may have compressed the plexus at the root or trunk level (fig. 3). After the chest tube was repositioned, by withdrawing it approximately 5 cm, the pain was immediately relieved. Partial ulnar nerve injury persisted for more than 3 weeks, ruling out neuropraxia. Also considered in our differential diagnosis was the adequate placement of the thoracostomy tube when the patient was in the left lateral decubitus; however, when the patient was placed supine, the tube may have further compressed the neural bundle. We propose that the mechanism of injury was secondary to the chest tube placement rather than patient positioning. As noted by Kroll et al.,12 the exact mechanism of nerve injury is often unclear; however, since anesthesiologists share in the task of positioning, any problem with patient care in associated brachial plexus palsy should be a joint postoperative effort.

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Response of Fetal Heart Rate to Maternal Administration of Esmolol

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Increases in blood pressure (BP) in the patient with an intracranial arteriovenous malformation (AVM) may precipitate intracranial hemorrhage. Esmolol, an ultrashort-acting β-adrenergic blocking agent, has been shown to effectively blunt the increase in systemic BP associated with tracheal intubation and emergence from anesthesia after intracranial surgery.1,2

The effect of esmolol on fetal heart rate (FHR) after maternal administration has not been reported previously. We describe the use of esmolol in a 22-week pregnant woman undergoing resection of a cerebellar AVM and its effect on FHR.

CASE REPORT
A 31-yr-old woman (gravida 2, para 1) had an intrauterine pregnancy at 22 weeks complicated by a subarachnoid hemorrhage. The patient was scheduled for a suboccipital craniectomy for excision of a cerebellar AVM. By ultrasound, the weight of the fetus was estimated to be 350 g.

Maternal BP on the ward prior to surgery ranged between 90/50 and 110/65 mmHg. The patient arrived in the operating room oriented without neurologic deficit. The patient was monitored with electrocardiogram, BP cuff, pulse oximeter, indwelling radial artery catheter, and mass spectrometer. FHR was monitored continuously intraoperatively and for the first 24 h postoperatively using a Hewlett-Packard 8041A FHR monitor. It is our practice to blunt the hemodynamic response to tracheal intubation and to treat emergence hypertension in the patient with an intracranial AVM by using short-acting agents such as esmolol and nitroprusside. Because the fetal response to esmolol was unknown, we elected to administer esmolol prior to inducing anesthesia to determine its effect on FHR in this patient. The patient was positioned supine with left uterine displacement. Prior to the administration of any anesthetic agents, maternal BP was 142/62 mmHg and heart rate (HR) was 94 beats per min. FHR ranged between 139–144 beats per min, and variability was present (fig. 1, awake control 1).

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In response to increasing maternal BP, three iv bolus doses of esmolol, 500 μg/kg each, were administered over the next 4 min along with a continuous infusion of esmolol 150 μg·kg⁻¹·min⁻¹, and the nitroprouside infusion was increased to 1.8 μg·kg⁻¹·min⁻¹. Approximately 15 min after instituting esmolol therapy, FHR had decreased only slightly, from approximately 120 to 116 beats per min (fig. 2, anesthetized esmolol 1). After an additional 500 μg/kg bolus of esmolol and increase in esmolol and nitroprousid infusion rates to 200 and 2.5 μg·kg⁻¹·min⁻¹, respectively, FHR remained essentially unchanged (fig. 2, anesthetized esmolol 2).

The patient emerged from general anesthesia and the trachea was extubated in the operating room. Six minutes after emergence and extubation, the nitroprousid infusion was discontinued, hydralazine 20 mg iv was administered, and the esmolol infusion maintained at 200 μg·kg⁻¹·min⁻¹. FHR had increased slightly, to 120–122 beats per min at this time (fig. 2, postemergence).

The patient received morphine sulfate 4 mg iv soon after arrival in the recovery room for control of pain. Approximately 1 h after maternal emergence from anesthesia, FHR had gradually increased to 180–192 beats per min, and maternal BP remained controlled at 125/57 mmHg (fig. 2, recovery 1). The esmolol infusion was discontinued several minutes later, and a second dose of hydralazine, 20 mg iv, was administered. FHR increased to 132–137 beats per min after discontinuation of the esmolol infusion (fig. 2, recovery 2).

During the 37th week of pregnancy, the patient underwent an emergency cesarean section because of uterine rupture and fetal distress. A 2,880-g baby boy was delivered with 1- and 5-min Apgar scores of 2 and 7, respectively. Both mother and her son were well at last follow-up, 9 months later.

**DISCUSSION**

The effect of esmolol on FHR in humans has not been previously reported for its administration to patients with normal uteroplacental circulation. However, the effect of esmolol on fetal hemodynamics has been investigated in chronic sheep models by several investigators. Ostein and co-workers examined the transcplanetal passage and hemodynamic changes after administration of esmolol to gravid ewes. After an infusion of esmolol 500 μg·kg⁻¹·min⁻¹ for 4 min and then 300 μg·kg⁻¹·min⁻¹ for 6 min, FHR and BP decreased maximally by 12 and 7%, respectively, while fetal acid–base parameters remained unaffected. The maximal reduction in maternal
mean arterial BP was 7%. At the completion of the infu-
sion, the fetal-to-maternal blood concentration of es-
molol was 0.13, and at 10 min after completion of the
infusion, esmolol was undetectable in the fetus. FHR was
not significantly different from its preinfusion control
value at 15 min after the discontinuation of the esmolol
infusion. The authors concluded that transplacental pas-
sage of esmolol was relatively small and that elimination
from the fetus was rapid.3

In chronically instrumented pregnant ewes, Eisenach
and Castro observed a dose-dependent decrease in ma-
ternal BP and FHR after 15-min stepped infusions of es-
molol (4–200 μg·kg⁻¹·min⁻¹).4 A maximal decrease in
FHR of 27% and decrease in fetal arterial oxygen tension
(PaO₂) from 18 to 14 mmHg and fetal pH from 7.37 to
7.51 occurred after the 200-μg·kg⁻¹·min⁻¹ infusion of
esmolol. The maximum decrease in maternal mean ar-
terial BP was 22% at the 200-μg·kg⁻¹·min⁻¹ infusion
rate. In contrast to Ostman’s study, FHR remained below
control levels 30 minutes after termination of the esmolol
infusion.

We had the opportunity to observe the effect of esmolol
on FHR when administered to our patient under several
circumstances. Prior to the administration of any anes-
thetic agents, FHR decreased less than 10 beats per min,
or less than 7%, in response to the administration of four
bolus doses of esmolol, 500 μg/kg each, and a stepwise
increase in esmolol infusion from 50 to 200
μg·kg⁻¹·min⁻¹ over the course of 10 min. Although ma-
ternal HR decreased from 94 to 79 beats per min after
the administration of esmolol, maternal mean arterial BP
was changed only minimally. FHR returned to near-con-
trol values within approximately 11 min after the termi-
nation of the esmolol infusion. This response is consistent
with the animal data of Ostman and co-workers, who ob-
served a similar reduction in FHR after a 10-min infusion
of esmolol.3

During the induction of anesthesia, it is difficult to sepa-
rate the effect of thiopental from that of esmolol on FHR.
As seen in figure 1, there was a progressive reduction in
FHR from approximately 140 to 120 beats per min after
the administration of thiopental and esmolol (postinduc-
tion esmolol 2 vs. awake control 2). Thus, although there
was a reduction in FHR, there was no evidence of severe
fetal bradycardia after the administration of esmolol with
thiopental for induction of anesthesia.

Based on their observations, Eisenach and Castro have
suggested that a significant and prolonged reduction in
FHR may occur with prolonged infusions of esmolol.4
During emergence and recovery from anesthesia, our pa-
tient received four bolus doses of esmolol, 500 μg/kg
each, in addition to infusion of esmolol at 200
μg·kg⁻¹·min⁻¹ for more than 1 h. We observed a small
reduction in FHR, less than 5 beats per min, after insti-
tution of the esmolol infusion (fig. 2, anesthetized esmolol
2 vs. anesthetized control). Despite the duration of the
esmolol infusion, we observed no further decrease in
FHR. However, undoubtedly there was a reduction in
the depth of fetal anesthesia between the time that the
esmolol infusion was initiated and terminated.

Compared to the longer-acting β-adrrenergic antago-
nists, advantages to the use of esmolol are its much shorter
duration of action and a lower lipid solubility,5 which may
result in reduced placental transfer. It has been suggested
that maternal administration of β-adrrenergic antagonists
could, in theory, produce a significant reduction in FHR
if sensitivity to β blockade is increased because of an in-
mature fetal adrenergic system.3 It must be noted that
although we did not observe a FHR tracing suggestive of
fetal distress after esmolol administration, we were unable
to assess changes in fetal oxygenation and acid–base status.
For this reason as well as the anecdotal nature of this
report, the current case does not establish the safety of
this drug in humans.

In conclusion, after the maternal administration of es-
molol in bolus doses of up to 2 mg/kg and by continuous
infusion of up to 200 μg·kg⁻¹·min⁻¹ in a patient in the
second trimester of pregnancy, esmolol produced small
decreases in FHR in a 350-g preterm fetus. Esmolol may
be an appropriate adjunctive drug in the hemodynamic
management of the pregnant patient with normal uter-
oplacental circulation. The response observed in this pre-
term fetus may well be different than that observed in
the term fetus. Until additional data on esmolol in humans
become available, it seems prudent to monitor FHR in
pregnant patients receiving esmolol.

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