American Academy of Pediatrics

Use and Abuse of the Apgar Score

Committee on Fetus and Newborn, American Academy of Pediatrics, and Committee on Obstetric Practice, American College of Obstetricians and Gynecologists

ABSTRACT. This is a revised statement published jointly with the American College of Obstetricians and Gynecologists that emphasizes the appropriate use of the Apgar Score. The highlights of the statement include: (1) the Apgar Score is useful in assessing the condition of the infant at birth; (2) the Apgar score alone should not be used as evidence that neurologic damage was caused by hypoxia that results in neurologic injury or from inappropriate intrapartum treatment; and (3) an infant who has had "asphyxia" proximate to delivery that is severe enough to result in acute neurologic injury should demonstrate all of the following: (a) profound metabolic or mixed acidemia (pH < 7.00) on an umbilical arterial blood sample, if obtained, (b) an Apgar score of 0 to 3 for longer than 5 minutes, (c) neurologic manifestation, eg, seizure, coma, or hypotonia, and (d) evidence of multiorgan dysfunction.

The Apgar score, devised in 1952 by Virginia Apgar, is a quick method of assessing the clinical status of the newborn infant. Ease of scoring has led to its use in many studies of outcome. However, its misuse has led to an erroneous definition of asphyxia. Intrapartum asphyxia implies fetal hypercarbia and hypoxemia, which if prolonged will result in eventual metabolic acidemia. Because the intrapartum disruption of uterine or fetal blood flow is rarely, if ever, absolute, asphyxia is an imprecise, general term. Terms such as "hypercarbia," "hypoxia," and "metabolic and respiratory or lactic acidemia" are more precise, both for immediate assessment of the newborn and for retrospective assessment of intrapartum management.) Although the Apgar score continues to provide a convenient shorthand for reporting the status of the newborn and the effectiveness of resuscitation, the purpose of this statement is to place the Apgar score in its proper perspective.

The Apgar score comprises five components: heart rate, respiratory effort, muscle tone, reflex irritability, and color, each of which is given a score of 0 through 2 (Table). Reliable Apgar scores require assessment of individual components of the score by trained personnel.

FACTORS THAT MAY AFFECT THE APGAR SCORE

Although rarely stated, it is important to recognize that elements of the Apgar score, such as tone, color, and reflex irritability, are partially dependent on the physiologic maturity of the infant. The healthy premature infant with no evidence of anoxic insult, acidemia, or cerebral depression may thus receive a low score only because of immaturity.

A number of maternal medications and infant conditions may influence Apgar scores, including, but not limited to, neuromuscular or cerebral malformations that may decrease tone and respiratory effort. Cardio-respiratory conditions also may decrease the infant's heart rate, respiration, and tone. Infection may interfere with tone, color, and response to resuscitative efforts. Additional information is required to interpret Apgar scores properly in infants receiving resuscitation. Thus, to equate the presence of a low Apgar score solely with asphyxia or hypoxia represents a misuse of the score.

APGAR SCORE AND SUBSEQUENT DISABILITY

A low 1-minute Apgar score does not correlate with the infant's future outcome. The 5-minute Apgar score, and particularly the change in the score between 1 and 5 minutes, is a useful index of the effectiveness of resuscitation efforts. However, even a 5-minute score of 0 to 3, although possibly a result of hypoxia, is limited as an indicator of the severity of the problem and correlates poorly with future neurologic outcome. An Apgar score of 0 to 3 at 5 minutes is associated with an increased risk of cerebral palsy in full-term infants, but this increase is only from 0.3% to 1.3%. A 5-minute Apgar score of 7 to 10 is considered normal. Scores of 4 through 6 are intermediate and are not markers of high levels of risk of later neurologic dysfunction. As previously mentioned, such scores are affected by physiologic immaturity, medication, the presence of congenital malformations, and other factors.

Because Apgar scores at 1 and 5 minutes correlate poorly with either cause or outcome, the scores alone should not be considered evidence of or a consequence of substantial asphyxia. Therefore, a low 5-minute Apgar score alone does not demonstrate that later development of cerebral palsy was caused by perinatal asphyxia.

Correlation of the Apgar score with future neurologic outcome increases when the score remains 0 to 3 at 10, 15, and 20 minutes but still does not indicate the cause of future disability. The term asphyxia in a clinical context should be reserved to describe a combination of damaging acidemia, hypoxia, and metabolic acidosis. A neonate who has had asphyxia proximate to delivery that is severe enough to result in acute neurologic injury should demonstrate all of the following:
The Apgar score alone cannot establish hypoxia as the cause of cerebral palsy. A full-term infant with an Apgar score of 0 to 3 at 5 minutes whose 10-minute score improved to 4 or higher has a 99% chance of not having cerebral palsy at 7 years of age. Conversely, 75% of children with cerebral palsy had normal Apgar scores at birth.

Cerebral palsy is the only neurologic deficit clearly linked to perinatal asphyxia. Although mental retardation and epilepsy may accompany cerebral palsy, there is no evidence that they are caused by perinatal asphyxia unless cerebral palsy is also present, and even then a relationship is in doubt.

**CONCLUSION**

Apgar scores are useful in assessing the condition of the infant at birth. Their use in other settings, such as collection of a child’s Apgar score at entry to school, is inappropriate. Low Apgar scores may be indicative of a number of maternal and infant factors. Apgar scores alone should not be used as evidence that neurologic damage was caused by hypoxia or inappropriate intrapartum management. In the infant who later is found to have cerebral palsy, low 1- or 5-minute Apgar scores are not sufficient evidence that the damage was caused by hypoxia or inappropriate intrapartum management. Hypoxia as a cause of acute neurologic injury and an adverse neurologic outcome occurs in infants who demonstrate the four perinatal findings listed above and in whom other possible causes of neurologic damage have been excluded. In the absence of such evidence, subsequent neurologic deficiencies cannot be ascribed to perinatal asphyxia or hypoxia.

### REFERENCES

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