The Effect of Head Compression on FHR, Brain Metabolism and Function

LEON I. MANN, MD, FACOG, ANDREW CARMICHAEL and SYBIL DUCHIN

The effect of fetal head compression on heart rate, cerebral metabolism and function was studied in 30 sheep experiments. Cerebral oxygen consumption decreased markedly as cerebral blood flow was significantly impaired by the increase in intracerebral pressure and vascular resistance. A fetal bradycardia or tachycardia occurred in 76% of the experiments. The fetal EEG showed a drop-out of faster frequencies, slowing and a decrease in amplitude prior to the onset of an isoelectric (flat) stage. All parameters recovered rapidly after the episode of cerebral ischemia caused by head compression.

THE FORCE OR PRESSURE exerted on the fetal head during labor and during the application and delivery of the fetal head by forceps has been studied in human beings by several investigators. Schwarcz *et al*[1] used flat pressure receptors introduced between the uterine wall and fetal head to evaluate the pressure exerted by uterine contractions on the head. Receptors placed at the level of the equator (plane of largest diameter) of the fetal head recorded pressures with a uterine contraction that were up to 2.5 times higher than amniotic pressure. As the distance from the equator increased, pressure decreased and equalled amniotic pressure. Recorded at the equatorial zone, pressure increased with rupture of the membranes and descent of the fetal head. The difference between amniotic fluid pressure and the higher pressures recorded at the equatorial zone represent pressure due to the resistance offered by muscular and bony pelvic structures. If the area upon which the pressure is exerted is unchanged, then the force (Force = Pressure × Area; kilograms or pounds) is similarly increased.

Fleming[2] and associates modified a forceps, by adding strain gauges, to study traction and compressive forces exerted on the fetal head during forceps delivery. Ullery *et al*,[3] Kelly[4] and Pearse[5,6] reported an average instrumental traction of approximately 30 to 50 pounds and an average head compression of 5 to 6 pounds (2 to 3 kg) during forceps delivery. The force exerted on the fetal head with forceps application and delivery is quite similar to that exerted by a contraction with an amniotic fluid pressure of greater than 40 to 50 mm Hg.

The effect of both uterine contractions and forceps application and delivery on fetal heart rate and EEG has been evaluated in the human being. Althabe *et al*[7] showed that the incidence of transient falls in fetal heart rate with uterine contractions (dip I) increased .50 to 60% when the membranes were ruptured, the cervix was dilated 6 to

From the Department of Obstetrics and Gynecology, Cornell University Medical College, 1300 York Avenue, New York, NY 10021.

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Address reprint requests to: Leon I. Mann, MD, Cornell Medical Center, 525 East 68th St, New York, NY 10021.

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10 cm and the fetal head was at station 0 to +4. During early labor, when the membranes were intact, the cervix dilated only 2 to 4 cm and the fetal head at −4 to −2 station, the incidence of dip's I was 2 to 15%. Kelly[4] reported that fetal heart rate slowed during traction in 84% of the fetuses delivered by forceps.

Garcia-Austt[8] reported irregular, high amplitude slow waves of 100 to 200 μV with a frequency of 0.5 to 1/sec in the EEG during contraction. During the interval between contractions, frequency was 2 to 3/sec and amplitude 50 to 100 μV. On occasion, random and/or rhythmic epileptiform activity, which continued into the newborn period, was associated with uterine contractions. Cacava[9] recorded neonatal EEG's after forceps delivery and noted frequent peak potentials localized in any region, intensified Δ waves (0.5 to 3/sec), their focal origin and occasionally depressed electric activity. These changes occurred in 45 of 75 cases in which forceps had been applied, in contrast to only 5 of 50 normal, spontaneously delivered newborns. Head compression, whether it followed a uterine contraction or forceps application and delivery caused similar changes in the EEG.

It has been suggested that the changes observed in fetal heart rate and EEG during head compression are the result of transient episodes of cerebral ischemia due to intracranial hypertension or deformation of the fetal head. Vagal stimulation could follow CNS hypoxia and/or stimulation of cephalic mechanoreceptors and explain the transient fall in heart rate.

The experiments that are reported herein were designed to study this hypothesis in the laboratory animal. The results indicate that the hypothesis is correct, with minor modifications. Preliminary observations have been reported previously.[10]

MATERIALS AND METHODS

Thirty experiments were conducted on 15 fetuses of mixed breed ewes. Gestational ages varied from 101 to 147 days (term approximately 145 days).

The ewes were prepared for surgery by withholding food and water for at least 12 hours. Anesthesia was induced and maintained with halothane (Fluothane®), 0.6 to 1.4%, delivered through a cuffed endotracheal tube with the aid of a negative-positive respirator (Bird) and the usual anesthetic apparatus.

Extradural electroencephalographic (EEG) electrodes, "T" catheters in the carotid and jugular vessels and a perivascular ultrasonic flow probe were placed on the carotid artery by methods described previously in detail.[11,12] Bipolar EEG potentials from each hemisphere, pressure in the carotid and jugular vessels and output from the flow transducer were recorded by direct write-out on a dynograph. The output of the flow transducer was calibrated in vivo for volume flow for each experiment.

Blood gases, pH and substrate determinations were performed as described previously.

A pediatric rib retractor was modified so that the distal end of each arm was attached to a 1½ inch-square metal plate. Force was calibrated electronically by a strain gauge placed in one arm of the device, the output of which was recorded continuously on the dynograph. The steel bar connecting the arms of the device was measured in centimeters so that deformation changes could be recorded. The force that would be exerted on the fetal head during a contraction can be estimated by determining the pressure within the amniotic cavity and by considering mean cephalic diameters. The force (kilograms or pounds) is equal to the product of pressure and area. In the human, the occipitofrontal diameter (mean: 11.8 cm) identifies the plane of largest dimensions (equatorial plane) which would have a maximum area of 108.5 sq cm, if assumed to be circular. A pressure of 50 mm Hg

would result in a maximum force of 16.3 pounds (7.4 kg) at that plane (1 mm Hg = 1,333 dynes/sq cm; 1 dyne = 2.25 × 10^{-6} pounds; 1 kg = 2.204 pounds). The force would decrease as either the plane of the diameter decreased in area or the pressure decreased so that it was difficult to know the exact magnitude of the force exerted on the total head. The force, however, will be distributed equally as long as the membranes remain intact and the fetal head unengaged.

After a baseline period of 15 to 30 minutes, a baseline blood sample was drawn and a force varying from 2–15 kg was applied over the temporal-parietal area of the fetal head. The force was held constant, as determined by the dynograph recording, until the onset of the isoelectric (flat) stage of the EEG. At this time, a second blood sample was drawn from the carotid and jugular vessels. The force was then released and further blood samples obtained at various intervals. Head compression, with different amounts of force, was applied one to three times in each of the 15 fetal preparations. The physiologic and biochemical parameters returned to control levels before the force was reapplied. In several other experiments the EEG was unaffected by the force applied, but these experiments were not included in the analysis.

In three experiments, intracerebral pressure was recorded by inserting a needle into the brain through a parasaggital burr hole and connecting the fluid-filled catheter to the pressure transducer (Statham).

Statistical significance, evaluated by comparing group means by Student's *t* test, is presented in terms of *P* values.

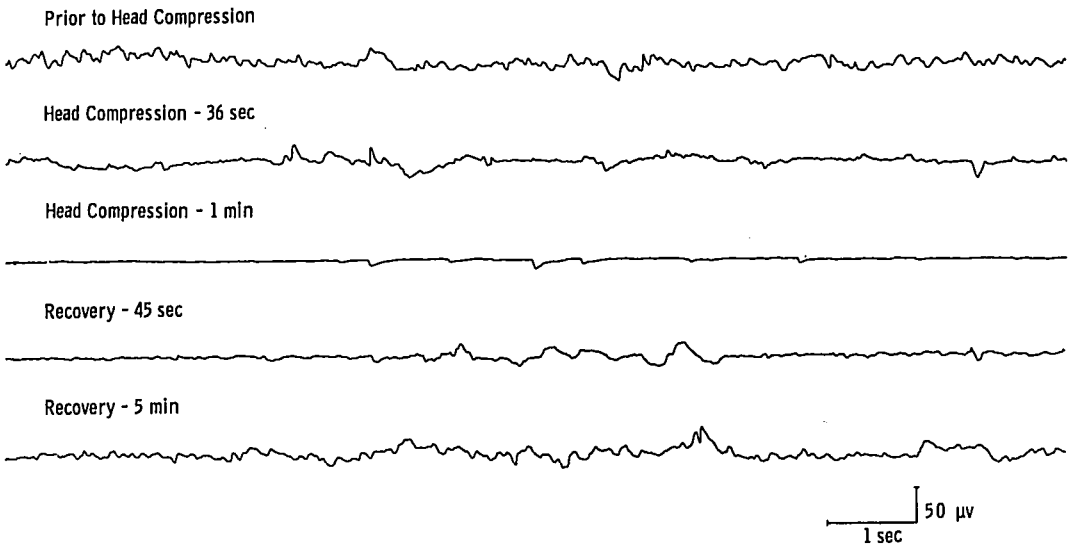
RESULTS

The physiologic and metabolic observations during the baseline period and at the onset of the isoelectric stage of the EEG during head compression in the 30 experiments are presented as mean values ± standard error in Table 1. In Figure 1, the physiologic and metabolic observations are shown along with representative tracings of

TABLE 1. PHYSIOLOGIC AND METABOLIC OBSERVATIONS AFTER HEAD COMPRESSION IN 30 SHEEP EXPERIMENTS

Observations	*Baseline*	*Compression*	*P*
Oxygen tension (mm Hg)	17.6 ±1.6	17.7 ±1.6	NS
pH	7.256 ±0.028	7.212 ±0.032	NS
Cardiovascular			
Heart rate (beats/min)	148.0 ±5.6	154.3 ±8.1	NS
Perfusion pressure (mm Hg)	31.7 ±0.8	39.9 ±6.8	<0.0005
Resistance (mm/liter/min)	544.2 ±57.1	1561.8 ±233.2	<0.0005
Blood Flow (ml/100 g/min)	157.7 ±14.8	84.4 ±9.6	<0.0005
Metabolic			
Oxygen consumption (ml/100 g/min)	3.8 ±0.6	1.4 ±0.4	<0.0025
Glucose metabolism (mg/100 g/min)	6.0 ±4.2	1.2 ±1.4	NS
Lactate metabolism (mg/100 g/min)	2.3 ±3.5	1.7 ±1.9	NS
Pyruvate metabolism (mg/100 g/min)	0.6 ±0.3	0.3 ±0.2	NS

NS = Not significant



	Heart Rate beats/min	pO_2 mm Hg	pH	Perfusion Pressure mm Hg	Resistance mm/L/min	Blood Flow ml/100g/min	Q O2 ml/100g/min
Prior to Head Compression	130	18	7.258	39	539	237	3.32
Head Compression - 1 min	128	17	7.255	48	5574	28	0.76

Fig 1. EEG, physiologic and cerebral metabolic observations during the application of a compressive force of 7 kg to the fetal head. EEG isoelectric (flat) for 45 seconds. Gestational age of the fetus 128 days.

the EEG during head compression in a single experiment.

Electroencephalograph

The changes observed in the EEG consisted of a rapid drop-out of faster frequencies, a decrease in amplitude and the appearance of an isoelectric (flat) EEG (Figure 1). The EEG became isoelectric in less than 3 minutes in all experiments and recovered after head compression was relieved, at which time the slow waves reappeared, amplitude increased and waves of faster frequencies were added (Figure 1).

The EEG of the younger fetuses (101 to 120 days) generally became flat with less of a cephalic compressive force (2 to 8 kg) than was required in more mature fetuses (120 to 147 days; 7 to 15 kg). The same amount of force (6 to 10 kg) resulted in the more rapid onset of the isoelectric EEG in the younger fetuses. These force-time relations are probably due to fusion of the cranial bones with fetal development in this species and do not reflect different gestational sensitivities to head compression.

Oxygen Tension and pH

There was no change in oxygen tension and pH in the carotid artery as a result of head compression. An oxygen tension of approximately 18 mm Hg was similar to values obtained previously from this laboratory.

Cardiovascular

The mean value for fetal heart rate did not change significantly as a result of head compression. However, heart rate decreased in 11 experiments, increased in 12 and remained unchanged in seven. The most significant alterations in heart rate occurred at >160 beats/min.

After head compression, blood flow decreased significantly from 157.7 ± 14.8 to 84.4 ± 9.6 ml/100 g/min. As shown in Figure 1, the percentage fall from the baseline was even more severe in specific experiments. The decrease in blood flow was due to a dramatic increase in cerebral vascular resistance. Perfusion pressure increased significantly after the head compression.

Intracerebral Pressure

In each of the three experiments on which intracerebral pressure was recorded, it increased rapidly to values well above pressure in the carotid artery. In the experiment shown in detail in Figure 1, intracerebral pressure was <10 mm Hg before head compression, rose to 78 mm Hg 36 seconds after head compression and was 109 mm Hg at 1 minute when the EEG was flat. Intracerebral pressure fell rapidly as soon as head compression was relieved.

Cerebral Metabolic Rate

As a result of head compression, the cerebral metabolic rate for oxygen decreased significantly from 3.8 ± 0.6 to 1.4 ± 0.4 ml/100 g/min. There was no significant change in the metabolic rate for glucose, lactate and pyruvate during the head compression.

Recovery Period

As soon as the compressive force was released, resistance decreased rapidly, perfusion pressure decreased and blood flow increased immediately, often to values above the baseline. Cerebral oxygen consumption recovered instantly in those few cases in which it could be measured. The EEG was indistinguishable from the baseline record in less than 10 minutes in all 30 experiments. All fetuses were alive for at least 30 minutes after the episode of head compression. Repetitive head compressions of a force significant enough to cause changes in the EEG seemed to be cumulative in their effect on the rapidity of onset of the isoelectric stage of the EEG. In one experiment, the EEG failed to recover after several head compressions and remained flat while cerebral blood flow was intermittently within normal levels.

DISCUSSION

Compression of the fetal head by an externally applied force caused severe cerebral ischemia due to a marked reduction in cerebral blood flow. The resistance to blood flow increased as intracerebral pressure was increased by vascular narrowing and collapse. The obstruction to flow prevented well-oxygenated blood in the carotid artery from reaching the fetal brain. As soon as the compressive force was released, blood flow returned rapidly and once again the brain was well oxygenated.

Observations by Garcia-Austt[8] on the human fetal EEG during uterine contractions and those by Cacava[9] on the human neonatal EEG after forceps delivery were analogous to changes in the EEG noted during head compression in the sheep fetus. These investigators did not record flat tracings, however, as the force that we applied was probably of a greater magnitude than that occurring during either clinical condition. The EEG changes were also similar to the fetal EEG records during hypoxic hypoxia that we have reported previously.[13] The appearance of slow waves with a decrease in amplitude and dropping out of faster frequencies would seem to be diagnostic of fetal cerebral hypoxia. Caution must be exercised, however, in interpreting this from a single tracing of the human fetal EEG where exact placement of the EEG electrodes is unknown. Continuous EEG recordings are necessary for the proper evaluation of fetal cerebral function.

Most investigators, consider head compression, rather than hypoxia, the cause of fetal heart rate slowing during the contraction (dip I) since fetal bradycardia has been illicited by abdominal-vaginal compression of the fetal head or compression of the head

of the neonate.[14] In 76% of the experiments, a change in fetal heart rate was observed during head compression. In these experiments, head compression was studied independently of a uterine contraction, thus avoiding the mild fetal hypoxia that occurs when a uterine contraction occludes uterine venous outflow. Preliminary experiments[10] designed to study the effect of uterine contractions on fetal brain function and metabolism revealed a fetal bradycardia in all cases in which the intensity of the contractions was greater than 50 to 60 mm Hg. The oxygen content of the fetal circulation decreased during the contraction so that both fetal hypoxia and head compression were acting simultaneously. Under these conditions, maximal vagal stimulation could be anticipated from both peripheral chemoreceptor and central regulatory activation. While the exact etiology of the dip I remains unsettled, changes in fetal heart rate would be expected during uterine contractions.

The significant question raised by the present experiments and clinical observations of others is whether head compression during labor and delivery has a detrimental effect on eventual motor and mental functioning of the child. Mc Intire and Pearse[15] found no difference in the incidence of neurologic developmental abnormalities at 2 to 3 years of age when a group of infants delivered with "high traction" forceps (traction greater than 60 pounds) were compared with a group delivered with traction of less than 42 pounds. However, it has been suggested that labor prolonged beyond 20 hours is associated with a higher incidence of mental and motor abnormalities of childhood. As the large majority of these cases would represent instances of cephalopelvic disproportion, prolonged head compression must be considered a causative factor. More precise identification and long-term followup of specific cases of prolonged head compression must be evaluated before conclusive comments can be made. Hopefully such studies will be forthcoming as investigators direct their attention to the relationship between chronic morbidity and obstetric factors.

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