nd NARCHI Executive meeting attended by 23 Executive Members was held on 31st August 2018 at old Lecture Theater, VMMC and Safdarjung Hospital. The agenda was planning for the forthcoming NARCHI Annual conference.
9. A free infertility checkup camp was conducted by Dr Reeta Bakshi at Yamuna Vihar, East Delhi on 2nd September 2018.
10. A CME along with teacher's day eve celebration was organized on 4th September 2018 at Maples Express, Kailash Colony by Dr Anita Sabharwal (FOGsd). Interactive sessions were held on Laparoscopic Urogynaecology by Padmashri Dr Alka Kriplani and Recent trends in treatment of urinary incontinence by Dr Amita Jain. This was followed by felicitation of esteemed teachers on account of Teacher's Day Celebrations.
11. A Programme on Management of Biomedical Waste in maternity predominant hospitals of North Delhi was done by North District team on 13th September, 2018.
12. A Health Promotion Camp was conducted at village Chandpir, Muzzafarnagar, UP by Health is Wealth Foundation, India under the aegis of NARCHI by Dr Shashi Prateek on 16th Septemper, 2018. More than 100 women were benefitted by extensive health checkup.
13. A CME was conducted on 22nd September 2018 at Govindas Restaurant, ISCKON temple by Dr Anita Sabharwal, FOGsd (Forum of Obstetricians and Gynaecologists of South Delhi).
14. A Cervical & Breast Cancer Screening Camp was conducted at MCD Health Centre, Delhi Cantt. on 22 Sep 2018 by Dr Raksha Arora and Dr Sucheta Bharti. Almost 70 females underwent screening for breast and cervical cancer.
15. A talk on menstrual hygiene was conducted by Dr Jyoti Bali at CRPF School Noida on 22nd September 2018.
16. A cleanliness and sanitation drive was organized under "Swachta Hi Seva Hai" abhiyaan by Dr Mamta Mittal on 26th September 2018 at Fortis Hospital, Shalimar Bagh.
17. NARCHI Delhi reprinted at 14th World Congress and 22nd Indian Conference of NARCHI held at Nagpur, Maharashtra on 28-30 September, 2018. There was active participation by the NARCHI Delhi office bearers, Dr Pratima Mittal, Dr Achla Batra, Dr Rekha Bharti, Dr Monika Gupta, Dr Divya Pandey and Dr Sheeba Marwah made wonderful presentations. Dr Monika Gupta, Dr Divya Pandey and Dr Rita Bakshi were awarded FICMCH honor at the convocation.
18. A Public Forum on Swachta hi Seva and Menstrual hygiene along with poster and slogan competition and health programme was conducted at New Emergency Block, VMMC and Safdarjung Hospital on 2nd October 2018.
19. A Cervical Cancer Awareness and Detection Camp was organized in collaboration with Rotary club South Delhi Metropolitan, Rajiv Gandhi Cancer Hospital, Rotary club of Delhi Ridge, IWC South Delhi metropolitan, on 13th October 2018 in Kusum Pur Pahari, Delhi.
20. A Breast cancer awareness and mammography Camp was organized by Dr Kusum Chopra in Rastriya pratibha Vikash Vidayala, Vasant Kunj in collaboration with Rotary club Delhi Ridge and Inner Wheel Club, Vasant Kunj, on 22nd October 2018.
21. A COLS (Compression Only Life Support) Workshop was conducted by Dr. Rekhi (Sr. Anaesthetist) & Mr. Vivek (OT Incharge) at Sant Nirankari Govt Girls Sr. Sec. school, Nirankari Colony, Delhi on 23rd October 2018.
22. A CME on "Obstetric Dyspnoea" was organized by VMMC and Safdarjung Hospital, on 12th November at old Lecture Theater. The CME was followed by 3rd executive meeting of NARCHI, the agenda was planning for NARCHI World Congress 2020 and Annual NARCHI conference to be held in February 2019.

Events held under aegis of NARCHI

2nd NARCHI Executive Meeting

3rd NARCHI Executive Meeting

CME on Breastfeeding for Nurses

Breastfeeding Awareness Week Celebration at Safdarjung Hospital

Interactive Session on Screening for Common Cancers

Awareness Programme on Postpartum Hemorrhage

CME & Independence Day celebrations with FOGsd

Menstrual Health and Hygiene

Awareness Camp on Cervical Cancer Prevention

Health awareness camp, Baprola vihar

Infertility Checkup Camp

CME & Teacher's Day Celebration

Programme on Management of Biomedical Waste

Events held under aegis of NARCHI

Health Promotion Camp, village Chandpir, Muzzafarnagar

Cervical & Breast Cancer Screening Camp, Delhi Cantt.

Talk on Menstrual Hygiene, CRPF School, Noida

"Swachta Hi Seva Hai" Abhiyaan

NARCHI Delhi reprinted at 14th World Congress and 22nd Indian Conference of NARCHI"

Cervical Cancer Awareness and Detection Camp

Breast Cancer Awareness and Mammography Camp

Compression Only Life Support Workshop

CME on "Obstetric Dyspnea"

Public Forum on Swachta hi Seva and Menstrual hygiene

Panel Discussion on "Obstetric Dyspnea" organized at Vardhman Mahavir Medical College and Safdarjung Hospital

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Empanelled NSV Surgeon (Public)		
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Pt. MMM Hospital Malviya Nagar	Dr. Suraj Ranjan Dr. Pankaj Aggarwal	9212383753 9873448149
MV Hospital Pooth Khurd	Dr. Sourav	9718502196
LBS Hospital Khichripur	Dr. Vijay Kumar Thakur	9582500330
DDU Hospital Hari Nagar	Dr. Bhavesh	8700640541
RTRM Hospital Jaffarpur Kalan	Dr. Kamal kumar Gautam Dr. Prafful	9999998600 9717196475
JPC Hospital Shastri Park	Dr. Manish Gupta	8800556262
BM Hospital Pitampura	Dr. Krishna Nandan	9650397989
RML Hospital Gole Dak khana	Dr. Nutan Mehta Dr. Rajneesh Rana	9250022929 9311645647
LN Hospital Jawaharlal Nehru Marg	Dr. Chandan Bortamuly Dr. Abhishek Kumar	9811190963 9953833530
ESI Okhla Okhla Indust. Area	Dr. H.K. Mittal	9811309221
GTB Hospital Dilshad Garden	Dr. Sanjay Gupta	9810587148
AIIMS Hospital Ansari Nagar	Dr. Mohit	9891539794
SRHC Hospital Narela	Dr. Sonkar	9968660971
GGS Hospital Raghubir Nagar	Dr. Beena Aggarwal	9718509992
SGM Hospital Mangolpuri	Dr. Nishi Gupta	8447734460
Hindu Rao Hospital Malka Ganj	Dr. R.N. Sahai	9811484864
Sucheta Kriplani Cannaught Place	Dr. Shadan Ali	8527356558
Mty. & Gynae Hospital R. K .Puram	Dr. Kumkum	9899449191
Dr. Hedgewar Hospital Karkardooma	Dr. Akshay Bahadur	9654958608
Dada Dev Hospital Dabri Mor	Dr. Mukesh	9990257111
Empanelled NSV Surgeon (Private)		
St.Stephen's Tis Hazari	Dr. M.S.Grover	9810102808
Max Hospital Vaishali	Dr. RCM Kaza	9312058368

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21 November to 04 December

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Emergent Therapy for Acute Onset Severe Hypertension in Pregnancy

Neha Pruthi[1], Megha Mittal[2]
[1]Research Officer, [2]Senior Resident, Vardhman Mahavir Medical College and Safdarjung Hospital

Definition: Acute onset severe systolic hypertension, diastolic hypertension or both persisting for more than 15 minutes during prenatal, intranatal and postnatal period is known as hypertensive emergency. If systolic blood pressure is ≥160 mmHg or diastolic blood pressure is ≥110 mmHg, confirmation within minutes is sufficient

Severe Systolic Hypertension is defined as systolic blood pressure ≥ 160 mm of Hg; and Severe Diastolic Hypertension is defined as diasyolic blood pressure ≥ 110 mm of Hg.

Goal of treating hypertensive emergency

- To stabilize the blood pressure to range of 140 -150 / 90-100 mm of Hg, within 30 to 60 minutes.
- Maternal stabilization before delivery

Risks associated with Hypertensive emergency

- Eclampsia
- Antepartum hemorrhage (Abruption)
- Hemorrhagic stroke
- Hypertensive encephalopathy
- Acute Heart Failure/ MI
- Renal impairment
- Fetal distress
- Maternal/Fetal Mortality

Prerequisites of management of hypertensive emergency

- Accurate BP monitoring is important- Mercury Sphygmomanometer is gold standard; use appropriate cuff size; and position patient in sitting/ semi-reclined position
- Though the first line therapy does not require cardiac monitoring and ventilator settings but a center with intensive monitoring is preferred.

Management

First Line Antihypertensive Therapy

Intravenous Labetalol or

Intravenous Hydralazine or

Oral Nifedipine (when I/V access is not available)

PLUS

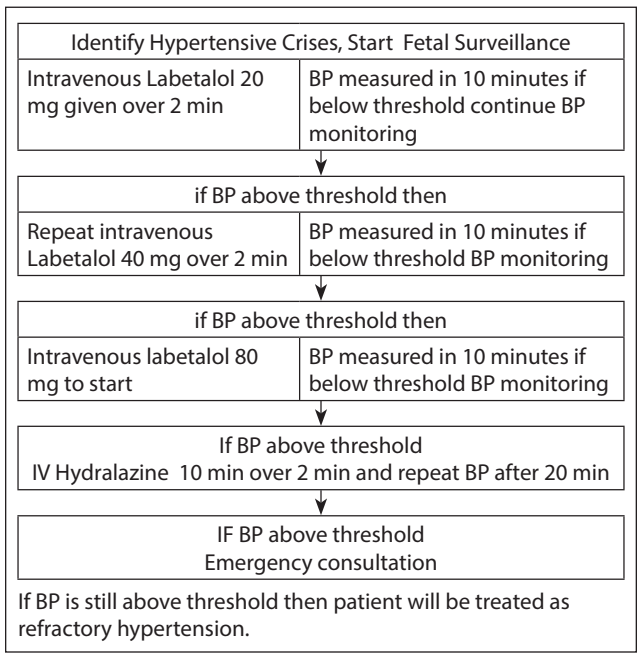
Magnesium sulphate

Drug of choice for seizure prophylaxis but not recommended as antihypertensive agent

PLUS

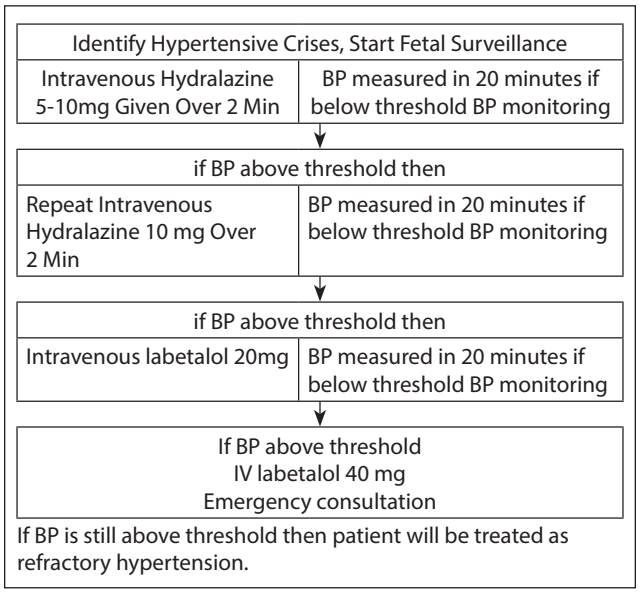
- Transfer the patient to tertiary care
- Intubation if required
- Start maternal & fetal monitoring
- Input/ output charting
- Corticosteroids if < 34 weeks (do not wait for completion of course)
- Delivery after stabilization of patient
- Induction of labor
- Caesarean Section for non vertex presentation, unfavourable cervix, failed induction

First Line Management with Intravenous Labetalol

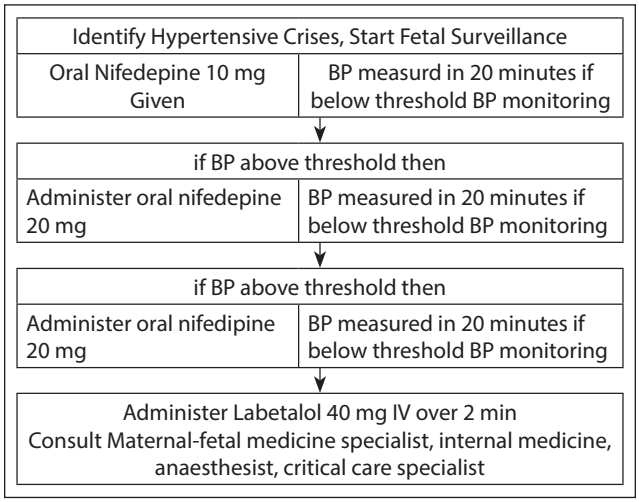


If BP is still above threshold then patient will be treated as refractory hypertension.

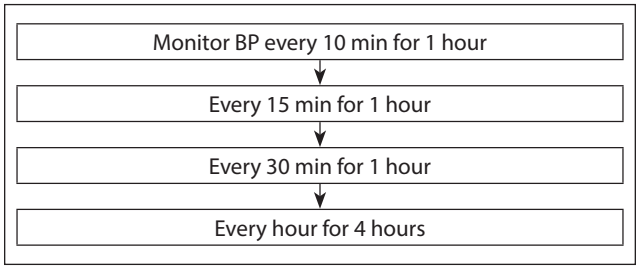
Flow Chart showing First Line Management with Intravenous Hydralazine



Flow chart showing First Line Management with Oral Nifedipine



BP Monitoring once patient has responded to treatment



Adverse effects of Hypertensive agents

Hydralazine- Maternal hypotension

Labetalol- Neonatal Bradycardia,avoid in women with Asthma, Heart disease, Congestive heart failure

Nifedipine- Tachycardia and Hypotension

Management of refractory hypertension

If First line agents fails to relieve acute hypertension then:-

- Review patient management with anesthesiologist, fetal medicine specialist
- Nitroglycerin (glyceryl trinitrate) is most commonly used drug given by intravenous infusion started at 5 µg/min and gradually increased every 3–5 min to a maximum of 100 µg/min. It is also first line antihypertensive in women with Pulmonary edema.
- Other second line drugs are nicardipine, esmolol administered by infusion pump
- Sodium nitroprusside to be reserved for extreme emergencies

Conclusion

Hypertensive emergency should be treated immediately to prevent maternal end-organ damage and fetal morbidity and mortality. First line antihypertensive therapy should be initiated immediately to improve fetal and maternal prognosis.

Suggested Reading

1. Emergent Therapy for Acute-Onset, Severe Hypertension During Pregnancy and the Postpartum Period. Committee Opinion No. 692. American College of Obstetricians and Gynecologists. Obstet Gynecol 2017:129:e90-5.
2. Dennis AT, Solnordal CB. Acute pulmonary oedema in pregnant women. Anaesthesia. 2012;67(6):646-659.
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Acute Cardiogenic Pulmonary Edema

Archana Kumari[1], Piyush Ranjan[2]
[1]Assistant Professor, Obstetrics & Gynaecology, VMMC and Safdarjung Hospital, New Delhi, [2]Associate Professor, Medicine, All India Institute of Medical Sciences, New Delhi

Introduction: Acute cardiogenic pulmonary edema (ACPE) refers to rapid onset or worsening of symptoms and/or signs of heart failure. It is a life-threatening medical condition requiring urgent evaluation and treatment. ACPE can cause severe maternal morbidity and mortality and is also the most common reason for obstetrics critical care admissions. It must be distinguished from pulmonary edema associated with injury of alveolar capillary membrane caused by various etiologies, i.e. direct pulmonary injury such as pneumonia and indirect pulmonary injury such as sepsis.

Risk Factors for ACPE

It may occur during the antenatal, intrapartum or postpartum periods. Risk factors and predisposing conditions are shown in Table 1.

Table 1: Risk factors for the development of acute pulmonary edema in pregnancy

Category	Specific risk factors
Pre-existing pre-pregnancy conditions	Cardiovascular disease (hypertension, ischaemic heart disease, congenital heart disease, valvular heart disease, arrhythmias, cardiomyopathy) Obesity, Increased maternal age, Endocrine disorders (phaeochromocytoma & hyperthyroidism)
Specific diseases in pregnancy	Pre-eclampsia, Cardiomyopathy, Sepsis, Preterm labour, Amniotic fluid embolism, Pulmonary embolism
Pharmacological agents	beta-Adrenergic tocolytic agents, Corticosteroids, Magnesium sulphate, Illicit drugs including cocaine
Iatrogenic intravenous fluid therapy	Positive fluid balance > 2000 ml
Fetal conditions	Multiple gestation

Pathophysiology

ACPE in pregnancy can be divided into two broad categories: Acute pulmonary edema without hypertension, and Acute pulmonary edema with hypertension.

Acute pulmonary edema without hypertension: Iatrogenic causes remain an important factor for acute pulmonary edema without hypertension. The beta adrenergic tocolytic agents cause pulmonary edema due to increased capillary permeability & reduced myocardial contractility along with fluid administration and the concurrent use of corticosteroids. Newer tocolytics are associated with less acute pulmonary edema. Concurrent use of magnesium sulphate and corticosteroids can precipitate acute pulmonary edema in antepartum or postpartum women.

Other causes of acute pulmonary edema without hypertension include, pre-existing cardiac disease, pregnancy associated cardiac disease (cardiomyopathy, ischaemic heart disease), amniotic fluid embolism with left ventricular systolic failure, and iatrogenic intravenous fluid administration. These women may be either normotensive or hypotensive.

Acute pulmonary edema with hypertension: Compared with healthy pregnant women, women with pre-eclampsia have a range of abnormalities like mildly elevated systemic vascular resistance, a reduction in plasma colloid osmotic pressure, altered endothelial permeability, and cardiac abnormalities. There is often preserved left ventricular ejection fraction, but the heart is unable to generate enough cardiac output to deliver oxygen to the vital organs.

The management of women with acute pulmonary edema without hypertension is similar to management in non-pregnant adults with some special considerations due to pregnancy. However, the mechanism of acute pulmonary edema in the presence of hypertension is unique to pregnancy and frequently misunderstood. In addition to stabilizing the woman and treating pulmonary edema, fetus should be delivered.

Diagnosis

Clinical Presentation: ACPE usually presents with a characteristic clinical picture of severe dyspnea with production of pink frothy sputum, and diaphoresis and cyanosis. Physical examination reveals a low-flow state, S3 gallop, jugular venous distention and fine crepitant rales in all the lung fields with auscultation.

Diagnostic testing: The diagnosis of pulmonary edema is made based on symptoms and clinical signs are found through history taking, physical examination, ECG, chest X-ray, echocardiography and laboratory tests including blood gas analysis and specific biomarkers.

1. **ECG**
   - It will often show a tachycardia and possible left ventricular hypertrophy.
   - It may reveal precipitating causes such as ST segment changes associated with an acute coronary syndrome (ACS) or an arrhythmia eg atrial fibrillation.
2. **Chest X-ray**
   - The CXR is usually helpful in excluding other causes of breathlessness, such as pneumonia or pneumothorax.
   - Typical findings include pleural effusions, cardiomegaly, interstitial and alveolar edema and upper lobe diversion.
   - A normal CXR in the acutely short of breath patient would be more likely to suggest a pulmonary embolus or COPD/asthma.
3. **Ultrasound**

   USG of lung is an important bedside tool to diagnose pulmonary edema (B lines) and also to assess fluid status of the patient (IVC collapsibility)
4. **Arterial Blood Gas**

   Hypoxemia is the commonest finding and is often out of proportion to the level of hypercapnea. i.e. Type 1 respiratory failure; this contrasts with COPD patients in extremis who have type 2 respiratory failure.
   - In pre-existing chronic lung disease or impaired conscious level hypercapnea and hypoxia may develop. i.e. Type 2 respiratory failure
   - Acid base balance and lactate will aid assessment of tissue perfusion.
5. **Venous Blood Tests**
   - Baseline bloods including CBC, KFT, LFT, and INR (Infection, renal or liver failure, anemia or electrolyte abnormalities can be identified which may precipitate or exacerbate pulmonary edema).
   - Hyponatremia and raised urea and creatinine are associated with poor outcome.
   - Troponin should be measured and may support diagnosis of acute coronary syndrome (ACS) but small rises in Troponin can occur in cardiogenic pulmonary edema (CPO) without ACS.
   - B Type Natriuretic Peptide (BNP) is a marker of impaired diastolic or systolic function. It has reasonable sensitivity and therefore can be used to rule out heart failure in patients with breathlessness but it is not very specific and levels can be high in sepsis, renal or liver failure, hypoxia, myocardial ischaemia, tachycardia as well as in many other conditions.
6. **Echocardiography**

   It should be performed to confirm suspected cardiac lesions such as valve dysfunction, cardiomyopathy, ventricular wall rupture or tamponade.

Management

The main aim is to relieve symptoms and to restore haemodynamic stability and tissue perfusion.

Airway: Allow the patient to find the best position for their airway and breathing. Usually the patient is placed in sitting position with legs dangling over the side of the bed as it facilitates respiration and reduces the venous return.

Restore Breathing: Start oxygen as women are often hypoxic. British Thoracic Society Guidelines suggest supplemental O2 only if the saturation (SPO_2) is <95% and the patient is short of breath. Oxygen should not be used routinely in non-hypoxaemic patients, as it causes vasoconstriction and a reduction in cardiac output.

*Non invasive ventilation***-** The aim of NIV is to improve oxygenation, decrease the work of breathing and increase cardiac output. Continuous Positive Airways Pressure (CPAP) provides a constant level of positive airways pressure preventing alveolar collapse. The ESC recommends that all CPO patients should be considered for NIV early on in their management (unless contraindicated). **NIV is contraindicated** when immediate endotracheal intubation is indicated; respiratory arrest or inadequate spontaneous ventilation is present; there is worsening life threatening hypoxia; and in unconscious patient unable to protect own airway.

Following initial management, medical treatment of CPE focuses on 3 main goals: (1) reduction of pulmonary venous return (preload reduction) (2) reduction of systemic vascular resistance (afterload reduction) and in some cases, (3) inotropic support. Avoidance of aortocaval compression is essential.

Circulation

Diuretics: Loop diuretics reduce preload by increasing fluid excretion and vasodilatory action. However, high doses may have detrimental effect, especially in preeclamptic women. They may cause dehydration, hyponatraemia and hypotension. The ESC Guidelines advocate *small intravenous boluses of furosemide at 20-40 mg* for patients with CPO and symptoms of fluid overload. It advocates high dose diuretic use only for those patients with clinical evidence of fluid overload and a history of chronic oral diuretic use.

After initial bolus of 20–40 mg over 2 min, repeated dose of 40–60 mg is administered after approximately 30 min, the maximum dose is 120 mg/hour.

Vasodilators: These agents have positive physiological effects by off-loading the heart through their venous and/or arteriolar vasodilatory effects causing a reduction in pre-load and/or after-load. Vasodilators should not be used in patients with a systolic blood pressure of <90 mmHg or in those with aortic stenosis (who are dependent on sufficient preload to force blood across the gradient). Majority of patients with CPO have a high-end-of-normal blood pressure at presentation and are able to tolerate nitrates. ***Nitroglycerin*** *(glyceryl trinitrate) is recommended as the drug of choice in pre-eclampsia associated with pulmonary oedema* (level 3 evidence). IV Nitroglycerine is started at a rate of 10-20 mcg/min increasing every 3-5 min by 5-10 mcg/min as needed and as BP allows, max dose 100 mcg/min. **Nitroprusside** is an alternative vasodilator which reduces preload and afterload. But, in antenatal women it should only be used when all other interventions have failed. The dose and duration of therapy should be minimized due to the risk of fetal cyanide poisoning by its metabolites thiocyanate and cyanide, as shown in animal models. The ESC 2008 Guidelines suggest cautiously commencing an infusion at 0.3 mcg/Kg/min and titrated up to 5mcg/Kg/min with invasive blood pressure monitoring. **Nesiritide** is a recombinant B-type natriuretic peptide with both a diuretic and natriuretic effect. It is both an arterial and veno dilator. It is pregnancy category C drug and should be used only if no other alternative is available.

If hypertension persists despite the combination of nitroglycerin or sodium nitroprusside and furosemide, then a calcium channel antagonist such as **nifedipine or nicardipine** may be considered (especially if diastolic dysfunction is diagnosed). **Hydralazine** (level 1++ evidence) may also be used to control BP; however, reflex tachycardia may be deleterious in this setting. **Intravenous morphine** 2–3 mg may also be given as a venodilator and anxiolytic, to be used with caution as nausea and hypopnea may occur.

High dependency care and close observation are essential: Continuous monitoring of vital signs, serial monitoring of respiration, cardiac, renal and haematological function and assessment of fetal wellbeing with multidisciplinary planning for safe birth is essential if acute pulmonary oedema occurs antenatally.

Avoidance of precipitants- Strict fluid balance and fluid restriction; Early intervention: Control of blood pressure; Prevention of further complications: Eclampsia prophylaxis with magnesium sulphate if woman has pre-eclampsia.

Inotropes: Inotropes should be considered if women is hypotensive or signs of end organ hypoperfusion persist despite use of vasodilators/diuretics. They should be commenced early once the need is recognized and stopped as soon as adequate tissue perfusion is achieved. Their use is associated with increased mortality, as they increase cardiac oxygen demand and myocardial injury. **Dobutamine** is the first choice agent, infusion is commenced at 2-3 mcg/kg/min and increased as required, *Should be avoided in moderate to severe hypotension, SBP <80 mmHg*. **Dopamine:** The vascular and myocardial receptor effects of dopamine, are dose dependent. Moderate and high dosages are arrhythmogenic and increase myocardial oxygen demand. Therefore, use these dosages only in patients with CPE who cannot tolerate dobutamine because of severe hypotension (eg, systolic blood pressure 60-80 mm Hg).

Vassopressors: **Norepinephrine**, is generally reserved for patients with profound hypotension (eg, systolic blood pressure < 60 mm Hg). After blood pressure is restored, add other medications to maintain cardiac output.

Management of peripartum Cardiomyopathy (PPCM)

Management guidelines are same as in non pregnant patients with the exception that Angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ACB) are contraindicated during pregnancy because of the risk of fetal renal agenesis.

The mainstays of medical therapy are **loop diuretics, afterload reduction with hydralazine and nitrates, digoxin and beta-adrenergic blockers**. Because

there is a high risk of venous and arterial thrombosis, anticoagulation with heparin is started when the ejection fraction is <30%.

Loop diuretics are recommended for patients with any degree of pulmonary congestion, thiazide diuretic may be added if volume cannot be adequately controlled with a loop diuretic alone. Although **hydralazine** in combination with nitrates is the preferred regimen during pregnancy, women should be switched to an angiotensin-converting enzyme inhibitor (ACEI) after delivery. Levels in breast milk of ACE inhibitors are low and not expected to cause adverse effects in breastfed infants. An ACE inhibitor that has been studied in breast milk and/or breastfed infants (such as enalapril, captopril, quinapril, or benazepril) is preferred. There are no data on ARB or ARNI safety during pregnancy & breastfeeding, therefore are not recommended. **Digoxin:** In nonpregnant patients, digoxin is not considered first-line therapy for acute decompensated HF, as it does not improve mortality. However, due to the contraindications to use of ACE inhibitors and ARBs in pregnancy, digoxin still continues to be a reasonable option for control of the ventricular rate in women with atrial fibrillation. If Ventricular rate is >110/min, Digoxin is given as IV bolus of 0.25–0.5 mg or in a dose of 0.0625–0.125 mg in patients with moderate to severe renal dysfunction. **Dobutamine** infusion is started in case of hypotension and/or cardiogenic shock. It improves cardiac output and uteroplacental perfusion. **Beta-blockers** are used as second-line agents during pregnancy. They are contraindicated in acute decompensated Heart Failure with reduced Ejection Fraction (HFrEF). In patients who are on chronic beta blocker therapy and develop acute decompensated HFrEF, dose of beta-blocker should be reduced or withheld during the initial treatment. **Calcium channel blockers** are considered safe in pregnancy. **Anticoagulants:** PPCM is associated with a high rate of thromboembolic complications. Because pregnancy is a hypercoagulable state, in PPCM prophylactic anticoagulation should be considered during pregnancy. Full-dose/therapeutic anticoagulation is started ante partum and continued for at least 6 weeks in women with atrial fibrillation and those with an ejection fraction of <30%. During pregnancy, UFH is preferred over LMWH because of the ease of monitoring the levels with activated partial thromboplastin time (aPTT). Also, protamine is not effective in reversing effect of LMWH in the setting of obstetric bleeding.

Route of delivery: Delivering the fetus decreases the metabolic demands on the mother, but afterload increases due to the loss of the low-resistance placental bed. Vaginal deliveries are preferred because complications are lower. If the woman is decompensating and not responding to medical therapy or there is obstetric indication for fetal delivery, the best plan is to induce labor with the goal of a vaginal delivery.

In PPCM normalization of left ventricular dysfunction occurs in about 50% of patients within 6 months after delivery. Normalization of cardiac function is more likely in patients with left ventricular EF >30% at the time of diagnosis. Future pregnancies should be discouraged in patients who do not recover their left ventricular function.

Key Learning Points

- Early antenatal recognition of known high-risk women, timely multidisciplinary referral and management with careful rational use of intravenous fluids is important for the prevention of ACPE.
- Treatment should consist of sitting the patient up, administering high flow O2 (Class I, level of evidence C), intravenous nitrates (Class I, level of evidence B) and instituting NIV if appropriate (Class IIa, level of evidence B)
- Low dose furosemide should also be administered; only use higher doses if the patient is fluid overloaded and/or on maintenance diuretic therapy (Class I, level of evidence B)
- In the agitated patient or those with coexistent chest pain small doses of morphine should be titrated. (Class I, level of evidence C)
- For patients with refractory HF, specialized strategies include intravenous inotropic therapy, mechanical circulatory support (eg, left ventricular assist device), and cardiac transplantation.

Suggested Reading

1. ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure European Heart Journal. 2016;37:21292200.
2. Dennis AT, Solnordal CB. Acute pulmonary oedema in pregnant women. Anaesthesia. 2012;67(6):646-659.
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Quiz Time

Q 1. Normal Pregnancy is a state of

i. Metabolic acidosis compensated by respiratory alkalosis
ii. Metabolic alkalosis compensated by respiratory acidosis
iii. Respiratory alkalosis compensated by metabolic acidosis
iv. Respiratory acidosis compensated by metabolic alkalosis

Q2. Regarding physiological dyspnea of pregnancy which of the following is true

i. Acute in onset
ii. Worsens with advancing pregnancy
iii. Associated with fine crackles on chest examination
iv. Gradual in onset

Q 3. Gold standard for diagnosing pulmonary embolism is

i. V/Q scan
ii. CT with pulmonary angiography
iii. Chest X ray
iv. ECG with echocardiography

Q 4. Which one of following is not a feature of severe preeclampsia

i. Cerebral symptoms – headache
ii. Platelet count < 1 lakh
iii. Significant proteinuria more than 300mg/24 hrs
iv. Serum creatinine >1.1

Q 5. First line management for acute severe hypertension in pregnant woman is

i. IV nitroprusside
ii. Sublingual nifedipine
iii. IV labetalol
iv. IV furosemide

Q6. Labetalol is contraindicated in

i. Liver disease
ii. Severe preeclampsia
iii. Heart failure
iv. Papilloedema

Q 7. First line anti-hypertensive in pregnant woman with acute severe hypertension and pulmonary edema is

i. IV Nitroglycerine
ii. IV Furosemide
iii. IV Hydralazine
iv. IV Labetalol

Q 8. What is delta anion gap

i. Observed AG – Upper normal AG
ii. (Urine Na^+ + urine K^+) – (Urine Cl^-)
iii. $Na^+ - (Cl^- + HCO3^-)$
iv. $24 \times \frac{pCO_2}{[HCO_3\text{-}]}$

Q 9. Oxygen saturation in a pregnant women should be maintained at

i. >98%
ii. 95-97%
iii. >95%
iv. >90%

Q10. What is the normal range for PCO2 in pregnancy

i. 27-34 mmHg
ii. 35-45 mmHg
iii. 27-45 mmHg
iv. 25-35 mmHg

Answers of the previous issue quiz: 1. (i), 2. (iii), 3. (ii), 4. (i), 5. (ii), 6. (ii), 7. (iii), 8. (ii), 9. (ii), 10. (ii)

Correct answers received from: Dr Anita Bhardwaj (22nd August), Dr Aparna Arya (23rd August), and Dr Shakuntla Kumar (25th August). Congratulations!

Arterial Blood Gas Analysis - What the Obstetrician Should Know?

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Acid-base disturbances are commonly encountered in critically ill patients in ICUs and result from a wide variety of metabolic and respiratory disorders. Besides, telling about the seriousness of the underlying diseases, acid-base disturbances have significant hemodynamic and other physiological effects. So an early diagnosis and treatment of these disturbances is an important component of the management of critically-ill patients. This requires a systematic approach to the interpretation of blood gases and simultaneously measured electrolytes.

Acid-Base Terminology

The acidity of body fluid is measured in terms of hydrogen ion concentration [H^+] and expressed as pH, which is the negative log of the [H^+]. The normal pH varies between 7.36 and 7.44 with an average of 7.4, which corresponds with the [H^+] concentration of 40 Nano equivalents.

- *Acidemia* is defined as increase in absolute [H^+] and fall in pH below 7.36, whereas
- Alkalemia is defined as decrease in [H^+] and a rise in pH above 7.44.
- *Acidosis* is a pathophysiological process that tends to acidify body fluids (lower plasma [$HCO3^-$], or increase PaCO2) and if unopposed will lead to a decrease in pH
- *Alkalosis* is a pathophysiological process that tends to alkalinize body fluids (raise plasma [$HCO3^-$], or lower PaCO2) and if unopposed will lead to increase in pH

It is important to note that there are changes in the normal values of ABG during pregnancy which can change the interpretation. The normal pH in pregnancy is between 7.40-7.46 as against 7.36-7.44; pCO2 is lower at 27-34 mm Hg (non-pregnant values 35-45mmHg) and bicarbonate is 18-21mEq/l (non- pregnant values 22-26 mEq/l).

Acid-Base Physiology

The acid-base status of the body is normally kept within the narrow range of pH despite the daily production of large amount of acid, as a result of various metabolic activities. The pH of the body fluids is determined by the amount of acid produced, the ability of the lungs and kidney to excrete the acid load and the buffering capacity of the blood.

If an extra acid or base is introduced, the body tends to mitigate the change in pH through the action of multiple buffers and activation of compensatory mechanisms. A buffer is a substance that can either absorb or donate protons to a solution. The important extra cellular buffers at physiologically relevant pH are:

Bicarbonate ($HCO_3^- + H^+ \leftrightarrow H_2Co_3 \leftrightarrow H_2O + CO_2$);

Plasma proteins (protein$^-$ + $H^+ \leftrightarrow$ H-proteins);

Hemoglobin ($Hb^- + H^+ \leftrightarrow$ H-Hb);

and phosphates ($HPO_4^{2-} + H^+ \leftrightarrow H_2Po_4^-$).

The principal buffering system for non carbonic acid in the extracellular fluid is the carbonic acid–bicarbonate pair.

Clinically when we assess a patient's acid–base status we evaluate the carbonic acid bicarbonate system, since it is easily measured. The CO_2–HCO_3, buffer system is reflected in the following formula

$$CO_2 \text{ gas} \rightarrow CO_2 \text{ (dissolved)} + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-$$

The relation of pH to this buffering system is expressed by the **Henderson–Hasselbalch equation**:

$$pH = pK + \frac{\log HCO_3}{(0.03) \times pCO_2} = \frac{\text{Kidney}}{\text{lung}} = \frac{\text{metabolic}}{\text{respiratory}}$$

Thus the pH, is determined by the ratio of the serum bicarbonate concentration to the partial pressure of CO_2 in the arterial blood. The bicarbonate concentration is regulated by the kidneys and the pCO_2 is regulated by the lungs.

The interrelation of [H^+], pCO_2 and [HCO_3^-] can also be illustrated by the **Henderson equation**

$$[H^+] = 24 \times \frac{pCO_2}{[HCO_3^-]}$$

This equation is helpful as a bedside tool to predict or evaluate the accuracy, in other terms the internal consistency of the three acid base parameters.

Simple Acid-Base Disorders

As already mentioned, the pH of a solution is determined by the ratio of HCO_3^- and pCO_2. The pathologic processes that primarily change the bicarbonate levels are referred to as metabolic disorders and pathologic processes that primarily alter the CO_2 levels in blood are referred to as respiratory disorders.

Metabolic Acidosis:	primary decrease in bicarbonate levels
Metabolic Alkalosis:	primary increase in the bicarbonate levels
Respiratory Acidosis:	primary increase in the CO_2 levels
Respiratory Alkalosis:	primary decrease in the CO_2 levels

In each of the four primary disorders, the initial process not only alters acid-base equilibrium directly, but sets in motion secondary compensatory responses that changes the other component of the pCO_2–Bicarbonate pair, so as to bring the ratio of HCO_3^- to pCO_2 back towards normal and thus helps normalize the pH.

In metabolic acidosis the primary disturbance is fall in the HCO_3^- level, the body in an attempt to return the pH back to normal induces a fall in pCO_2 via hyperventilation. Similarly, in metabolic alkalosis the primary increase in bicarbonate is compensated for by a decrease in ventilation and increase in pCO_2. In respiratory acidosis the compensatory response is a rise in HCO_3^- due to decreased renal excretion of HCO_3^-. The compensatory responses have following characteristics.

1. The compensatory process tends to return the pH back to normal but never completely, except in cases of primary respiratory alkalosis.
2. Compensatory process requires normal functioning kidneys and lungs and take time to occur.
3. The lack of compensation in an appropriate interval defines the presence of a second primary disorder.
4. The compensatory response creates a second laboratory abnormality.
5. The appropriate degree of compensation can be predicted.
   a. *Metabolic acidosis*: the expected change in pCO_2 is as follows:
      $pCO_2 = [1.5 \text{ X (Serum } HCO_3^-)] + 8 \pm 2$ or
      pCO_2 = last two digits of the pH
   b. *Metabolic alkalosis*: the expected change in pCO_2 is as follows:
      $pCO_2 = 40 + 0.6 (\Delta [HCO_3^-])$ or
      $\Delta pCO_2 = 0.6$ (Measured HCO_3^- – 24)
   c. *Acute respiratory acidosis*: the expected increase in bicarbonate is as follows:
      $$\uparrow \Delta [HCO_3^-] = \frac{\Delta pCO_2}{10}$$
   d. *Chronic respiratory acidosis*: the expected increase in bicarbonate level is as follows:
      $$\uparrow \Delta [HCO_3^-] = 3.5 \times \frac{\Delta pCO_2}{10}$$
   e. *Acute Respiratory alkalosis* : the expected decrease in the HCO_3^- level are:
      $$\downarrow \Delta [HCO_3^-] = 2 \times \frac{\Delta pCO_2}{10}$$
   f. *Chronic Respiratory alkalosis*: the expected decrease in the HCO_3^- level are:
      $$\downarrow \Delta [HCO_3^-] = 5 \times \frac{\Delta pCO_2}{10}$$

As mentioned earlier the Primary defect in metabolic acidosis is fall in HCO3 which can occur due to one of the following three mechanisms:

a. Excess acid production that overwhelms renal capacity for excretion, e.g., diabetic ketoacidosis.
b. Loss of alkali that leaves un-neutralized acid behind, e.g., diarrhea.
c. Renal excretory failure, i.e. normal total acid production in face of poor renal function, e.g., chronic renal failure of any cause.

In order to differentiate between these causes, it is important to calculate anion gap. The anion gap is shown in the following equation:

Unmeasured anion (UA) + Cl^- + HCO_3^- = Unmeasured cation (UC) + Na^+

UA – UC = Anion gap = $Na^+ - (Cl^- + HCO_3^-) = 12$

If the metabolic acidosis is caused by the addition of Cl^- as anion; then it will be designated as normal anion gap acidosis, and if the metabolic acidosis is caused by the addition of anion other than Cl^-, then it is designated as high anion gap acidosis.

Delta anion gap (ΔAG):- It is the difference between the patient's anion gap and a normal anion gap.

ΔAG = observed AG – upper normal AG

ΔHCO_3 = lower normal HCO_3^- – observed HCO_3^-

In an uncomplicated high anion gap metabolic acidosis, delta anion gap is equal to the delta bicarbonate.

Any significant deviation from this rule implies the existence of a mixed acid-base disorder.

a. When delta anion gap (ΔAG) is greater than delta HCO_3^- ($\Delta HCO3^-$) it indicates mixed high anion gap acidosis and primary metabolic alkalosis.
b. When Δ $HCO3^-$ is greater than ΔAG it indicates mixed high anion gap and normal anion gap acidosis, or a mixed high anion gap acidosis and chronic respiratory alkalosis with a compensating hyperchloremic acidosis.

Urine anion gap: In patients with a hyperchloremic metabolic acidosis, one can use urine anion gap to distinguish between renal tubular acidosis (RTA) and acidosis caused by diarrhea. Urine anion gap is calculated as follows:

(Urine Na^+ + urine K^+) – (Urine Cl^-)

A negative urine anion gap suggests diarrhea as a cause of metabolic acidosis where as a positive urine anion gap suggests the presence of RTA with a distal acidification defect.

Approach to a patient with acid-base disorder

The approach to acid-base derangements should emphasize a search for the cause, rather than immediate attempt to normalize the patient. A full consideration of the careful history such as vomiting, diarrhea, sepsis, diabetes, renal disease, alcohol or other toxin ingestion should be given. A detailed physical examination for evidence of fever, signs of volume depletion, tachypnea or bradypnea, hypo or hypertension should be carried out. Serum electrolytes such as Na^+, K^+, Cl^- and HCO_3^- should be measured in every case. A stepwise, conventional, approach is as follows:

Step I: Do the numbers make sense? Check for the internal consistency of various parameters with the help of Henderson equation: $[H^+]=24 \text{ X } pCO_2/HCO_3^-$

Step II: Determine whether the patient is acidemic (pH<7.36) or an alkalemic (pH>7.44). In mixed disorders the pH may be in the normal range, but the bicarbonate level, the pCO_2 or the anion gap will be abnormal and signal the presence of an acid-base disturbance.

Step III: Is the primary or overriding disturbance respiratory or metabolic? In the patients with acidemia an increase in pCO_2 levels indicate primary respiratory acidosis and a decrease in bicarbonate levels indicate metabolic acidosis, where as in patients with alkalemia, a decrease in the level of pCO_2 levels indicate primary respiratory alkalosis and an increase in the levels of HCO_3^- indicate primary metabolic alkalosis. A quick trick is to see the direction of change in pH and pCO2 and interpret is as:

- *Primary Metabolic Condition:* pH changes in the same direction as pCO2 or pH is abnormal but pCO2 remains unchanged
- *Primary Respiratory Condition:* pH changes in the opposite direction as pCO2 or pH is abnormal but HCO3- remains unchanged

Step IV: Is there appropriate compensations for the primary disturbance? In an effort to preserve the pH, the primary disorder sets off a compensatory reaction which is predictable as described earlier.

Step V: If a metabolic acidosis is present, is there an increased anion gap?

Step VI: If a metabolic disturbance is present, is the respiratory system compensating adequately?

Step VII: If there is an increase in anion gap, is the delta anion gap equal to delta bicarbonate. If not, there is an additional non-gap acidosis or a metabolic alkalosis.

Step VIII: Put it all together – what is the most likely diagnosis?

Case Studies

Case 1

A 22 year postpartum woman presents with fever and foul lochia

pH=7.32	pCO_2=22.5	pO_2=92
Na=137	K=4.1	Cl=100
HCO_3=11	Lactate- 4	

1. pH shows acidemia
2. Associated with CO2 and pH both moving in same direction (decrease) and hence metabolic.
3. Compensation: predicted pCO_2 should be = 1.5 X $[HCO_3]$ + 8 $\pm$ 2 = 1.5 X [11] + 8 $\pm$ 2 = (16.5 + 8) $\pm$ 2 = 24.5 $\pm$ 2. Given pCO_2 is 22.5; thus appropriate respiratory compensation is present.
4. Anion gap (AG) = {Na – (HCO_3 + Cl)} = 137-111 = 26; thus wide AG metabolic academia (due to addition of lactic acid).
5. Delta anion (14) = delta bicarbonate (13); hence simple disorder

Diagnosis – Post partum sepsis with lactic acidosis

Case 2

A 35 years old Para 1 preeclamptic patient presents with discomfort in the left leg.

Her BP is 170/95 HR=100 RR=28 Temp=99.5° F

pH=7.53	pCO_2=16	pO_2=97
Na=136	K=4.0	Cl=100
HCO_3=14		

pH and pCO_2 show evidence of respiratory alkalemia.

Predicted delta $HCO_3 = 2 \times \frac{40-16}{10} = 4.8$; or predicted $[HCO_3]$ should be 19.2.

However given $[HCO_3]$ is 14. Therefore metabolic acidosis is also present. AG = 136 – 114 = 22; hence respiratory alkalemia with wide AG metabolic acidosis.

Diagnosis: Deep vein thrombosis with pulmonary embolism

Case 3

A 34 year-old diabetic patient presents with nausea, vomiting and abdominal pain

pH=7.46	pO_2=88	pCO_2=33
HCO_3=22	Na=133	K=3.5
Cl=86	Finger-prick glucose=430	

pH and pCO_2 suggest respiratory alkalosis. Predicted delta $HCO_3 = 2 \times \frac{7}{10} = 1.4$;

Hence appropriate. AG = 133 – (86 + 22) = 25; delta AG = 13; delta $[HCO_3]$ is 1.4. As delta AG > delta $\{HCO_3\}$ there is primary respiratory alkalosis with wide AG metabolic acidosis and metabolic alkalosis

Diagnosis: Diabetic ketoacidosis (metabolic acidosis) with vomiting (metabolic alkalosis)

Case 4

A 34 year old lady is admitted to the hospital with persistent community acquired pneumonia. That has poorly responded to a weak-long antimicrobials therapy. She mild cyanosis and tachypnoeic. Her lab data are as follows.

pH=7.44	pCO_2=25	pO_2=48
HCO_3=17		

History, pH and pCO_2 suggest chronic respiratory alkalosis. Compensation: predicted

Delta $HCO_3 = \frac{(40 - 25)}{10} \times 5 = 7.5$; hence appropriate metabolic compensation..

Diagnosis: Chronic respiratory alkalosis with appropriate metabolic compensation.

Suggested Reading

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Half a million women die each year around the world in pregnancy. It's not biology that kills them so much as neglect- **Nicholas Kristof**

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SFM Academic Calendar for 2018

1. **10th-12th August, 2018:** Fetal CardioCon 2018, Annual Cardiology Congress of the Society of Fetal Medicine, New Delhi. Star Speakers: Simon Meagher and Paul Brooks from Australia. For details please visit our conference website www.fetalcardiocon.org. For any queries please contact: Vishal Mittal at +91 9312227181 (Email sfmsecretariat2017@gmail.com).
2. **19th August, 2018:** SFM Vishakapatnam Meeting. For details contact Dr. KV Sridevi at +91 9440515253 (Email: kvsridevi2000@yahoo.co.in).
3. **29th August, 2018:** SFM Delhi Chapter 1st Quarterly Meet (2018-2019) at LT-4, UCMS & GTB Hospital, Delhi. Members registration free. For details contact Dr. Krishna Gopal at 9818624184 (Email: dablookg@yahoo.co.in)
4. **1st-2st September, 2018:** SFM Nagpur Meet. For details contact Dr. Vivek Kashyap at +91 9811116050 (Email: drvkashyap@yahoo.com).
5. **22nd-23nd September, 2018:** SFM Corbett Meet. For details contact Dr. Bharti Gahtori at +91 9837259947 (Email bharti.gahtori@gmail.com).
6. **20th-24th October 2018:** ISUOG World Congress 2018, 28th World Congress on Ultrasound in Obstetrics and Gynecology, Singapore. For details please visit our website or click on https://www.isuog.org/events/world-congress.html. SFM Associates are eligible for discounted fees of US$500 only. Please use code "LOCPAR1808" while registering under "Special Registration Code" option.
7. **28th October, 2018:** Annual Meeting of the Kerala Chapter of the Society of Fetal Medicine to be held at Trivandrum. For details contact Dr. Meenu Batra at +91 9947013900 (Email: drmeenubatra@yahoo.com).

For any query please contact the secretariat at +91 9312227181 or email at sfmsecretariat2017@gmail.com.

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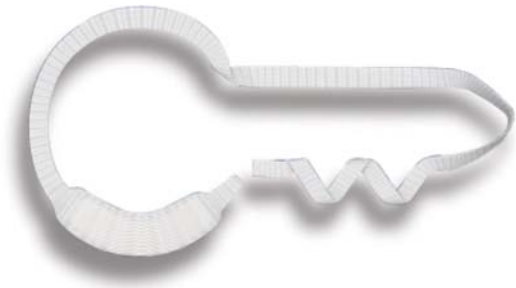

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- 1st Test at the time of 1st ANC visit to screen of glucose intolerance
- 2nd Test between 24 and 28 weeks of gestation
- 3rd Test around 32 - 34 weeks of gestation

Diagnosis (In pregnancy)	2-hour plasma glucose[3]
Normal	< 120 mg/dl
Gestational glucose intolerance	120 - 139 mg/dl
Gestational Diabetes Mellitus (GDM)	140 - 199 mg/dl
Diabetes	≥ 200 mg/dl

GT-75 ... *save Generation next.*

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