ABG بند ناف

دکتر علی روشنی تبریزی فوق تخصص نوزادان

اندیکاسیون ها

- Umbilical cord blood gas analysis may be recommended after all births
- where there is suspicion of intrapartum hypoxia ischaemia and is a useful guide to the condition of the fetus at the time of birth.

Cord venous ph is a reflection of placental gas transfer

cord arterial ph indicates fetal response to labour

therefore important to take paired

تنظیم اسید و باز در جنین

▶ The fetus has an intact extracellular buffer system with the car-bonic acid-bicarbonate buffer system serving as the predominant buffer system. For the fetus, the placenta is the organ of respiration and quickly eliminates the excess carbon dioxide generated by the development of fetal metabolic acidosis, provided that placental function, uterine and umbilical blood flows, and mater- nal respiratory status are uncompromised. 13 Intracellular buffering capacity is considerably larger than the extracellular one

despite the fact that the fetus has a significantly smaller intracellular compartment compared to a child or adult.

مقادير نرمال PO2

		UV	UA	5-10 min	20 min	30 min	60 min	5h	24h	2 days	3 days	4 days	5 days	6 days	7 days
Po ₂ (mm Hg)	χ	15.9	27.4	49.6	50.7	54.1	63.3	73.7	72.7	73.8	75.6	73.3	72.1	69.8	73.1
	SD	3.8	5.7	9.9	11.3	11.5	11.3	12.0	9.5	7.7	11.5	9.3	10.9	9.5	9.7
	Range	7	15	33	31	31	38	55	54	62	56	60	56	55	57
		23	40	75	85	85	83	106	95	91	102	93	102	96	94

SD, standard deviation; UA, umbilical artery; UV, umbilical vein; X, sample mean.

مقادير نرمال PCO2

		UV	UA	5-10 min	20 min	30 min	60 min	5 h	24 h	2 days	3 days	4 days	5 days	6 days	7 days
Pco ₂ (mm Hg)	X	49.1	37.8	46.1	40.1	37.7	36.1	35.2	33.4	33.1	33.1	34.3	34.8	34.8	35.9
	SD	5.8	5.6	7.0	6.0	5.7	4.2	3.6	3.1	3.3	3.4	3.8	3.5	3.6	3.1
	Range	35	26	35	31	28	28	29	27	26	26	27	28	28	30
		60	52	65	58	54	45	45	40	43	40	43	41	42	42

SD, standard deviation; UA, umbilical artery; UV, umbilical vein; χ, sample mean.

مقادیر نرمال PH

PIII

		UV	UA	5-10 min	20 min	30 min	60 min	5h	24h	2 days	3 days	4 days	5 days	6 days	7 days
рН	χ	7.320	7.242	7.207	7.263	7.297	7.332	7.339	7.369	7,365	7.364	7.370	7.371	7.369	7.37
	SD	0.055	0.059	0.051	0.040	0.044	0.031	0.028	0.032	0.028	0.027	0.027	0.031	0.032	0.02
	Range	7.178	7.111	7.091	7.180	7.206	7.261	7.256	7.290	7.314	7.304	7.320	7.296	7.321	7.32
		7.414	7.375	7.302	7.330	7.380	7.394	7.389	7.448	7.438	7.419	7.440	7.430	7.423	7.43

SD. standard deviation: UA umbilical artery: UV. umbilical vein: Y. sample mean.

مقادیر نرمال Base Excess

		UV	UA	5-10 min	20 min	30 min	60 min	5h	24h	2 days	3 days	4days	5 days	6 days	7 days
Base excess	χ	-5.5	-7.2	-9.8	-8.8	-7.8	-6.5	-6.3	-5.2	-5.8	-5.9	-5.0	-4.7	-4.7	-3.2
6/10000	SD	1.2	1.7	2.3	1.9	1.7	1.3	1.3	1.1	1.2	1.2	1.1	1.1	1.1	0.6

(Calculated from data in Koch G. Wendel H. Adjustment of arterial blood gases and acid base balance in the normal newborn infant during the first week of life. Biol Negnate.

ABG riemur

- ◄ تعیین وجود اسیدوز یا آلکالوز
 در شریان نافی وجود PH کمتر از ۷/۳۶
 در ورید نافی وجود PHکمتر از ۷/۴۱
- ◄ در صورت وجود اسیدوز قدم بعدی ارزیابی مقدار PCO2,
 کمبود بازبرای تعیین نوع اسیدوز (تنفسی یا متابولیک یا هردو)
 در شریان نافی وجود PCO2 بالاتر از ۴۹ نشانه اسیدوز تنفسی
 در ورید نافی وجود PCO2 بالاتر از ۴۰ نشانه اسیدوز تنفسی
 در شریان نافی وجود کمبود باز بیشتر از ۴/۰۱ نشانه اسیدوز متابولیک
 در ورید نافی وجود کمبود باز بیشتر از نشانه ۲/۷ اسیدوز متابولیک

علل اسیدوز تنفسی جنین

▶ Fetal respiratory acidosis develops when prolonged maternal hypoventilation occurs with maternal asthma, airway obstruction, narcotic overdosing, maternal anesthesia, severe hypo-kalemia, and magnesium sulfate toxicity.

علل اسیدوز متابولیک در جنین

- The most frequent cause of fetal metabolic acidosis is fetal hypoxemia due to abnormalities of uteroplacental function, blood flow, or both.
- Primary maternal hypoxemia or maternal metabolic acidosis secondary to maternal diabetes mellitus, sepsis, or renal tubular abnormalities are unusual causes of fetal metabolic acidosis.

فیزیولوژی ایجاد اسیدوزمتابولیک در جنین

- After 4 to 5 hours of fetal hypoxemia, blood lactate reaches a plateau despite continued anaerobic metabolism ,Thus
- despite ongoing fetal anaerobic metabolism in cases of chronic and severe fetal hypoxemia, the fetal serum lactate level does not rise beyond the point of equilibrium
- When fetal hypoxemia is caused by umbilical cord occlusion, metabolic acidosis develops more rapidly compared with maternal hypoxia-induced fetal hypoxemia

the fetal response to hypoxemia is also altered when hypoxia is caused by decreased uterine blood flow.

In this model, the critical threshold of arterial O2 saturation is lower than observed in the maternal hypoxia or cord occlusion models(%**-); rapid lactate accu mulation and fall in pH only occur when fetal O2 saturation is in the 15% to 20% range.

- Cord compression or prolapse likely to lead to larger differences (venous- arterial pH difference >0.15)
- Venous- arterial differences likely to be smaller if placental perfusion compromised (e.g. abruption, uterine rupture)

علل آلكالوز متابوليك در جنين

Metabolic alkalosis rarely affects the fetus, but it may occur in

women with hyperemesis gravidarum.

علل آلکالوز تنفسی در جنین

Maternal metabolic alkalosis does not affect fetal pH or PaCO2, at least in the short-term.

severe acute maternal hyperventilation results in decreased

umbilical arterial flow and the development of fetal hypoxia and metabolic acidosis.

maternal hyperventilation also results in fetal acidosis, decreased oxygen saturation in the umbilical blood vessels and perinatal depression at birth

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معبار های تشخیصی آسفیکسی

- Perinatal assessment of risk
- Umbilical cord or first blood gas

severe acidosis was defined as pH ≤7.0 or basedeficit ≥16 mmol/L. •

Risk of hypoxic ischaemic encephalopathy (HIE) and poor neurological outcome only significantly increased if cord arterial pH <7.05 (<7.0 in preterm).

- Low Apgar scores
- ▶ If the Apgar score is >6 by 5 minutes, perinatal asphyxia is not likely
- the differential diagnosis for a term newborn with an Apgar score ≤3 for ≥10

minutes includes depression from maternal anesthesia or analgesia, trauma, infection, cardiac or pulmonarydisorders, neuromuscular, and other central nervous system (CNS) disorders or malformations.

- 1. PROLONGED (>1 HOUR) ANTENATAL ACIDOSIS
- 2. FETAL HR < 60 BEATS PER MINUTE
- 3. APGAR SCORE ≤3 AT ≥10 MINUTES
- 4. NEED FOR POSITIVE PRESSURE VENTILATION FOR >1 MINUTE OR FIRST CRY DELAYED >5 MINUTES
- 5. SEIZURES WITHIN 12 TO 24 HOURS OF BIRTH
- 6. BURST SUPPRESSION OR SUPPRESSED BACKGROUND PATTERN ON EEG OR AMPLITUDE-INTEGRATED ELECTROENCEPHALOGRAM