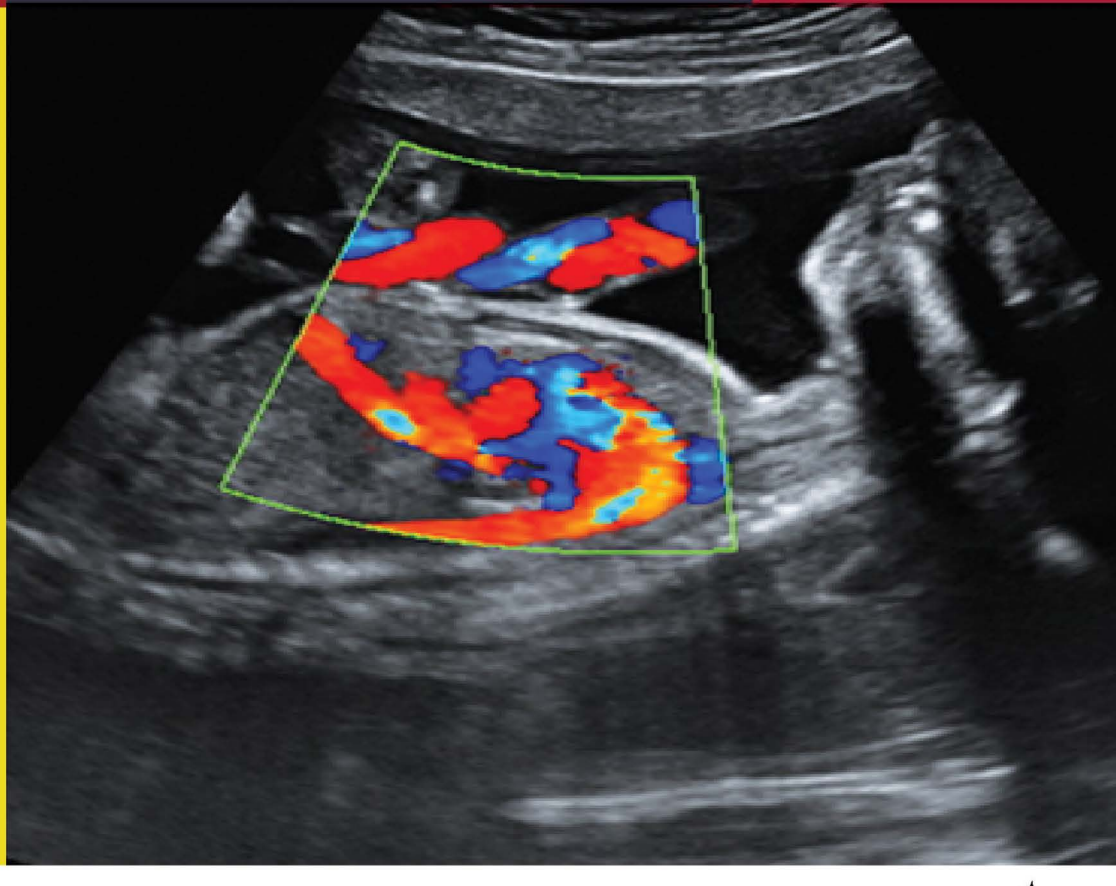
ks, visit our website www.jaypeebrothers.com, for detailed information on obstetrics & gynecology books, visit our website www.jaypeebrothers.co

Donald School Textbook of Diabetic Pregnancy & Ultrasound



Badreldeen Ahmed
Asim Kurjak

Foreword
Frank A Chervenak

JAYPEE

Contents

1. Diabetes in Pregnancy: Diagnosis and Treatment **1**

Cihat Sen, Murat Yayla, Olus Api

- Prevalence *1*
- Pathophysiology and Risk Factors *2*
- Frequent Obstetric and Perinatal Problems in Gestational Diabetes *3*
- Postnatal Long-Term Risks *6*
- Type 1/Type 2 Diabetes during Pregnancy *14*

2. General Aspects of Diabetes in Pregnancy **23**

Sertac Esin, Cihat Sen

- Classification and Physiology *23*
- Epidemiology *24*
- Significance *25*
- Risk Factors *26*
- Screening and Diagnosis *26*
- Management *28*

3. Overview of the Role of Ultrasound in the Management of Diabetes in Pregnancy **32**

Badreldeen Ahmed, Justin Konje

- Role of Ultrasound in Estimation of Fetal Weight *32*
- Role of 3D Ultrasound in EFW in Diabetic Pregnancy *33*
- Role of Ultrasound in Diagnosis of Congenital Malformation *34*
- Role of Ultrasound in Monitoring Diabetic Pregnant Patients *39*

4. Fetal Behavior in Normal Pregnancy and Diabetic Pregnancy **46**

Asim Kurjak, Sonal Panchal

- Understanding the Normal Fetal Activity *46*

5. Overview of the Role of Ultrasound in the Management of Diabetic Pregnancy **62**

Sonal Panchal

- Incidence *63*
- Causative Mechanism *63*
- Biochemical Parameters *64*
- Malformations *64*

6. Obstetric Ultrasound for Diabetes-related Congenital Anomalies **92**
Karim D Kalache, Farhat Gothey, Alexander Weichert, Daniel Kamil

- Pregestational and Gestational Diabetes and the Risk of Major Congenital Anomalies *92*
- Incidence and Impact of Congenital Anomalies *92*
- Caudal Regression Syndrome *93*
- Structural Cardiac Defects and Cardiac Anomalies *95*
- Central Nervous System Anomalies and Neural Tube Defects *98*
- Gastrointestinal Anomalies *100*
- Femoral Hypoplasia-Unusual Facies Syndrome *101*
- Ideal Window to Perform the "Screening" Ultrasound Examination *103*

7. Offspring of Diabetic Mother **105**
Milan Stanojevic

- Fetal Effects of Maternal Hyperglycemia *107*
- Neonatal Effects of Maternal Hyperglycemia *107*
- Perinatal Outcome of the Offspring from Diabetic Pregnancies *108*
- Inhospital Care for the Newborns from Diabetic Pregnancies *108*
- Respiratory Pathology in Neonates from Diabetic Pregnancies *109*
- Hypertrophic Cardiomyopathy and Offspring of Diabetic Mother *110*
- Offspring of Diabetic Mother: Prenatal and Postnatal Growth *111*
- Congenital Malformations in the Offspring of Diabetic Mother *113*
- Neurodevelopmental and Psychiatric Outcome of the Newborns from Diabetic Pregnancies *114*

8. Artificial Maturation of Fetus in all Kinds of Diabetes **121**
Miroslava Gojnic, Milan Perovic, Igor V Pantic, Badreldeen Ahmed

- Physiology Processes in Fetal Lung Maturation *121*
- Pathophysiological Processes that Influence Fetal Lung Maturation in Pregnancies Complicated by Diabetes *122*
- Synthetic Corticosteroids for Antenatal Therapy and Prophylaxis *123*
- Implications of Antenatal Corticosteroids for Fetal Lung Maturation in Women with Diabetes *124*
- Recommendations for Management Policy for Women with Preexisting Diabetes Requiring Antenatal Corticosteroid Therapy *127*
- Recommendations for Management Policy for Women with Gestational Diabetes Requiring Antenatal Corticosteroid Therapy *128*
- Recommendations for Management Policy for Women with Diabetes Requiring Antenatal Corticosteroid Therapy in the Setting with Predominately Subcutaneous Insulin Treatment *128*

- Contemporary Testing of Fetal Lung Maturity in Predicting the Risk of Respiratory Distress Syndrome in Pregnancies Complicated with Diabetes by Magnetic Resonance Imaging *129*
- Contemporary Role of Ultrasonography in Pregnancies Complicated with Diabetes with Emphasis on those that Require Antenatal Steroid Therapy *129*

9. Diabetes and Obesity in Pregnancy **134**

Hisham Arab

- Burden of a Disease: Influencing Maternal Mortality Rate *134*
- Obesity and Gestational Diabetes Mellitus: Are they Independent? *135*
- Obesity and Gestational Diabetes Mellitus: Future Risk *136*
- Obesity and Gestational Diabetes Mellitus: Adverse Pregnancy Outcome *136*
- Nutritional Deprivation in Pregnancy *140*
- Metabolism during Pregnancy *141*
- Intervention during Pregnancy *142*
- Intervention before pregnancy *144*

Index *151*

Chapter 4

Fetal Behavior in Normal Pregnancy and Diabetic Pregnancy

Asim Kurjak, Sonal Panchal

INTRODUCTION

Ultrasound has been a modality of choice for assessment of the development of the embryo and fetus in the womb. Three-dimensional (3D) ultrasound has made the study of the fetal anatomy even more accurate and understandable. But the fetal development is not only the development of the structures, it is also their functionality. The functionality of the kidneys, for example can be confirmed by urine production and filling of the bladder, the functionality of the bladder by its periodically emptying and filling and that of the heart by fetal circulation. The function of the nervous system is the most complex and this can be studied by fetal movements and fetal expressions. The development of fetal motor behaviors can be studied by real-time ultrasound.[1] The fetal activity observed or recorded with ultrasound equipment is fetal behavior.[2]

Study of the fetal movements has been found to be more correlating with the fetal central nervous system (CNS) development. Studies have shown that the development and maturation of the fetal nervous system is reflected by quality and quantity of fetal movements.[3,4] On comparing the fetal movements with morphological studies, it was found that the fetal behavioral patterns directly reflected the development and maturation of fetal CNS. Therefore, assessment of fetal behavior in different periods of gestation can help to distinguish normal brain from abnormal during different phases of development.[5] It also helps to make the diagnosis of functional and structural abnormalities earlier.[6]

B-mode or a two-dimensional (2D) ultrasound is impossible to understand the complexity of these movements and so till the invent of four-dimensional (4D) ultrasound it was not possible to correctly evaluate the fetal movements, especially fetal expressions. The details of fetal face and hands studied by 4D ultrasound have potential to generate information regarding fetal movement and behavior.[7] There is a specific fetal behavioral pattern that corresponds to each week or trimester of fetal life and this pattern reflects the steps of human brain development and maturation.[8-10]

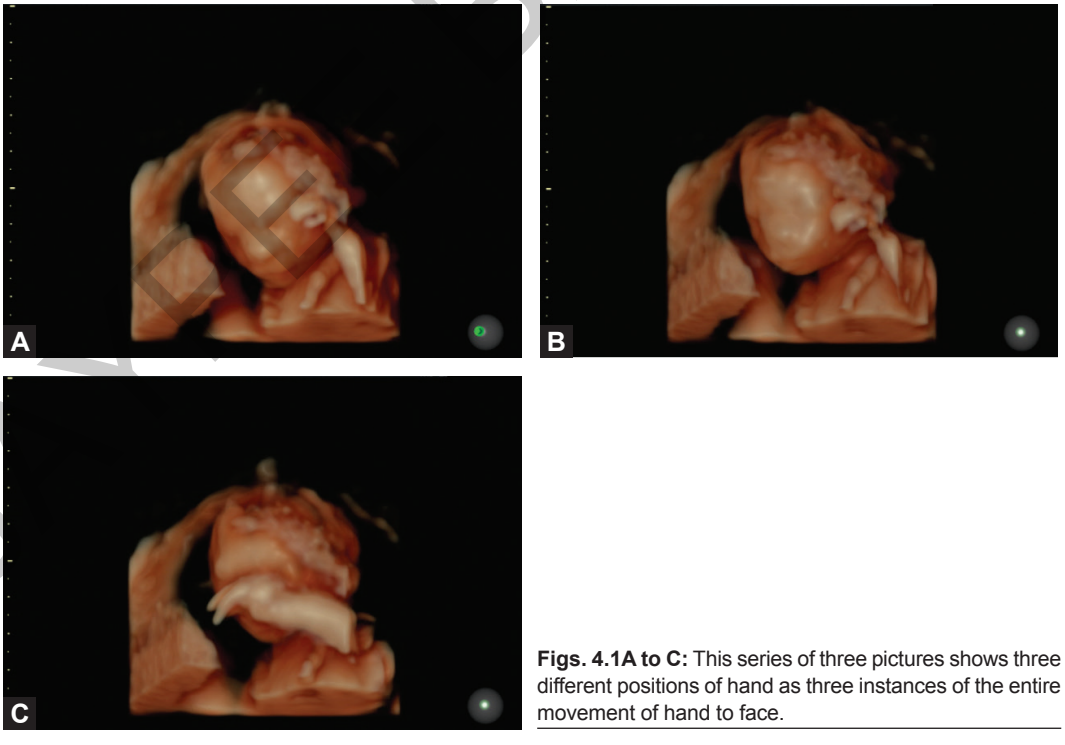
UNDERSTANDING THE NORMAL FETAL ACTIVITY

Spontaneous fetal movements can be observed using 2D ultrasound around 8th of gestational week, but 4D ultrasound may show fetal motility at 7th week of gestation on transvaginal scans.[11,12] From 10 weeks onwards the frequency of fetal movements increase and are maximum between 14 weeks and 19 weeks.[12] The longest interval between movements at this age is 5–6 minutes. Between 9 weeks and 14 weeks of fetal age mostly general movements are observed.[13]

JAYPEE BROTHERS

Using 4D sonography, Kurjak et al. found that from 13 gestational weeks onwards, a "goal orientation" of hand movements (Figs. 4.1A to C) appears and a target point can be recognized for each hand movement. At 15 weeks, 15 different types of movements have been recorded.[14,15] But according to a study by Kurjak et al., 16 different types of movements can be observed at 15 weeks of gestation.[16] Eye movements are detectable at 16–18 weeks, organized complex movements at 20 weeks and facial expressions also start at 20 weeks.[17] Facial expressions like smiling, yawning, eyelid movement, mouthing, grimacing, tongue expulsion/swallowing, and sucking were observed during 2nd and 3rd trimesters. Mouthing was the most frequent facial movement during early 3rd trimester, whereas scowling and sucking were the least frequent.[15-18] In the second half of pregnancy, the frequency of general movements gradually decreases, particularly during the last 10 weeks of pregnancy.[19] In the last trimester of gestation, the range of hand and face movements is the widest.[19] Fetal neuromuscular development is due to alternate periods of increased and decreased movements, though the exact functional significance of the same is still not understood.[20,21]

The movement in an unstimulated fetus is the result of spontaneous behavior without sensory stimulation and is the best method to assess its CNS capacity,[5] and can be used as a marker for fetal brain status.[22] Any fetal brain insult will interfere with endogenous motor activity of the fetus. Genetic factors, external stimuli, pathological conditions or even environmental changes, can affect the fetal human brain up to a degree that may be difficult to assess, especially prenatally. Most times the neurological damage and its effects are difficult to predict.[23]



Figs. 4.1A to C: This series of three pictures shows three different positions of hand as three instances of the entire movement of hand to face.

JAYPEE BROTHERS

Table 4.1: Types of hand and facial movements in fetus.[28]

Hand and head movements	*Facial expression*
• Head retroflexion • Head rotation • Head anteflexion • Hand to head direction • Hand to eye • Hand to mouth • Hand to face • Hand to ear	• Isolated eye blinking • Mouthing • Yawning • Tongue expulsion • Grimacing • Swallowing

Establishment of neural connections as a developmental process leads to development of new movement patterns of fetus or transformation of existing patterns.[24]

Integrity of the fetal CNS can be assessed by behavioral states of the fetus viz. movement of individual organs like head, trunk or limbs in an unstimulated but awake fetus and also by qualitative assessment of general movements.[25] When fetal behavior is studied by 4D ultrasound, the altered movement pattern can diagnose abnormal brain development and assist early diagnosis of various abnormalities.[26-28]

The centers in the cerebellum impose controls on the unrestricted movements induced by the lower centers of the brain.[19] The movements also become more complex as pregnancy advances that represents the cerebral maturation. Inhibition is a marker of neurological development (especially cortical centers) and most longitudinal studies have also proved that the fetus becomes less active as gestation advances.[29]

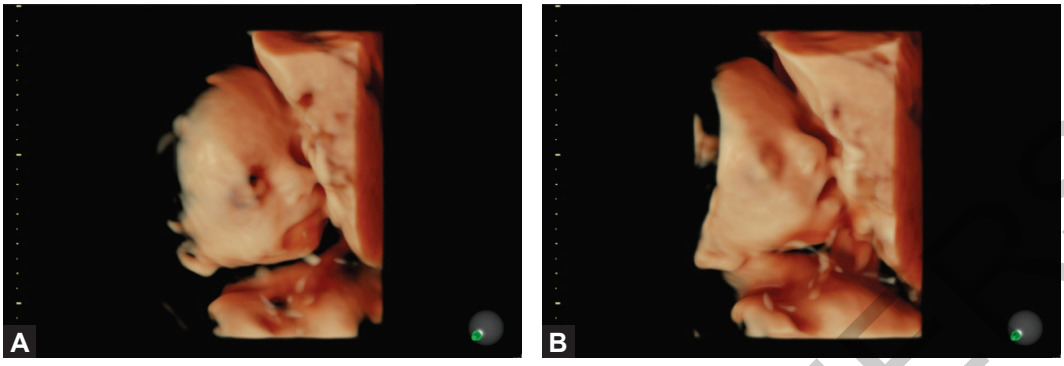
Movements observed and analyzed in 2nd and 3rd trimesters are listed in Table 4.1.

The spinothalamic tract is established at the 20th week and myelinized by 29 weeks of gestation and the thalamocortical connections penetrate the cortical plate at 24–26 weeks.[16] Evoked potentials can be detected from the cortex at the 29th week, indicating that the functional connection between periphery and cortex operates from that time onwards.[30,31] This is because of maturation process in the brainstem.[32] Facial expressions are representation of the maturation of the brain. Expressions like grimacing, tongue expulsion and eyelid movements (Figs. 4.2A and B) similar to emotional expressions in adults and can be seen by 4D US.[17]

General movements (GMs) or Gestalt perception[33] involve the whole body in a variable sequence of arm, leg, neck, and trunk movements. The movements increase and decrease in intensity, but are fluent and elegant, complex, and variable. These are called fetal or preterm from 28 to 36 to 38 weeks of postmenstrual age. GMs are considered to be a better predictor of postnatal neurological disability than clinical neurological examination alone.[33]

Normal GMs are complex but fluent and involve neck, trunk, and limb movements. Moreover these should also increase and decrease in their intensity.[34] GMs are complex and fluent and also are not repetitive.[35] If that is not the case, they are considered abnormal.[15,35] Slow, monotonous repetitive movements mimicking cramps are abnormal movements. Even when the movements are occurring with generalized simultaneous muscle contractions and relaxations, it is considered abnormal pattern.[5,22] These also may show variability in strength and the amplitude of movements.[22] These are often seen in the fetuses of mothers with diabetes

JAYPEE BROTHERS



Figs. 4.2A and B: (A and B) Eyelid and lip movements are seen. (A) The eye is open and mouth is closed and (B) Eye is closed and the mouth is open in the same fetus.

mellitus, and also other pregnancies at risk like intrauterine growth retardation (IUGR), pregnancy-induced hypertension (PIH), and prematurity.[15] Both term and preterm newborns, who had cerebral insult of any type during their prenatal period may also show abnormal general movement.[11] The identification of fetal CNS depression is based on precompetences (opening of eyes, variety of facial expressions) also along with the primary reflexes (rhythmical bursts in the sucking pattern) and quality of GMs.[5,36]

Evaluation of GMs must be done on video recordings, may it be prenatal or postnatal. These must be evaluated with "Gestalt perception", which could be described as overall impression of GMs with standardized procedure. It is important to recognize, document and classify the movement pattern and then their complexity, variability, and fluency.[23] These can be classified as normal-optimal, normal-suboptimal, mildly abnormal, and definitely abnormal.[5,37] Quality of neurological movement is more important than the quantity. These can better predict neurodevelopmental outcome than classical neurologic examination alone.[38]

Why do Fetuses of Diabetic Mothers have Higher Risk of Neurological Derangements?

Gestational diabetes mellitus, with it carries multiple risk factors for the developing fetus, and each have independent effects on neurodevelopment that may impact mnemonic behavior.[39] These risk factors are: (1) chronic hypoxia,[40] (2) hyperglycemia/reactive hypoglycemia, and (3) iron deficiency.[41] On the basis of animal models, these factors selectively affect regions of the fetal brain that are involved in explicit memory (e.g. the hippocampus).[39,42-44]

Women with diabetic vascular disease have the highest malformation rates, leading to the belief that vascular disease and hypoxia are teratogenic.[44]

Maternal ketonuria in diabetic mothers is associated with reduced intelligence quotient (IQ) in their infants,[44-46] as ketones may interfere with normal development of fetal brain.[44] Abnormal protein synthesis might also result from a lack of glucogenic amino acids or an overabundance of branched-chain amino acids as is seen in poorly controlled diabetes.[44,47] Jovanovic and co-workers (1980) were able to study the pregnancy in diabetic women before

JAYPEE BROTHERS

the 10th week of gestation. Women with uncontrolled diabetes had estradiol, prolactin, and human chorionic gonadotropin levels below the range associated with normal pregnancy.[44,48]

When these risk factors co-occur, oxygen consumption of the fetus increases and causes fetal hypoxia[39,49] that can ultimately result in brain iron deficiency through shifting of available fetal iron away from the brain and into the expanding red cell mass.[39,41] Prenatal hypoxia and hyperglycemia/reactive hypoglycemia cause poor behavioral and neurologic outcomes presenting as motor and cognitive deficits in humans[41] and damage to memory areas such as the cerebral cortex, striatum, and hippocampus in animal models.[39,50] These factors together lead to altered normal maturation of the behavioral states in fetuses with IUGR, maternal diabetes mellitus or alcohol consumption.[51]

What to Observe for?

Assessment of Neurological Function

Even when these movements were documented by 4D ultrasound it was a very difficult task to classify these movements and based on these to judge the neurological developmental status of fetus. For the first time a test was structured to assess the fetal CNS integrity and was named as Kurjak's antenatal neurodevelopmental test (KANET).[52] KANET is the first test that is based on 4D ultrasound, with an original scoring system and has been standardized, so it can be implemented in everyday practice, overcoming the practical difficulties and covering the gaps of methods that were used in the past for the evaluation of fetal behavior (Table 4.2).[53-54] Improvement of 4D USG technology enabled introduction of KANET, which is a powerful tool in the assessment of fetal behavior and a guide to predict cerebral palsy.[55] After experimental use for 10 years, a consensus was passed in Bucharest concluding that KANET test can be used in everyday clinical practice for follow-up of fetuses at neurological risk with strong recommendations for strict and reliable multidisciplinary postnatal follow-up for at least 3 years. It has acceptable sensitivity, specificity, positive, and negative predictive value for neurodevelopmental anomalies.[56]

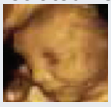
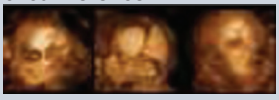
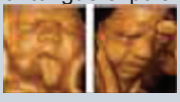
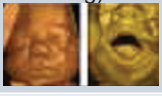
The test is of more significance than the morphological studies of the brain. Analysis of the fetal behavior compared with morphological studies has concluded that fetal behavioral patterns are direct reflection of developmental and maturational processes of fetal CNS.[57]

The parameters that have been incorporated in the KANET test are isolated head anteflexion, overlapping cranial sutures, head circumference, isolated eye blinking, facial alterations, mouth opening (yawning or mouthing), isolated hand and leg movements and thumb position, and Gestalt perception of GMs.[44,58]

Based on multicentric studies over several years, and on the theory of central pattern generators for GMs, certain fetal movements were chosen as parameters to assess neurological development.[58,59] It is to be performed between 28 weeks and 38 weeks, when fetus is awake. Fetus is observed for 15–20 minutes. If fetus is asleep, the scan is repeated after 30 minutes or after 14–16 hours. Each movement depending on its frequency is given a score of 0, 1, and 2 for nine parameters. A score of 0–5 is abnormal, a score of 6–13 is borderline and a score of more than 14 is normal.[60] If the test is abnormal or borderline, it is to be repeated every 2 weeks, till delivery.[59]

JAYPEE BROTHERS

Table 4.2: Score chart of fetal movements according to KANETs.[62]

	Score			*Sign score*
Sign	*0*	*1*	*2*	
Isolated head anteflexion 	Abrupt	Small range (0–3 times of movements)	Variable in full range, many alternation (>3 times of movements)	
Cranial sutures and head circumference 	Overlapping of cranial sutures	Normal cranial sutures with measurement of HC below or above the normal limit (–2 SD) according to GA	Normal cranial sutures with normal measurement of HC according to GA	
Isolated eye blinking 	Not present	Not fluent (1–5 times of blinking)	Fluency (>5 times of blinking)	
Facial alteration (grimace or tongue expulsion)  Or Mouth opening (yawning or mouthing) 	Not present	Not fluent (1–5 times of alteration)	Fluency (>5 times of alteration)	
Isolated leg movement 	Cramped	Poor repertoire or Small in range (0–5 times of movement)	Variable in full range, many alternation (>5 times of movements)	
Isolated hand movement  Or Hand to face movements 	Cramped or abrupt	Poor repertoire or Small in range (0–5 times of movement)	Variable in full range, many alternation (>5 times of movements)	
Fingers movements 	Unilateral or bilateral clenched fist, (neurological thumb)	Cramped invariable finger movements	Smooth and complex, variable finger movements	
Gestalt perception of GMs	Definitely abnormal	Borderline	Normal	
			Total score	

(GA: Gestational age; GMs: General movements; HC: Head circumference; KANETs: Kurjak's antenatal neurodevelopmental tests).

JAYPEE BROTHERS

Neurological Function in Fetus of a Diabetic Female

The results of a study by Edelberg et al. (1987) and Robertson et al. have shown that it is actually the changing maternal blood glucose level that affects the cyclicity and frequency of fetal motor activity, rather than persistently high blood glucose levels.[21,61,70]

Schulte et al. and Visser et al.[62,80] studied the neurological development of infants of diabetic mothers, and found longer rapid eye movement (REM) sleep in newborn infants of diabetic mothers.

There is evidence that it is the concurrent maternal blood glucose levels according to which the fetal movements may be affected in diabetic and nondiabetic pregnancies, but different studies have mixed results. Some studies have reported increased and some have shown decreased fetal movement with elevated blood glucose levels.[21,63-68]

Some have reported decreased fetal movement,[69-71]and others have reported no effects.[72-76] An increased rate of minor neurological dysfunction was found in a group of 32 children born to mothers with gestational diabetes, including some fine and gross motor deficits, compared with a group of control children.[77,78]

Abnormalities in the fetal motor activity may consist of a delayed first emergence of specific movements, quantitative changes and an abnormal quality of movements (i.e. changes in execution of movement patterns) and abnormal development of fetal behavioral states.[50,56,57] Qualitative and quantitative assessment of fetal movements can be used for the recognition of cerebral dysfunctions and probably neuromuscular ailments. Alteration in the normal movement pattern in terms of frequency and strength is seen in IUGR, the pathophysiology of which is fetal hypoxia. In the fetuses of diabetic mothers, fetal hypoxia is the main pathophysiological factor and so similar changes in fetal movements can be observed. A study on diabetes-related influence on fetal motor activity revealed 1–2 week delayed appearance of almost all fetal behavioral patterns in first 12 weeks of pregnancy except the fetal breathing movements.[15,50]

Fetal breathing pattern is considered to be one of the important parameters of fetal well-being in late diabetic pregnancies. It is not affected by Braxton–Hicks contractions. This means that the fetal neural control of fetal breathing like movements differ in diabetic pregnancy, than in normal.[15,79]

Cyclic Motility

Other aspects of fetal neurobehavioral organization are influenced by the altered metabolic environment.[70,80-90] In spite of good clinical control of diabetes, the infants of these mothers have a risk of compromised neurological developmental outcome.[21,91-99]

Spontaneous fetal movement in the last trimester of human gestation is dominated by irregular oscillations on a scale of minutes [cyclic motility (CM)].[21] The movement pattern (increased and decreased movements in cyclicity in normal females) is steady but is altered in mothers with increased blood glucose levels.[21] Early in the 3rd trimester, changes in the rate of oscillation in fetal CM between the two periods of activity were inversely related to changes in maternal blood glucose levels.[21] It is seen that relatively short-term fluctuations in maternal glucose metabolism, rather than chronically elevated blood glucose, per se, is

JAYPEE BROTHERS

the effective perturbation of the intrinsic cyclic patterns in spontaneous fetal motor activity in diabetic pregnancies. The results revealed that fetal CM is more sensitive to fluctuations in maternal blood glucose levels during the early part of the 3rd trimester of gestation than during the middle or end of the 3rd trimester. The results suggest that disruption of the temporal organization of spontaneous fetal motor activity in diabetic pregnancies represents an acute response to fluctuations in the metabolic environment rather than alteration of CM development.[100,101]

But according to other studies, the transient abnormality maternal glucose metabolism may affect fetal CM but does not cause any increased risk of poor general developmental outcome in children of diabetic mothers.[21,100] The effects are similar in the fetuses of mothers with type I or gestational diabetes, and no difference when fetuses later classified as appropriate for large gestational age were considered separately ($p > 0.05$).[21,100]

Effect during Infancy and Childhood

When gross motor functions were studied in children of diabetic mothers by the Bruininks–Oseretsky test of motor proficiency, it was observed that these children were weak performers as compared to the controls.[77] Maternal diabetes adversely affects some fine neurological functions in children at school age, but not their cognitive scores.[77] These effects are not correlated with the degree of glycemic control.[77] Developmental delay, learning difficulties at school, and a high rate of attention deficit hyperactivity disorders (ADHDs) are more often seen in the children born after high-risk pregnancies.[77,102] These children in their early school age have more soft neurological signs (signs of mild, nonspecific brain damage), and lower gross and fine motor achievements than pair matched control children born to nondiabetic mothers.[77,103,104] Variability in muscle tone (hypertonicity or hypotonicity) may cause delayed or abnormal motor development (Miyahara M, Department of Kinesiology, UCLA; unpublished observations).

Children born to the mothers having diabetes mellitus (IDM) had a risk of shorter gestational age [mean 38 weeks, standard deviation (*SD*) 2], greater standardized birth weight scores (mean 3,797 grams, *SD* 947), and lower iron stores (mean ferritin concentration 87 µg/L, *SD* 68) in comparison with the control group.[39]

Children born to the mothers whose diabetes was diagnosed late in pregnancy, had lower cognitive scores and verbal performance compared with controls.[77,105,106]

Can KANET Predict these Abnormalities?

Kurjak's antenatal neurodevelopmental test can be useful for early diagnosis of neurological disorders that become manifest in perinatal and postnatal period.[46] The authors observed that a low KANET score is predictive of both intrauterine or neonatal death.[53] The study demonstrated the evaluated and accepted KANET to detect and discriminate normal and abnormal fetal behavior in normal and in high-risk pregnancies.[107] Except for higher incidence in the abnormal group, there was no marked difference in the different motor patterns studied.[108,109]

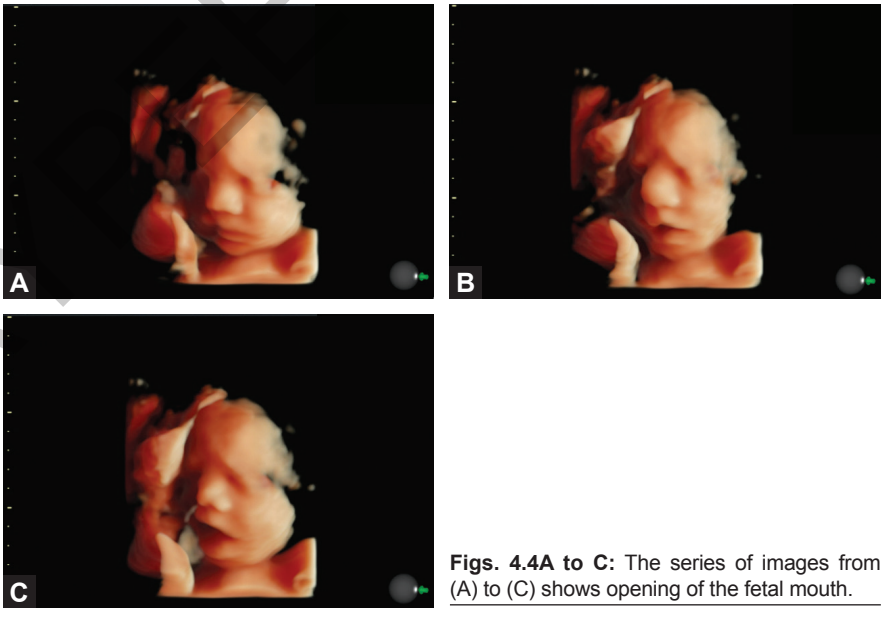
Analysis of sick preterm infants revealed a "reduction of elegance" and fluency, variability, fluctuation in intensity, and speed instead of change in incidence of distinct motor patterns.[30,110-112] Abo-Yaqoub et al. showed in their study that the difference between

JAYPEE BROTHERS

the two groups were isolated head anteflexion (Figs. 4.3A to C), isolated eye blinking, facial expressions, mouth movements (Figs. 4.4A to C), finger movements (Figs. 4.5A to C), isolated hand movements, hand-to-face movements (Figs. 4.6A to C), and GMs (Figs. 4.7A to D). For isolated leg movements (Figs. 4.8A and B), and cranial sutures, the difference was not significant.[113]

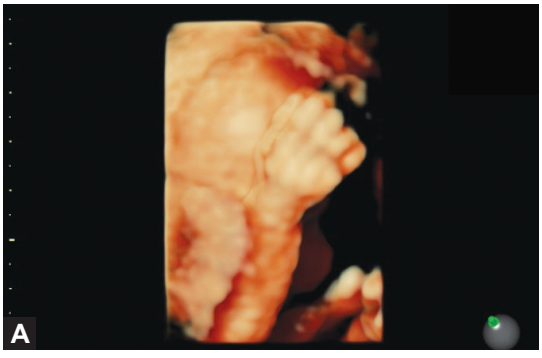


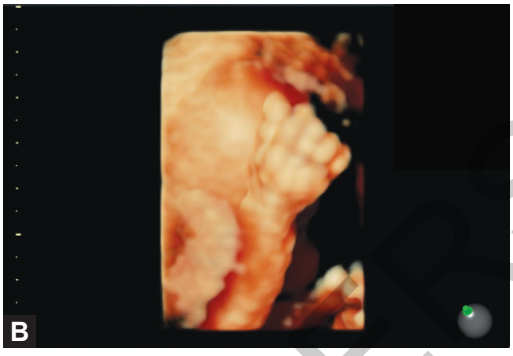
Figs. 4.3A to C: Anteflexion of the head of the fetal head seen in the (A) image, reverted back to the neutral position in (B) and (C) images again show a slight anteflexion.

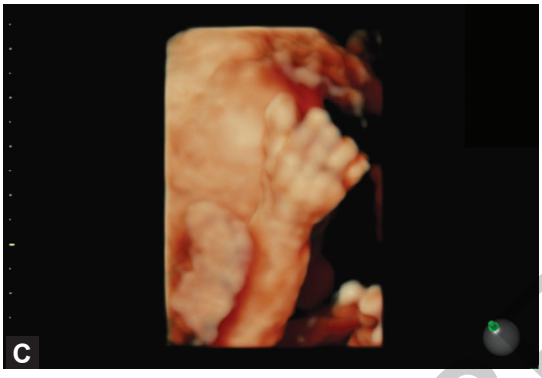


Figs. 4.4A to C: The series of images from (A) to (C) shows opening of the fetal mouth.

JAYPEE BROTHERS

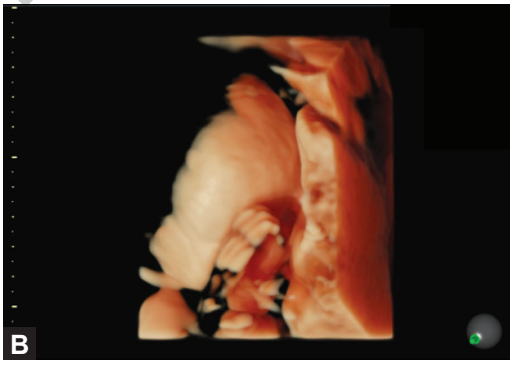


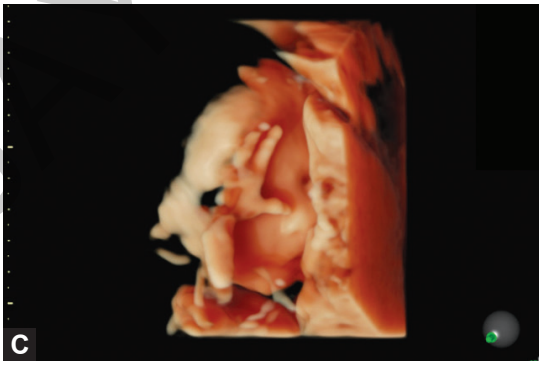




Figs. 4.5A to C: Movement of thumb noted as thumb fixed to fingers and flexed in (A) image; Thumb close to fingers but extended in (B) image; and Thumb separate from fingers in (C) image.

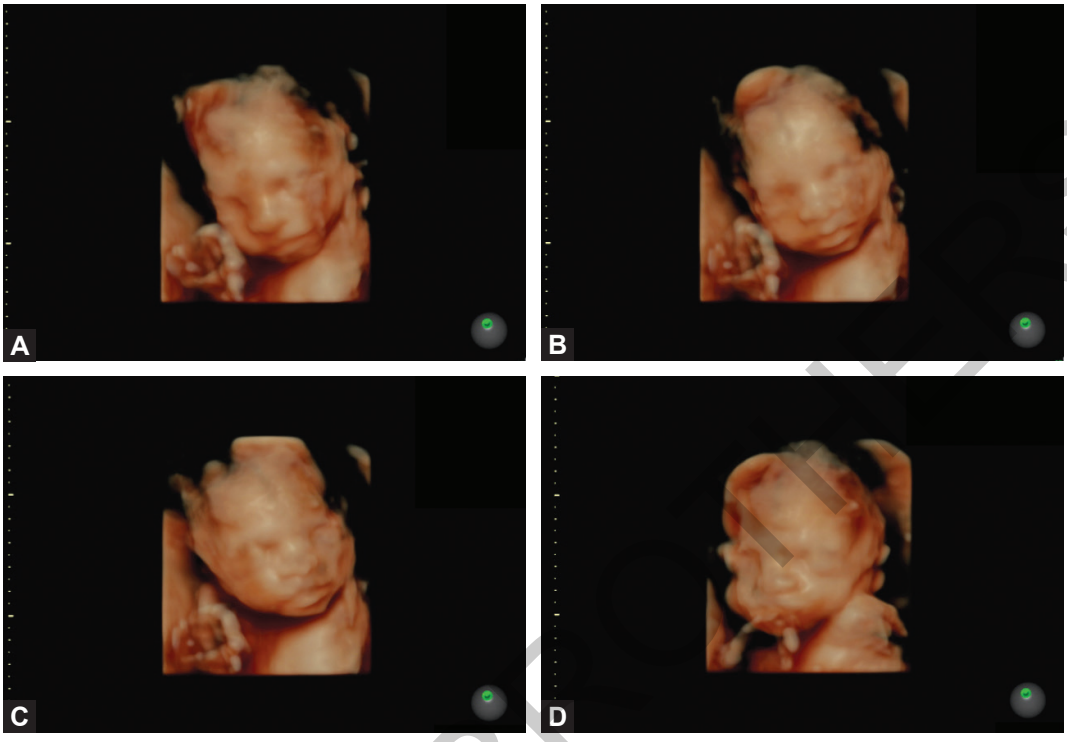




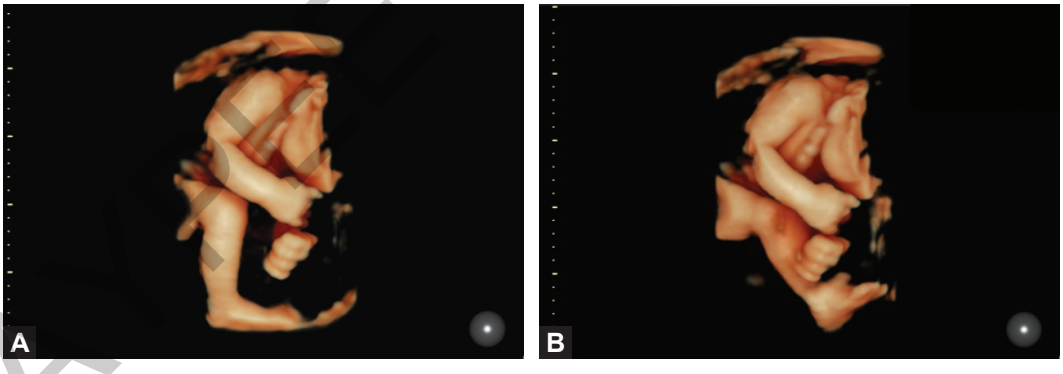


Figs. 4.6A to C: (A) Image of the fetal face with eye open and hand close to the chin; (B) The hand raised to fetal eye and eye is hidden by the hand; and (C) Supination of the hand and abduction of thumb and extension of fingers.

JAYPEE BROTHERS



Figs. 4.7A to D: Fetal head rotation and anteflexion movement with movement of the hand seen from (A) to (D), as a part of general movement.



Figs. 4.8A and B: (A) Partially flexed leg is seen and (B) Extension of the same is seen.

Athanasiadis et al., in 2013, applied KANET test to assess and compare fetal behavior and neurodevelopment in 152 pregnant women, classified as low-risk (n = 78) and high-risk (n = 74) pregnancies in the 2nd and 3rd trimester.[39] The neurodevelopmental score was statistically significantly higher in the low-risk group compared to the high-risk group.[39] Though the score was higher in diabetes subgroup compared to the IUGR and the preeclampsia subgroup.[113]

CONCLUSION

Inadequate glycemic control and vascular pathologies are the chief causes of neurological developmental inadequacies in fetuses of diabetic mothers. These can be assessed and predicted antenatally by KANET test.

REFERENCES

1. Roodenburg PJ, Wladimiroff JW, van Es A, et al. Classification and quantitative aspects of fetal movements during the second half of normal pregnancy. Early Hum Dev. 1991;25(1):19-35.
2. de Vries JI, Fong BF. Normal fetal motility: an overview. Ultrasound Obstet Gynecol. 2006;27(6): 701-11.
3. Prechtl HF. Qualitative changes of spontaneous movements in fetus and preterm infant are a marker of neurological dysfunction. Early Hum Dev. 1990;23(3):151-8.
4. DiPietro JA. Neurobehavioral assessment before birth. Ment Retard Dev Disabil Res Rev. 2005;11(1):4-13.
5. Kurjak A, Predojevic M, Stanojevic M, et al. Intrauterine growth restriction and cerebral palsy. Acta Inform Med. 2010;18(2):64-82.
6. Einspieler C, Prechtl HF, Ferrari F, et al. The qualitative assessment of general movements in preterm, term and young infants—review of the methodology. Early Hum Dev. 1997;50(1):47-60.
7. Moster D, Wilcox AJ, Vollset SE, et al. Cerebral palsy among term and postterm births. JAMA. 2010;304(9):976-82.
8. Yigiter AB, Kavak ZN. Normal standards of fetal behavior assessed by four-dimensional sonography. J Matern Fetal Neonatal Med. 2006;19(11):707-21.
9. Rees S, Harding R. Brain development during fetal life: influences of the intra-uterine environment. Neurosci Lett. 2004;361(1-3):111-4.
10. Kurjak A, Carrera JM, Stanojevic M, et al. The role of 4D sonography in the neurological assessment of early human development. Ultrasound Rev Obstet Gynecol. 2004;4(3):148-59.
11. Araki M, Nishitani S, Ushimaru K, et al. Fetal response to induced maternal emotions. J Physiol Sci. 2010;60(3):213-20.
12. Salihagić-Kadić A, Medić M, Kurjak A, et al. Four-dimensional sonography in the assessment of fetal functional neurodevelopment and behavioral patterns. Ultrasound Rev Obstet Gynecol. 2005;5(2):154-68.
13. Andonotopo W, Kurjak A, Azumendi G. Ultrasound studies on early pregnancy. In: Carrere JM, Kurjak A (Eds). Atlas of Clinical Application of Ultrasound in Obstetrics and Gynecology, 1st edition. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2006.
14. Azumendi G, Arenas JB, Andonotopo W, et al. Three-dimensional sonoembryology. In: Kurjak A, Arenas JB (Eds). Donald School Textbook of Transvaginal Sonography, 1st edition. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2006.
15. Kurjak A, Predojevic M, Stanojevic M, et al. The use of 4D imaging in the behavioral assessment of high risk fetuses. Imaging Med. 2011;3(5):557-69.
16. Kurjak A, Carrera J, Medic M, et al. The antenatal development of fetal behavioral patterns assessed by four-dimensional sonography. J Matern Fetal Neonatal Med. 2005;17(6):401-16.
17. Andonotopo W, Medic M, Salihagic-Kadic A, et al. The assessment of fetal behavior in early pregnancy: comparison between 2D and 4D sonographic scanning. J Perinat Med. 2005;33(5): 406-14.
18. de Vries JI, Visser GH, Prechtl HF. The emergence of fetal behavior. I. Qualitative aspects. Early Hum Dev. 1982;7(4):301-22.
19. D'Elia A, Pighetti M, Moccia G, et al. Spontaneous motor activity in normal fetuses. Early Hum Dev. 2001;65(2):139-47.

JAYPEE BROTHERS

for detailed information on obstetrics & gynecology books, visit our website www.jaypeebrothers.com, for detailed information on obstetrics & gyn

Donald School Textbook of
Diabetic Pregnancy and Ultrasound

Salient Features

- This book is one of the very rare monographies on diabetes in pregnancy
- Contributors are renowned authors from several countries.
- Contains most significant advances in this new field in particular study of neurobehavior in fetuses of diabetic mothers.



Badreldeen Ahmed FRCOG MD (Newcastle-Upon Tyne) MBCHB MFFP CCST FAcadEd is currently Director of Feto Maternal Medicine Centre, Professor at Qatar University, and Professor of Obstetrics at Weill Cornell Medical College in Doha, Qatar. Dr Ahmed was Ex-chairman of Obstetrics and Gynecology at Women's Hospital and founder of Fetal Maternal Medicine unit at Hamad Medical Corporation in Doha. He is Board Member of The World Association of Perinatal Medicine and The Fetus as the Patient Society and Founder of the African Association of Perinatal Medicine. Dr Ahmed is Regional Director of the Ian Donald Inter-University School of Ultrasound in Obstetrics and Gynecology. Prof Ahmed published over 70 papers in peer reviewed journals. He was editor of Basic Book of Ultrasound in Obstetrics and Gynecology and author of several chapters in books.



Asim Kurjak MD PhD is currently serving as Professor of Obstetrics and Gynecology at Medical School Universities of Zagreb and Sarajevo. From 1985 to 2002, Dr Kurjak served as Head of the Department of Obstetrics and Gynecology, Medical School University of Zagreb, Sveti Duh Hospital.

Dr Kurjak has published over 500 papers in peer review literature. His papers have been cited more than 11,000 times. He has authored, co-authored or co-edited 113 books. He is the President of International Academy of Perinatal Medicine and Director of Ian Donald Inter-University School of Medical Ultrasound. He is also Editor-in-chief of the Donald School Journal of Ultrasound in Obstetrics and Gynecology. He received a Doctorum Honoris Causa from the Universities in Banja Luka, Budapest, Barcelona, Bucharest, Athens, Montevideo, Tomsk, Buenos Aires, St Petersburg, Moscow, Tirana and Khartoum.

Available at all medical bookstores
or buy online at www.jaypeebrothers.com

JAYPEE BROTHERS
Medical Publishers (P) Ltd.
www.jaypeebrothers.com

Join us on facebook.com/JaypeeMedicalPublishers

Shelving Recommendation
OBSTETRICS & GYNECOLOGY

ISBN 978-93-5270-196-4