The study of the onset and ontogeny of human behaviour has made it clear that a multitude of fetal movement patterns are spontaneously generated, and that there is a close association between activity and the development of peripheral and central structures. The embryo starts moving by 7.5 week’s gestation; 2 to 3 weeks later, a number of movement patterns including general movements, isolated limb and head movements, hiccup, and breathing movements appear. Some movements (e.g. yawning, smiling, ‘pointing’; we show these in eight videos in this review) precede life-long patterns; others have intrauterine functions, such as sucking/swallowing for amniotic fluid regulation, breathing movements for lung development, or eye movements for retinal cell diversity. In cases of developmental brain dysfunction, fetal general movements alter their sequence and gestalt, which suggests a dysfunction of the developing nervous system. The scarcity of longitudinal studies calls for further comprehensive research on the predictive value of prenatal functional deviations.

The term ‘sensory’ is always used first when we discuss the central nervous system as a functional unit. ‘Sensory’ functions, ‘sensorimotor’ functions, ‘sensorimotor’ neuroscience, ‘sensorimotor’ deficits) but the actual ontogeny of behaviour is quite the opposite. The developing nervous system in its early stages ought to be seen as a motor-sensory organ, therefore, we should put the term motor first. Teaching at the University of Jena, Germany, in 1885, the British physiologist William T Preyer was the first to describe overt motor activity in the chick embryo several days before sensory stimulation effectively evoked reflexes. Preyer’s observation was confirmed by Viktor Hamburger and his associates, who proved that a motor output can indeed occur in the absence of an input from the sensory receptors. A series of pioneering experiments established non-reflexogenic, spontaneous motility as the primary component of embryonic behaviour in birds and mammals, including humans. Modern neurobiology has coined the term ‘central pattern generator’ for the underlying neural circuitry of endogenously generated activity that is not triggered by sensory stimulation but can be modulated by the periphery. Spontaneous motility is characterized by rhythmicity, which is inherent not only in alternating movements for locomotion, sucking, and breathing movements, but also in the alternation of short phases of activity followed by longer phases of inactivity, which is a typical pattern of fetal behaviour. Embryonic/fetal movements are an integral part of typical development, as skeletal, muscular, and neural developments in particular are activity-dependent. If embryonic/fetal neuromuscular activity is silenced pharmacologically or by disease, the population of spinal motor neurons, the transmitter specification, the pattern of neuromuscular synaptic contact, and genetically determined cell death develop atypically.

**THE ORIGINS OF HUMAN BEHAVIOUR: WHEN IT ALL BEGINS**

Expectant mothers feel the first quickening between 14 and 22 weeks’ gestation (gestational age refers to postmenstrual age) when the fetus moves forcefully enough to press against the maternal abdominal wall. As long as 2400 years ago, Hippocrates suspected that fetal movements set in 2 to 3 weeks before the mother can feel them, but it was not until the 1970s that the first sonar studies – though not yet real-time – enabled the Viennese obstetrician Emil Reinold to describe lively movements such as ‘swimming in the amniotic cavity’ alternating with ‘slow and lazy movements’ followed by minutes of rest. With the advent of real-time ultrasonography a few years later, an attempt was made to determine the age at which certain movement patterns emerge. Van Dongen and Goudie saw the heart pulsating in embryos with a crown-rump length of 5 to 12 mm, and determined the onset of heart motion at 5 to 6 weeks’ gestation. Two weeks later, body movements set in. They consist of cyclically performed sideways bending of the head and/or rump (Video S1, online supporting information), sometimes accompanied by a little activity in the limbs a few days later.
GENERALIZED AND ISOLATED MOVEMENTS: AN ALMOST SIMULTANEOUS ONSET

By 8 to 10 weeks’ gestation, movements involve the entire body. They can be brisk, in the form of startles initiated in the limbs and spreading to the neck and trunk, or slower and more complex, in which case they are called general movements (Fig. 1). It seems that the simultaneous ontogenetic onset of these two movement patterns has an adaptive function, since startles, which lead to a displacement of the body, often induce a general movement.8,12,15 Transvaginal ultrasonographic recordings have shown that from 9.5 weeks’ gestation onwards, general movements vary in speed, amplitude, and direction, but also in the sequence of body parts involved (Video S2, online supporting information). Lüchinger et al.15 have suggested a timely coincidence between the formation of the subplate and the onset of variation. Prechtl also assumed that general movements are generated and modulated supraspinally.5 Various pregnancy-related and maternal factors have an effect on both their quantity and modulation, the latter resulting in less variable (poor repertoire) general movements (Table 1).

Though one might assume that, at this early stage, isolated movements should be more difficult to produce for the nervous system than movements of the entire body, isolated limb movements emerge only a few days after startles and general movements (Fig. 1). From 12 weeks onwards, arm extension is frequently accompanied by finger extension. At the same age, the hand regularly touches the head, face, and sometimes the mouth. Isolated finger movements emerge at around 13 weeks’ gestation; cloni and twitches appear after 14 weeks, but are rare even then.8,12 During the third trimester, the fetus manipulates and even squeezes the umbilical cord (Fig. 2a), which may result in increased vagal activity causing fetal heart-rate decelerations by 15 to 60 beats per minute.18 Any hypoperfusion is most likely to be transient because the grasp reflex usually ceases after a short while. Most remarkably, isolated index finger extension, which only becomes functional as a pointing gesture by the end of the first year of life (Fig. 2b), can already be observed during pregnancy.19

Isolated leg movements are less frequent than isolated arm movements but emerge at the same time (10–11wks’ gestation), which indicates that motor system development does not progress in a craniocaudal sequence. Rapid or slow isolated leg movements can occur (1) unilaterally, as a sporadic kick displacing the fetus from its resting position, or (2) bilaterally, as alternating leg movements (Fig. 2c), which also reposition the fetus.5,8

DIAPHRAGMATIC MOVEMENTS: WHY SO EARLY?
The formation of the diaphragm is completed by 10 weeks’ gestation, providing the anatomical substrate for the onset of hiccup and breathing movements, although the former can sometimes already be observed after 8 to 9 weeks,12 when phrenic motor neurons seem to possess sufficient synapse contacts.20 It has been suggested that hiccup is a manifestation of a programmed isometric inspiratory muscle exercise that smooths the subsequent diaphragmatic motion necessary for breathing movements.21 Hiccup decreases during the last 10 weeks of gestation, which suggests that brain development may have an inhibitory effect on hiccup.8,22 Only compromised, near-term fetuses continue to show a high frequency of hiccup occurrence after all other movements have ceased.8

| Heart motion | Sideways bending of the head and/or rump | Startles | General movements | Hiccup | Isolated limb movements | Isolated head movements | Jaw opening | Hand-to-face contact | Breathing movements | Opening and closing of the fingers | Sucking | Swallowing |
|--------------|----------------------------------------|---------|-------------------|-------|-------------------------|------------------------|-----------|---------------------|------------------|-------------------------------|---------|-----------|
| 5            | 6                                      | 7       | 8                 | 9     | 10                      | 11                     | 12       | 13                  | 14               | 15                            | 16      | 17        |

Gestational weeks

What this paper adds
- Motor output can occur in the absence of sensory input.
- Structural development is activity-dependent.
- Fetal general movements are among the first movement patterns to occur.
- Pregnancy-related and maternal factors impact quantity and modulation of fetal general movements.
- Prenatal general movement assessment has not yet brought the expected breakthrough.
Two to 4 weeks after the onset of hiccup, fetal breathing movements set in. The diaphragm smoothly shifts in a caudal direction, causing the anterior chest to move inwards and the abdominal wall to move outwards, and vice versa, in alternation (paradoxical breathing movements; Video S3, online supporting information). Episodes of fetal breathing movements occur irregularly (until 00:10 in Video S3) or regularly (after 00:10 in Video S3).

Interestingly, both general and breathing movements are unrelated within pairs of twins, which suggests that the motor development of twins is largely independent of their co-twins’ development.23

An adequate intrathoracic space and amount of amniotic fluid as well as normal fetal breathing movements are required for lung development. Insufficiency or absence of these movements is associated with decreased proliferation and increased programmed death of pulmonary cells. Fetal breathing movements are also required for the morphological differentiation of type I (gas exchange) and type II pneumocytes (secreting pulmonary surfactant).8,24

Fetal breathing movements can best be observed after maternal glucose intake, especially in the early morning, but they decrease after maternal fasting and in the late evening. During labour, they are absent. Pathophysiological conditions that decrease fetal breathing movements include maternal smoking, alcohol and methadone abuse (Table 1), maternal hyperventilation, preterm rupture of the membrane, and severe fetal hypoxia.8

**Table 1: Determinants of fetal activity8,16**

| Body movements                          | Breathing movements                                      | Behavioural states          |
|-----------------------------------------|----------------------------------------------------------|-----------------------------|
| Oligohydramnios                         | Smaller amplitude, slow                                  | Decreased*                  |
| IUGR                                    | Decreased, smaller amplitude, slow, poor repertoireb     | Delayed, disorganised       |
| Severe maternal stressc                 | Hyperactive with disordered, vigorous movements for 2–8 hours, followed by reduced motility for up to 72 hours | -                           |
| Substance abuse                         |                                                          |                             |
| Nicotine                                | Temporarily reduced                                      | Decreasedd                  |
| Alcohol                                 | Decreased, slow                                          | Prolonged state 1F          |
| Cocaine                                 | Excessive                                                | Suppressed                 |
| Methadone                               | Decreased                                                | Tremendously disorganized   |
| Therapeutic drugs                       |                                                          | Delayed, disorganized       |
| Barbiturates                            | Decreased                                                |                             |
| Benzodiazepines                         | Decreased                                                |                             |
| Aminophylline                           | No effect                                                |                             |
| Morphine                                | Decreased for 1–3 days                                    |                             |
| Glucocorticoidsa                        | Dose-related increase                                    |                             |
| Selective serotonin                     | Small amplitude,                                         |                             |
| Reuptake inhibitors                     |                                                          |                             |
| Antiseizure medicationf                 | Poor repertoireb                                         |                             |
| Maternal type-I diabetes                | Abnormal, poor repertoireb                                |                             |

*Often associated with pulmonary hypoplasia. bThe sequence of movement components is monotonous, and the amplitude, speed, and intensity lack the normal variability. cAccidents, earthquakes, serious illness, death of a close relation, marital discord, rape. dRelated to plasma nicotine but unrelated to carboxyhaemoglobin concentration. eBetamethasone effects fetal behaviour more than dexamethasone.

**FETAL ONSET, LIFELONG PRESENCE**

Apart from the movement patterns described above, a 12-week-old fetus also demonstrates isolated retro- or anteflexion of the head, the latter of which sometimes co-occurs with hand-to-mouth contact (Fig. 1). Also at 12 weeks, two other complex motor patterns set in and will remain throughout life: stretches and yawns. Fetal stretching is always slow. It is characterized by marked trunk extension, head retroflexion, and arm elevation in outward rotation.12 Sometimes it is accompanied by a brief deceleration of the fetal heart rate. Fetal yawning is an antecedent of a lifelong and phylogenetically old pattern that occurs across species – in fish, amphibians, reptiles, birds, and mammals. Its most probable purposes are arousal and communication, since it increases the circulation of cerebrospinal fluid.25 A fetal yawn starts with a 9- to 13-seconds-lasting slow, usually wide opening of the jaw and simultaneous downward movement of the tongue as well as retroflexion of the head. When fully opened, the mouth remains open for 2 to 8 seconds, then it shuts quickly and the fetus returns to the initial position (Video S4, online supporting information).8,12

From 14 weeks onwards, the fetus performs rhythmic bursts of regular jaw opening and closing followed by swallowing movements, which indicate drinking amniotic fluid. This is evidenced by flow signals in the oronasal cavity (Fig. 3).8,12 These movements are generated by central pattern generators in the brainstem (nucleus tractus solitarius.
and the dorsal medullar reticular formation) and modulated sensorily by the reticular formation." Fetal sucking/swallowing is both preparatory and functional as one of the main regulators of the amniotic fluid volume. In addition, fetal sucking/swallowing changes the composition of the fluid in the nasal and laryngeal pathways and thus plays a crucial role in the development of chemosensation. During the third trimester, sucking increases, as does the amount of amniotic fluid swallowed (500ml per day). By that time, the fetus will occasionally suck on the umbilical cord, which does not cause bradycardia and is, therefore, not associated with cord occlusion.

FACIAL EXPRESSIONS AND FETAL EYE MOVEMENTS

Facial expressions such as smiling movements, pouting, or scowling can be observed during the last trimester of pregnancy by means of 4D ultrasound recordings, but their early ontogeny is not yet known. Fetal eye movements are particularly interesting (Video S5, online supporting information): fast deviations with slower/faster reposition, single prolonged eye movements, complex sequences, and nystagmoid eye movements can be observed from 15 to 17 weeks onwards. From about 30 weeks onwards, clusters of rapid eye movements can be observed. The fetus also starts to display blinking by opening and closing eyelids at 23 to 26 weeks' gestation. Birnholz, who proposed the four types of fetal eye movements described above, noticed that blinking could be experimentally elicited through light. This observation was recently confirmed with laser dot diodes in last-trimester fetuses. Amazingly, blood-oxygen-level-dependent resting state functional magnetic resonance imaging (MRI) has shown that fetal eye movements are already linked to networks in visual and frontal cerebral areas from 30 weeks onwards. On a cellular level, animal experiments have demonstrated that the genesis of retinal cell diversity depends on fetal eye movements. The differentiation of cholinergic amacrine cells in the retina is particularly activity-dependent and related to postnatal motion vision.

THERE IS NOTHING LIKE A TYPICAL FETAL POSTURE

There is a common misconception, probably inspired by Leonardo Da Vinci's drawings, that all joints are flexed in the typical fetal posture, but in fact both the posture and the position of the fetus are variable and change frequently, especially during the first half of pregnancy. Positional changes are usually achieved through rotational movements along the longitudinal axis of the fetus, through alternating leg movements (Fig. 2c) and, sometimes, somersaults. In relation to the surface that the fetus is resting on, the posture can be supine, prone, sitting or standing upright, or upside down. During the first 20 weeks of pregnancy especially, fetuses occasionally raise their hands and place them behind the head; sometimes they even cross their legs and lie as though ‘relaxing in a hammock’.

FETAL MOTILITY CLUSTERS IN REST AND ACTIVITY CYCLES

By 20 weeks' gestation, fetal activity shows a diurnal variation with peaks in the late evening. In 1982, Nijhuis et al. described four fetal heart rate patterns coinciding with the presence/absence of fetal body and eye movements (Table 2). Such coincidences occur from around 30 weeks' gestation onwards and develop into distinct behavioural states in the near-term fetus. As fetal states reflect fetal brain maturation, they are analysed to assess the integrity of the nervous system. Fetuses with a synchronous change of state variables (heart rate, eye and body movements; Table 2) reach high levels of self-regulation and effortful control during childhood and adolescence. Dysfunctions in behavioural states are reflected in a high percentage of non-coincidence of state parameters and in a lack of synchronicity during state transitions. Fetal growth restriction, various congenital abnormalities as well as maternal type-I diabetes, maternal intake of corticosteroids, caffeine, or alcohol, are associated with fetal state dysfunction (Table 1).
Since fetal MRI was established more than 20 years ago, fetal brain development can be studied in vivo from 18 weeks’ gestation onwards, and in some advanced centres even in the form of dynamic MRI. Dynamic MRI allows us to watch 5 to 20 images per second and thus visualize fetal movements in real-time including swallowing (Fig. 3; Video S6, online supporting information), yawning and distal limb movements (Fig. 2; Video S6), as well as atypical movements (rhythmic jerks, Video S7, online supporting information; vomiting, Video S8, online supporting information), enabling us to draw conclusions about the underlying pathology. Apart from this advanced technology, 2D ultrasonography is still the most widely used and reliable tool for assessing the quantity and quality of fetal movement patterns. So far, several approaches that include fetal movements in their protocols have either disregarded typical developmental characteristics of behavioural states or have based their assessment of fetal motor functions on 3D or 4D ultrasound recordings. The latter are insufficient for comprehensive analyses as they fail, for example, to identify fluent and smooth movements.

Relying on 2D ultrasonography in the 1980s and 90s, Heinz Prechtl and associates were convinced that the general movements assessment (GMA; now a cornerstone of infant neurology) could also be applied prenatally. They were sure that it would make a significant contribution to fetal neurology. However, there is still a dearth of systematic empirical evidence on the changing quality of fetal general movements (though not on their quantity), as de Vries and Fong have reiterated for more than a decade. It takes a lot of training and experience to decode the complexity of normal or poor repertoire abnormal general movements by assessing their sequence, amplitude, speed, and rotatory parameters. In cases of brain dysfunction, movements alter these parameters and appear strikingly monotonous. Brisk, jerky, sometimes excessive general movements can be seen in early stages of fetal akinesia deformation sequence, myotonic dystrophy, and anencephalic fetuses, but might also indicate acute fetal distress before fetal demise. Poor repertoire general movements with a reduced amplitude and slow pace have been observed in growth-restricted fetuses, fetuses exposed to oligohydramnios, maternal diabetes, or maternal anti-convulsive medication (Table 1). This non-exhaustive list makes it clear that poor repertoire general movements are not specific enough to be associated with individual neurodevelopmental alterations. What is more, although the reproducibility and consistency of these findings has been demonstrated in other infants during the postnatal period, hardly any longitudinal studies have applied GMA pre- and postnatally within the same cohort. Among the few exceptions are: (1) short-term observations of fetuses/newborn infants, e.g. with spina bifida aperta (where general movements, including the legs, are normal in utero...
and remain normal during the first 2 days after birth if the myelomeningocele is located caudally from L3; then leg movements become abnormal and eventually disappear); and (2) a study on almost 100 fetuses exposed to maternal hypertensive disorder and/or oligohydramnios with a 64% rate of abnormal fetal general movements, which correlated with moderate echogenicity alterations (in the periventricular, basal ganglia, and thalamic area) but normalized to a great extent during the first week after birth. Follow-up studies are still scarce. For example, one study demonstrated that poor repertoire general movements in fetuses of mothers with type-I diabetes were related to lower Bayley developmental indices at 10 months of age. Thus, prenatal GMA has not brought the expected breakthrough, yet. The value of these spontaneously generated movements for typical development remains undisputed, and we also continue to believe in their potential value for identifying atypical neurological development, but our recording and assessment tools simply lack the refinement (and we lack the knowledge) required to understand the meaning of various motor patterns. Furthermore, compared to dynamic MRI recordings, ultrasound recordings hardly allow bringing the whole fetus into a single video frame from mid-pregnancy onwards. This makes prenatal GMA difficult and requires extensive training and expertise. Dynamic MRI is only available in very few specialized centres, apart from being much more expensive.

We need to learn more about the onset, co-occurrence, pattern structure, and ontogeny of fetal motor functions in their physiological context if we are to identify maturational deviations through neurofunctional analysis. Therefore, while we remain convinced of the significance of fetal general movements for brain maturation, subsequent behavioural functions, and development in general, we also concede that there is an urgent need for comprehensive multidisciplinary studies on the origin of human behaviour, starting with intrauterine assessments, if we are to better understand this critical period of development.

ACKNOWLEDGEMENTS

Dedicated to Heinz F R Prechtl (1927–2014), a pioneer in observing and understanding fetal behaviour. CE and PBM are supported by FWF (P25241, KLI-811, TCS24), the Leibniz ScienceCampus, Laerdal Foundation, and the Volkswagen Foundation (IDENTIFIED). The authors thank Gunter Vogrinec for his support in the graphical presentation of findings, and Miha Tavcar (www.scriptophil.at) for initial copy-editing of the paper. The authors have stated that they had no interests that might be perceived as posing a conflict or bias.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

SUPPLEMENTARY INFORMATION

The following additional material may be found online:

Video S1: First body movements – transvaginal ultrasound recording at 7.5 weeks’ gestation.

Video S2: General movements – ultrasound recording at 14 weeks’ gestation.

Video S3: Irregular and regular breathing movements – ultrasound recording at 14 weeks’ gestation.

Video S4: Fetal yawning – ultrasound recording at 14 weeks’ gestation.

Video S5: Fetal eye movements – dynamic MRI at 30 weeks’ gestation.

Video S6: Fetal movement repertoire – dynamic MRI at 27 weeks’ gestation.

Video S7: Rhythmic jerks – dynamic MRI at 33 weeks’ gestation.

Video S8: Vomiting due to brainstem hypoplasia – dynamic MRI at 23 weeks’ gestation.
REFERENCES

1. Preyer WT. Special Physiology of the Embryo. Leipzig: Grieben, 1885 [in German].
2. Hamburger V. Some aspects of the embryology of behaviour. Q Rev Biol 1963; 38: 342–65.
3. Hamburger V, Wenger E, Oppenheim R. Motority in the absence of sensory input. J Exp Zool 1966; 162: 133–60.
4. Bekoff A. Spontaneous embryonic motility: an enduring legacy. Int J Dev Neurosci 2001; 19: 155–60.
5. Prechtl HFR. General movement assessment as a method of developmental neurology: new paradigms and their consequences. THE 1999 Ronnie MacKeith Lecture. Dev Med Child Neurol 2001; 43: 836–42.
6. Grillner S, El Manira A. Current principles of motor control, with special reference to vertebrate locomotion. Physiol Rev 2020; 100: 271–320.
7. Barlow SM. Central pattern generation involved in oral and respiratory control for feeding in the term infant. Curr Opin Otolaryngol Head Neck Surg 2009; 17: 187–93.
8. Einspieler C, Prayer D, Prechtl HFR. Fetal Behaviour: A Neurodevelopmental Approach. Clinics in Developmental Medicine, No. 189. London: Mac Keith Press, 2012.
9. Marder E, Rehm KJ. Current principles of motor control, with special reference to vertebrate locomotion. Physiol Rev 2020; 100: 271–320.
10. Yaginuma H, Tomita M, Takashita N, et al. A novel method of developmental neurology: new paradigms and their consequences. THE 1999 Ronnie MacKeith Lecture. Dev Med Child Neurol 2001; 43: 836–42.
11. Moessinger AC. Fetal akinesia deformation sequence: an animal model. Pediatr Res 1983; 24: 855–67.
12. de Vries JIP, Visser GHA, Prechtl HFR. The emergence of fetal behaviour. I. Qualitative aspects. Early Hum Dev 1982; 7: 301–22.
13. van Dongen LGR, Goudie EG. Fetal movement patterns in the first trimester of pregnancy. Br J Obstet Gynaecol 1980; 87: 191–3.
14. Felt RHM, Mulder EJH, Löchinger AB, van Kan CM. Transverse MAM, de Vries JIP. Spontaneous cyclic embryonic movements in humans and guinea pigs. Dev Neurosci 2012; 34: 1133–9.
15. Löchinger AB, Hadders-Algra M, van Kan CM, de Vries JIP. Fetal onset of general movements. Pediatr Res 2008; 63: 191–5.
16. Mulder EJH, Ververs FF, de Heus R, Visser GHA. Selective serotonin reuptake inhibitors affect neurobehavioural development in the human fetus. Neuropharmacology 2011; 66: 1961–71.
17. Marchik PB, Zhang D, Esposito G, Bölte S, Einspieler C, Sigristo J. A new pathway of motor development in children with neurodevelopmental disorders? Behav Brain Sci 2017; 40: 1–5.
18. Habeck D, Habeck JC, Barbir A, Barbir M, Granič P. Fetal grasping of the umbilical cord and perinatal outcome. Arch Gynecol Obstet 2003; 268: 274–7.
19. Marchik PB, Prechtl HFR, Prayer D, Peyton C, Einspieler C. An antecedent of later developing communication functions: the fetal index finger. BMJ 2013; 347: f7232.
20. Greer JJ, Martin-Cabrallo M. Developmental plasticity of phrenic motoneuron and diaphragm properties with the inception of inspiratory drive transmission in utero. Exp Neurol 2017; 287: 183–47.
21. Kharilas PJ, Shi G. Why do we hiccup? Gut 2001; 41: 271–5.
22. Kamata H, Ryo E, Seto M, Morita M, Nagaya Y. Counting fetal hiccups using a fetal movement acceleration measurement recorder. J Matern Fetal Neonatal Med 2017; 30: 475–8.
23. Tendais I, Figueredo R, Mulder EJH, Lopes D, Montenegro N. Developmental trajectories of general and breathing movements in fetal twins. Dev Psychobiol 2019; 61: 626–33.
24. Inanlou MR, Baguma-Nibasheka M, Kahlar B. The role of fetal breathing-like movements in lung organogenesis. Int J Dev Neurosci 2019; 36: 274–8.
25. Kainer F, Prechtl HFR, Engele H, Einspieler C. The relationship between eye movement and visual development before birth. Front Hum Neurosci 2014; 8: 775.
26. Baguma-Nibasheka M, Reddy T, Abbas-Butt A, Kahlar B. Fetal ocular movements and retinal cell differentiation: analysis employing DNA microarrays. Hum Mol Genet 2006; 15: 1311–7.
27. Nijhuis JG, Prechtl HFR, Martin CB, Bors RSGM. Are there behavioral states in the human fetus? Early Hum Dev 1982; 6: 177–94.
28. Brandle J, Preusl H, Draganova R, et al. Heart rate variability parameters and fetal movement complement fetal behavioral states detection via magnetoencephalography in newborn infants. Front Hum Neurosci 2015; 9: 147.
29. van den Bergh BRH, Mulder EJH. Fetal sleep organization: a biological precursor of self-regulation in childhood and adolescence? Biol Psychol 2012; 89: 584–90.
30. Mallath-Pokorny M, Kasprau G, Miller C, Schöpf V, Nemec U, Prayer D. Magnetic resonance methods in fetal neurology. Semin Fetal Neonatal Med 2012; 17: 278–84.
31. Novak I, Morgan C, Adde L, et al. Early, accurate diagnosis and early intervention in cerebral palsy: advances in diagnosis and treatment. JAMA Pediatr 2017; 171: 897–907.
32. Prechtl HFR, Einspieler C. Is neurological assessment of the fetus possible? Eur J Obstet Gynaecol Reprod Biol 1997; 75: 81–4.
33. de Vries JIP, Fong BF. Changes in fetal motility as a result of congenital disorders: an overview. Ultrasound Obstet Gynecol 2007; 29: 590–9.
34. Whitehead CL, Cohen N, Visser GHA, Farine D. Are increased fetal movements always reassuring? J Matern Fetal Neonatal Med 2020; 33: 3713–8.
35. Sival DA, Weerden TWV, Vles JSH, et al. Neonatal loss of motor function in human spina bifida aperta. Pediatr Res 2004; 55: 427–34.
36. Reier-van Dunne FM, van Wezel-Meijler G, Bakker MP, de Groot L, Olendzaal HJ, de Vries JIP. General movements in the perinatal period and its relation to echocardiographic and electrocardiographic changes in the brain. Early Hum Dev 2010; 86: 83–6.
37. Kainer F, Prechtl HFR, Engele H, Einspieler C. Assessment of the quality of general movements in fetuses and infants of women with type-I diabetes mellitus. Early Hum Dev 1997; 50: 13–25.
38. van der Knoop BJ, van Schar PEM, Vermeulen RJ, Pistorius LR, van Weissenbruch MM, de Vries JIP. Effect of (minor or major) maternal trauma on fetal motility: a prospective study. Early Hum Dev 2015; 91: 511–7.