Obstetrica şi Ginecologia LXVI (2018) 135-140 **Review article**

CHILDBIRTH PAIN. FROM MYTH TO REALITY

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Abstract

Childbirth pain is considered one of the most intense human experiences. At the level of the spine it follows the same pathways as common acute pain.

Nerve impulses generated by pain determines an information transfer to the neurons localized in the anterior and lateral horns resulting in a segmental response; at the lebel of the brainstem a supra segmental response and in the upper brain a cortical response. This is the site where the pain is perceived and generates a psychological reaction (emotion, anxiety, fear, motivation, judgment) and a behavioral response (verbal, somatic).

On the other hand pain and emotional stress modifies uterine contractions and the span of labor as a result of rising levels of catecholamines. Further more, pain determines the increase of pelviperineal muscles tonus with subsequent modifications in the dynamic and progression of labor.

During uterine contraction an alteration of placental changes occur. Severe hyperventilation and respiratory alkalosis induced by pain worsen these changes trough modifications of hemoglobin dissociation curve (decreases O2 transfer from mother to fetus) and trough maternal hypoxemia in between uterine contractions. A "fragile" fetus can develop acute distress as a result O2 and CO2 altered exchanges induced by pain.

All these date sustained a continuous research for best methods to cope with childbirth pain. Numerous techniques and methods were utilized trough time (Lamaze psycho prophylaxis, hypnosis, acupuncture, opioid, tranquilizers, sedatives administration, inhalatory agents). Today, the answer is continuous epidural

Rezumat

Durerea la naştere este considerată una dintre cele mai intense dureri din experienţa umană. La nivelul măduvei, aceasta urmează acelaşi traseu şi aceeaşi modulare ca orice altă durere acută.

Impulsurile nervoase generate de durere determină la nivel medular un transfer de informaţie spre celulele din coarnele anterioare şi antero – laterale, rezultând un răspuns reflex segmentar; la nivelul trunchiului cerebral, răspunsul reflex suprasegmentar, iar la nivel cortical, răspunsul cortical. La acest nivel are loc percepţia durerii, reacţia psihologică (emoţie, anxietate, teamă, motivaţie, judecată) şi reacţia comportamentală (verbală, motorie).

Pe de altă parte, durerea şi stresul emoţional modifică contracţiile uterine şi durata travaliului prin creşterea nivelului de catecolamine. Mai mult, durerea determină creşterea tonusului musculaturii pelviperineale, cu tulburări de dinamică şi de progresiune a travaliului.

În timpul contracţiei are loc o alterare a transferului la nivel placentar. Hiperventilaţia severă şi alcaloza respiratorie, induse de durere, agravează aceste schimburi prin devierea spre stânga a curbei de disociere a hemoglobinei (scade transferul de O2 de la mamă la făt) 'i prin hipoxemie maternă între contracţiile uterine. În cazul unui făt "fragilizat" această alterare a schimburilor de O2 şi CO2 indusă de durere, poate favoriza instalarea suferinţei fetale.

Toate aceste date au stimulat cercetările pentru găsirea unor metode cât mai eficiente de analgezie la naştere.S-au încercat de-a lungul timpului o multitudine de metode şi tehnici (psihoprofilaxia Lamaze, hipnoza, acupunctura, administrarea sistematică de opioide, tranchilizante, sedative, agenţi inhalatori). În zilele noastre răspunsul în ceea ce priveste analgezia la naştere îl reprezintă peridurala continuă.

Cuvinte cheie: durere, nastere, analgezie, travaliu

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Introduction

The origin, purpose and treatment of childbirth pain are subjects debated from immemorial times. Understanding and controlling this pain was an endless preoccupation for humans.

First depictions of childbirth pain are found in ancient civilizations (Egyptians, Chinese, the Old Testament). Seen as a divine punishment for human beings in some religions and ancient mythology, the pain endured by women during childbirth continues to be a social conundrum that raises many questions and asks for yet to be proven safe solutions (1).

For Christians, this is part of the punishment for the original sin, as explained in the Bible, Genesis 3:16: "To the woman He said: I will greatly multiply your pain in childbirth. In pain you will bring forth children; Yet your desire will be for your husband, and he will rule over you." For Christians, only Mary being "the arc of the new covenant", free from the original sin, is thought to be fully absolved from childbirth pain. On the other hand, the Qur'an tells us that even Mary was not spared but for Muslims there is no punishment, only the acknowledgement of childbirth pain, with the glorification of the women who endures childbirth: ... And every soul earns not [blame] except against itself, and no bearer of burdens will bear the burden of another. ... — Qur'an 6:164 and the martyrization of maternal death . Any time a woman leaves this world because of labor pains, on the Day of Judgement, Allâh will raise her from the grave pure and without an account (of sins) ... [Bihâr al-Anwâr, vol. 101, pg. 108]. Descriptions of labor pain without explaining the origin are present in Judaism also. The author of the Book of Jeremiah, for example, refers to the "cry of a woman in travail, the anguish of one bringing forth her first child, gasping for breath, stretching out her hands crying `Woe is me!". Tribal rituals from Africa and South America reveal the acknowledgement of childbirth pain also without explaining it, a joyful and happy punishment from the Gods, with ceremonial efforts to ease the pain of the suffering women (2).

Even if it is prone to a purely subjective evaluation the pain during childbirth is perceived as highly severe, more intense than dental pain, ghost member pain or fracture bone pain (3).

Martius, in a more recent but incomplete analysis explains the occurrence of this suffering during childbirth as a tribute the humans are paying for biped position. The orthostatic position limited the inferior abdominal opening thus increasing the resistance to a pain free delivery (3, 4).

With the ongoing evolution of human society and women condition, in time, the pain and endurance during childbirth became more and more less accepted: "The position of woman in any civilization is an index of the advancement of that civilization; the position of woman is gauged best by the care given her at the birth of her child." (H. W. Haggard, 1929). With all the interest in this matter it would still take almost 20 years until January 19, 1847, the date when a Scottish obstetrician, James Young Simpson, administered for the first-time diethyl ether to a woman with a deformed pelvis to ease the delivery of her child. This is the first record of an anesthetic administration for childbirth in modern times. On the same day, Simpson received notice of his appointment as Queen Victoria's "Physician in Scotland" (2, 5)

The origin and pathways of pain during childbirth.

Pain during first stage of labour (dilatation).

During first stage of labour the pain is thought to have more origins: pressure at the level of nerve endings serving the uterine muscular fibers, contractions of an ischemic myometrium (vasoconstriction as a result of sympathetic hyperactivity), inflammatory modifications of the myometrium, sympathetic hyperactivity with consecutive cervical and isthmic constriction, cervical dilatation and segmental distension (6, 7).

Pathways of childbirth pain during first stage of labour.

The uterine body, inferior segment and the upper cervix all have ascendant nerve fibers accompanying sympathetic nerves on the following pathway: uterine plexus, cervical plexus, inferior hypogastric plexus, middle and superior hypogastric

plexus, aortic plexus, sympathetic lumbar plexus, inferior thoracic sympathetic pathway and from this level trough communicating branches of T10, T11, T12, L1 and their posterior roots to the spine and the posterior horn. This is a visceral type innervation (8, 9).

Pain the second stage of labour.

During the expulsion of the foetus the pain originates from the distension and traction of the parietal pelvic peritoneum and uterine ligaments, traction of the bladder, urethra and rectum, stretching and intermittent traction at the level of ligaments and fascia of pelvic musculature, pressure at the level of the nerve endings from lumbar and scral plexus (10, 11).

Pathways of childbirth pain during the second stage of labour.

Having a somatic type of innervation, pain triggers nerve impulses that are conducted trough internal pudendal nerve (S2-S4), posterior cutaneous nerve of thigh (S1-S4), ilioinguinal nerve (L1) and genitofemoral nerve (L1-L2). When these impulses reach the spine, they follow the same pathway and same modulatory action as any other acute pain (5, 12).

The nerve fibers involved with the conduction of this type of pain are high speed conducting nerve fibers, thus the pain is perceived as sharp and localized. This makes it harder to approach when compared with the visceral pain, characteristic to the first stage of labour (13).

Modulation of pain during childbirth.

During dilatation and expulsion of the foetus the uterine contractions and subsequent tissular lesions rise the sensitivity threshold of peripheral and central nervous structures. The peripheral sensitivity lowers its threshold for pain signals at the level of afferent nerve endings and the ongoing activation of the rises the excitability of the spine neurons (14). The mechanism of central sensitivity activation is linked to with the central summation of pain signals induced trough C and Aä nerve fibers which levels up the response capacity of the neurons from the posterior horn (phenomena known as “wind up”). This is further boosted by the release of three modulators at the level of C type nerve endings: glutamate, which acts trough AMPA and NMDA receptors, P substance which acts trough neurokinin 1 receptors and neurokinin A which acts at the level of neurokinin 2 receptors (15).

Tissular damage throughout the labour induce a primary and a secondary hyperalgesic state: primary hyperalgesia occurs at the level of the actual tissular destruction and is the consequence of activation of C and Aä sensitive nerves receptors, and secondary hyperalgesia which appears at a distance from the site of the lesions and is modulated trough central sensitivity which signals pain with the activation of Aâ nerve fibers (16).

As a result the central sensitivity alters the extension and threshold of the peripheral areas initiating the pain signals (17, 18).

The effect of pain during childbirth.

At the level of the spine nerve impulses generated from pain are set to determine a transfer of signals to neurons located in the anterior and antero-lateral horns resulting in a segmental reflex. At the level of the brainstem this signaling results in a suprasegmental reflex and at the level of the cortex, a cortical response (19). At the very end of this pathway the actual perception of the pain occurs with a subsequent psychological reaction (emotion, anxiety, fear, motivation, judgement) and a behavioral response (verbal, somatic) (20).

Effects on the mother.

Vital functions modifications – ventilation.

Pain is a powerful respiratory stimulator (trough the suprasegmental reflex response) and induces an increase of tidal volume, alveolar volume, a lowering of the Pa CO2 and rising pH levels (respiratory alkalosis). Once the uterine contraction stops, without a pain signal, under hypocapnic conditions a transient hypoventilation occurs with the

subsequent fall of Pa O2 by 25 to 30% (21, 22).

Vital functions modifications – cardio-vascular.

Pain and anxiety set a rise of the sympathetic state with the increase of the cardiac output (tachycardia and stroke volume). During labour the cardiac output rises with 50% and with each uterine contraction with further 25-30% (as a result of the additional uterine blood discharged in the systemic circulation after the contraction). Also, the rising levels of peripheric vascular resistance leads to the increase of both systolic and diastolic arterial pressure by 25-30 mmHg (21, 22). During uterine contractions the output of the left ventricle is also augmented. These al all cardio-vascular modifications well tolerated by a healthy woman but harmful to a mother with a underlying cardiac pathology when measures to control the pain are required (21, 22, 23).

Neuroendocrine modifications.

Severe pain and anxiety induces a 3 to 6 time fold rising of adrenaline levels, 2 to 4 times for the noradrenaline levels and 2 to 3 times fold for cortisol levels (17, 24, 25, 26).

Metabolic effects.

During labour a progressive maternal acidotic state is installing which will be transmitted to the fetal compartment from one point to a progressive higher extent. These metabolic modifications are the result of the increasing release of catecholamine agents with lipolysis and the appearance of free fatty acids and lactate (27, 28). The effort of the mother (contraction of uterus and concomitant voluntary and involuntary expulsion effort – the pushing) leads to the increase of metabolic activity and O2 consumption trough sympatheticotonia, the loss of renal bicarbonate (for the compensation of respiratory alkalosis induced by pain) (29, 30).

Gastro-intestinal modifications.

Pain, anxiety and stress lead to increased gastrine levels, inhibits gastro-intestinal motility and postpone the evacuation of the stomach (31).

Psychologic effects.

Severe pain during childbirth can induce negative emotional modifications in the relationship with both the newborn and the father or the idea of a future pregnancy. These modifications occur especially when the mother is convinced before birth, she would have an easy delivery and her expectations are not matched in reality. This emphasizes the role of theoretic, practical and psychological training of women before birth with thorough discussions regarding the management of pain during delivery if necessary (32).

The effects of pain on uterine activity and labour.

Pain and emotional stress modify uterine contractions and the duration of labour by increasing the catecholamine levels. Noradrenaline increases the uterine activity whereas the adrenaline and cortisol have an opposing effect. With severe pain the adrenaline cane rises to extreme levels that are capable of inhibiting the uterine activity. Pain determines the increase of pelviperineal tonus with dynamic modifications and subsequent abnormal progression of labour which further increases the pain (33).

The effects of pain on the foetus

During uterine contraction there is a transient altered transfer to the placenta. Severe hyperventilation and respiratory alkalosis induced by pain worsens these changes by changing the dissociation curve of hemoglobin (decreases the O2 transfer from mother to the foetus) and by inducing maternal hypoxemia in-between uterine contractions (19).

The increase of noradrenaline and cortisol decreases the uterine blood inflow. During a normal labour these modifications that occur at the level of the placenta are well handled by a normal foetus (O2 reserve in fetal circulation, compensatory fetal modifications). If the uterine activity is intense fetal hypoxia, hypercapnia, acidosis will be tolerated only

for a limited time period by a normal foetus. On the other hand, fetal distress can occur in the case of a "fragile" foetus due to the O2 and CO2 altered changes induced by pain at the level of maternal circulation (33, 34).

Conclusion

All these findings supported the research for effective methods for the management of pain during childbirth. Numerous methods and techniques were proposed over time (Lamaze psycho-prophylaxis, hypnosis, acupuncture, systematic administration of opioids, tranquilizers, sedatives, inhalator agents).

In 1880 Klikovich utilized nitrous oxide was first used in Sankt Petersburg for obstetrical analgesia. In 1853 John Snow administers chloroform to Queen Victoria at her 8th delivery. Still, only in 1966 Chunchill – Davidson considers obstetrical analgesia calling it "the Cinderella of anesthesiology". Today this is the queen of obstetrical analgesia – continuous epidural.

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