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Blunt abdominal trauma in pregnancy

G.C.E. STUART, MD; P.G.R. HARDING, MD, M SC, FRCS[C]; E.M. DAVIES, MD, FRCP[C]

Two cases are presented in which the effects of blunt trauma to a pregnant woman's abdomen were apparently minor but resulted in fetal death. Blunt trauma may result in serious injury to the fetus or the placenta. Three-point restraint systems should be worn by pregnant women travelling in automobiles to minimize the risks to mother and fetus. Awareness of the potential for injury in these circumstances is essential to reduce the risks to the fetus.

On rapporte deux cas où les effets de contusions de l'abdomen d'une femme enceinte, dont les conséquences apparaissaient mineures, ont résulté en la mort du fœtus. Les contusions peut entraîner une atteinte sérieuse du fœtus ou du placenta. Les femmes enceintes qui voyagent en automobile devraient porter un système de support en trois endroits afin de minimiser les risques à la mère et au fœtus. Être conscient des possibilités de blessures dans ces circonstances est essentiel à la réduction des risques pour le fœtus.

This article documents two fetal deaths following apparently minor blunt trauma to the maternal abdomen that caused no significant external trauma. The fetuses bled to death in utero because of a tear in the fetal surface of the placenta. The effects of blunt trauma in automobile accidents can be minimized by the use of three-point restraint systems and by prompt and appropriate management, which can only be carried out if the potential for significant fetal and placental injury in such accidents is recognized.

Case reports

Case 1

A 25-year-old multiparous woman was involved in a high-speed head-on motor vehicle accident while driving and wearing a lap-belt restraint. She was unable to remember details of the mechanisms of injury, but the steering wheel had remained intact. She was 34 weeks pregnant according to her menstrual history and prior clinical assessment. Normal fetal movement

had been present on the morning of the accident. The woman had remained within the vehicle but had been unconscious for approximately 20 minutes. She arrived in the emergency room complaining of low lumbosacral pain, severe headache and pain across the anterior chest.

Her vital signs were stable. There was extensive bruising across the anterior chest. The abdomen was soft and not visibly bruised, but the uterus was contracting every 2 to 3 minutes. The fetus was in an unengaged vertex presentation. The fetal heart was not heard with the fetoscope or Doppler ultrasonography. There was a small amount of bright red vaginal bleeding, and the thick cervix was dilated to 1 to 2 cm.

An intravenous infusion was established through a large-bore needle, and a Foley catheter was inserted into the bladder. Chest roentgenography showed fractures of the third, fifth and sixth ribs on the left side. Real-time ultrasonography showed a fetus whose development was compatible with 36 weeks' gestation but there was no evidence of fetal heart activity. The placenta was noted to be implanted posteriorly in the fundus.

Closed head injury, possible myocardial contusion (later confirmed by elevation of cardiac enzyme levels), fractures of the ribs and fetal death secondary to the trauma were diagnosed. The patient, in labour, was transferred to the delivery room for observation of the pattern of labour and monitoring of her vital signs.

Twelve hours later the patient's clinical and neurologic status was stable but the pattern of labour had become more desultory. She was given 50 mg of meperidine subcutaneously because of the significant low lumbosacral pain. The hemoglobin level was now 9.9 g/dl, although at the time of admission it had been 11.3 g/dl. Serial coagulograms showed evidence of mild disseminated intravascular coagulopathy.

The pattern of labour then improved. When the cervix was dilated 7 to 8 cm the membranes were ruptured and a large amount of grossly bloody amniotic fluid was obtained. Eighteen hours after the patient's admission a 2405-g stillborn boy was spontaneously delivered. The placenta was also spontaneously delivered. Palpation of the uterine cavity revealed no tears and confirmed the posterior site of implanta-

From the departments of obstetrics and gynecology and of pathology, University of Western Ontario, London

Correspondence to: Dr. P.G.R. Harding, St. Joseph's Hospital, 268 Grosvenor St., Ste. 353, London, Ont. N6A 4V2

tion of the placenta. The infant's development was compatible with 35 weeks' gestation; there was no evidence of external trauma, but the infant was extremely pale. Autopsy showed only anoxic petechial hemorrhages and congestion of the viscera. The placenta weighed 434 g. Approximately 3 cm from the insertion of the umbilical cord was an elliptical laceration of the placenta through both the fetal and the maternal surfaces; it measured 6.5×2.8 cm (Fig. 1). The cotyledons were intact, but there were masses of clot adherent to the fetal surface. Microscopically the placenta and the umbilical cord were normal.

Post partum the patient did well and her coagulogram returned to normal. Roentgenograms of the spine were normal. She was discharged home on the fourth postpartum day.

Case 2

A 31-year-old multiparous woman was involved in a motor vehicle accident when the car she was driving left the road, travelling approximately 65 km/h, and struck a tree. The patient was not wearing a seat belt and crushed the steering wheel on impact. She remained within the vehicle and did not lose consciousness. She was 28 weeks pregnant according to a reliable menstrual history. She presented to the emergency room approximately 15 minutes after the accident.

Her blood pressure and pulse were stable, and there were no neurologic problems. Minor bruises of the face and chest, several abrasions and bruises of the anterior and lateral abdominal wall, and a fractured second metacarpal were noted. The abdomen and the uterus were soft, and the uterus was not irritable. The fetus was in an unengaged vertex presentation, and the uterine fundus was at the expected height. There was no vaginal bleeding. External monitor tracing showed a fetal heart rate of approximately 180 beats/min. Once the fetal heart was heard the monitor was removed and the fractured metacarpal splinted.

An intravenous infusion was started and the patient's status appeared stable. After about 60 minutes

the patient was transferred to the antenatal ward, where real-time ultrasonography was performed. The scan showed no fetal heart valve activity, and intra-uterine fetal death was diagnosed.

She was kept on the ward for observation. Over the next 72 hours there was no uterine activity, vaginal bleeding or loss of amniotic fluid. The patient's hemoglobin level and coagulogram were determined serially and remained stable. On the fourth hospital day uterine cramping developed and bloody vaginal fluid began leaking. Twelve hours later a 1430-g stillborn boy was spontaneously delivered, as was the placenta. Palpation of the uterine cavity revealed no tears. The placenta weighed 322 g and showed two irregular lacerations 4 cm long near the lateral margin (Fig. 2). The umbilical cord was inserted centrally, and both the placenta and the umbilical cord were microscopically normal. The infant was extremely pale and showed no evidence of external injury.

The patient did well post partum and was discharged home on the sixth hospital day.

Discussion

Value of restraints

Seat belts decrease the frequency of maternal injury and death in motor vehicle accidents by preventing ejection.¹ However, the type of restraint system makes a great difference to the frequency of uterine rupture and of fetal death. The use of a lap belt alone may allow enough forward flexion and subsequent uterine compression to rupture the uterus. If the lap belt is worn too high the force of the impact is transmitted directly to the uterus and likewise may rupture it.²

The outcome for the fetus is improved by the use of shoulder restraints. Crosby, King and Stout,³ in a study of 22 baboons in the third trimester of pregnancy, used an impact sled with a decelerative force of 24.5 to $29.0 \times g$ to compare the effectiveness of lap belts and shoulder harnesses. They found a significant difference ($P < 0.05$) in the fetal death rates,

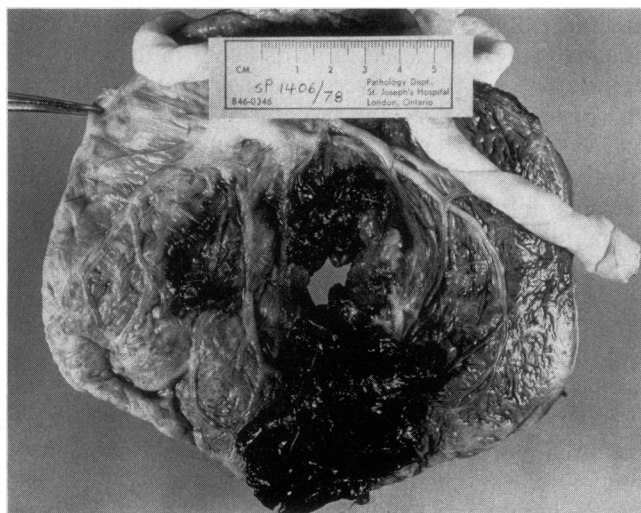


FIG. 1—Fetal surface of placenta, showing laceration through fetal and maternal surfaces, with clot adherent to lower edge.

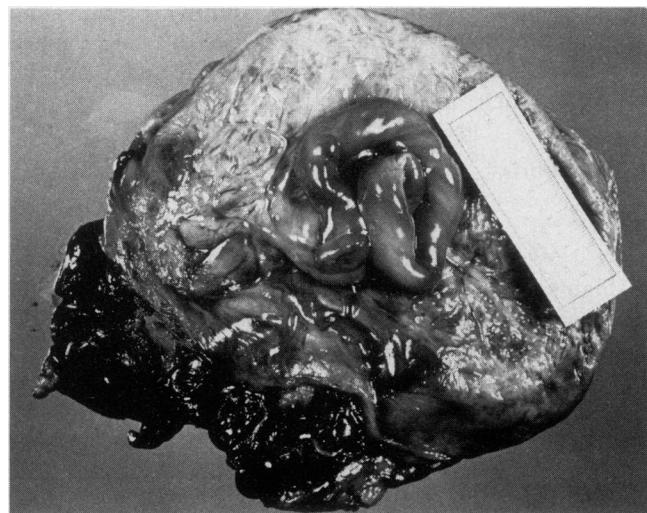


FIG. 2—Fetal surface of placenta, showing lacerations along lateral margin, with adherent clot.

which were 8% when a shoulder harness was used and 50% when a lap belt was used. There were no maternal deaths or instances of uterine rupture. Those investigators suggested that the improvement in fetal survival with the use of a shoulder harness was due to the greater surface area over which the decelerative force was dissipated as well as the prevention of forward flexion of the mother. Individual case reports by Matthews² and by Connor and Curran⁴ also support the use of shoulder restraints with a lap belt. In the general population a three-point restraint system is now considered preferable to a lap belt alone.⁵

In the first case we described, a shoulder harness might have restricted forward movement of the mother's abdomen and thus lessened the force of impact with the steering wheel or dashboard. The second patient was not wearing any restraint; a shoulder harness with a lap belt would have significantly cushioned her forward movement and decreased the force of impact.

Mechanisms of injury

Direct injury to the fetus as a result of blunt trauma to the mother's abdomen is not as common as might be thought because the amniotic fluid serves as a natural buffer. The fetuses in our two cases were free of internal or external evidence of trauma. The relative frequency of fetal injury following trauma is not known, but the most common fetal injuries are skull fracture and intracranial hemorrhage. Isolated fractures of the mandible, the clavicle, the vertebrae and all the long bones have been reported.⁶ In these direct injuries either the maternal abdominal wall is struck by a blunt instrument or the maternal abdomen strikes the car's dashboard, steering wheel or other area. Such injuries may not be diagnosed at the time of the accident, and the pregnancy may well continue to term in the absence of concomitant placental or uterine injuries. Intrauterine fetal fractures may spontaneously heal in utero, as evidenced by callus formation at the fracture site.⁶

Indirect injury of the fetal viscera — that is, injury in the absence of external evidence — has been reported, but one can only speculate on the mechanism of injury. Connor and Curran⁴ reported a case in which the fetus suffered hepatic, renal and adrenal contusions and hemorrhage without showing external evidence of trauma. They suggested that the injury was due to rapid compression and impact of the organs during deceleration, but whether it resulted from a contracoup effect within their attachments or was secondary to a shearing force within the organs could only be conjectured.

Placental abruption secondary to trauma is uncommon, as Buchsbaum⁶ has pointed out. Traumatic laceration of the placenta following blunt abdominal trauma is even more infrequent. However, two cases resulting in injuries to the placenta similar to those we have reported have been described in the literature. In 1942 VanSante⁷ reported a case in which blunt trauma to the maternal abdomen resulting from a fall off a stepladder was followed by spontaneous

delivery of a stillborn infant and a placenta showing a clean laceration through both the fetal and the maternal surface at the area of insertion of the umbilical cord. In 1969 Peyser and Toaff⁸ reported a similar case following a motor vehicle accident in which a radial tear involved the whole thickness of the placenta; the fetus subsequently bled to death in utero. Spontaneous laceration of a placental vein resulting in intra-amniotic hemorrhage has also been described.⁹ The uterus, umbilical cord and fetus were completely intact in all of these cases and in the two we have reported.

The placental injury may be mediated by either a contracoup or a direct force. In our first case the placenta was implanted posteriorly in the uterus, and a contracoup mechanism similar to that in closed head injuries probably occurred. Although the initial decelerative force was applied to the anterior abdominal and uterine walls, the incompressible amniotic fluid immediately anterior to the fetus would have retarded the forward movement of the fetus. The sudden anterior force to the uterus would have momentarily caused the posterior uterine wall and the placenta to move away from the fetus, the result being a "vacuum" between the fetus and the posterior wall. When the anterior decelerative force was no longer applied to the uterus the posterior wall would have stopped moving and the vacuum would have caused the fetus to be projected against the placenta on the posterior uterine wall. In this situation the amniotic fluid, instead of acting as a buffer for the fetus, is operating as a vehicle for fetal movement, allowing sufficient mobility for a contracoup injury of the placenta. This is analogous to injury of the occipital region of the brain following a frontal blow, when the cerebrospinal fluid acts as the vehicle for contracoup movement of the brain.¹⁰

In our second case a similar injury was sustained but with the placenta implanted anteriorly in the uterus. With the initial decelerative force applied to the anterior uterine wall the fetus would have been propelled forward. The amniotic fluid, though incompressible, would have allowed the fetus to travel forward with sufficient momentum to apply a sudden force to the surface of the placenta implanted on the anterior uterine wall, causing a "bursting" or irregular laceration of the fetal surface of the placenta. Following disruption of the placental circulation the fetus bled to death in utero, as in our first case.

Uterine injury in the pregnant woman may occur following blunt trauma. Prior to the third month of pregnancy the uterus is protected in the bony pelvis, but beyond 12 weeks it is not, and several cases of uterine rupture following blunt trauma in automobile accidents have been reported. Usually the uterus is compressed between the maternal vertebral column and the steering wheel or the dashboard.^{11,12} Fetal death usually occurs, and the maternal mortality is high.¹³

Management

Recognition of the potential for significant injury

to the fetus and placenta following apparently minor blunt trauma to the mother's abdomen is critical to the initial management of the pregnant trauma victim. Even with relatively few clinical findings on abdominal examination the pregnancy may be seriously compromised. The immediate priorities must be the same as for any patient involved in trauma: ensuring the airway is adequate, ventilating the patient if necessary and maintaining adequate blood pressure and perfusion of the vital organs. In the seriously injured patient an intravenous infusion through a large-bore needle must be established and the urinary bladder catheterized. Pregnant trauma victims should be instructed to lie in the lateral position when possible to avoid further complications of the supine hypotensive syndrome resulting from vena caval compression by a pregnant uterus.¹¹ The pregnant uterus may obscure other abdominal visceral injuries. Occasionally a patient will have obvious evidence of serious or life-threatening intra-abdominal injury; then laparotomy should not be delayed because of the pregnancy. Up until 14 weeks of gestation laparoscopy can be safe and useful in the assessment of lower intra-abdominal injury (A.A. Yuzpe: personal communication, 1979). In questionable cases Rothenberger and colleagues¹⁴ suggest that diagnostic peritoneal lavage can safely be carried out in pregnant women when indications are present.

The presence of a fetal heart beat must be sought at the time of admission, and if a beat is present its rate must be sonographically monitored to assess the well-being of the fetus. Monitoring should be continued until there is good presumptive evidence that the fetus has tolerated the traumatic episode well (such as a reactive pattern to the heart rate in the prolonged nonstress test). If over a period of 24 hours the fetus does not demonstrate evidence of distress, then it probably was not significantly compromised by the accident. The second case we have reported exemplifies the importance of this practice. If the external monitor had been left in place longer, the decline and subsequent cessation of fetal heart activity might have been detected and appropriate measures instituted. In such cases as this, in which fetal compromise is suspected, amniocentesis, either in the initial assessment or intra-operatively, might be a useful adjunct in the assessment of the status of a fetus at least 28 weeks old. Bloody amniotic fluid in the presence of a fetal heart beat might indicate the need for immediate operative delivery, depending on gestational age. Indeed, this could have been the result in our second case. The management of a pregnancy of less than 28 weeks or of a case of prior fetal death, as in our first case, would not be altered, however. If both a good fetal heart tracing and clear amniotic fluid are obtained, continued fetal and maternal monitoring would be appropriate.

If vaginal bleeding is present a modified Singer's test or Kleihauer-Betke staining can be performed on a sample of the blood to determine if fetal erythrocytes are present.¹⁵ The identification of fetal bleeding should prompt immediate delivery by cesarean section

if the fetus is alive and old enough to survive.

If a fetal heart beat is not present at the time of admission it is essential to confirm fetal death to plan further management. Although following blunt trauma to the abdominal wall it may be difficult to hear a fetal heart beat with a fetoscope, examination by Doppler ultrasonography should confirm the presence of fetal heart activity if it exists. If this technique is not satisfactory real-time ultrasonography will be invaluable in documenting the presence of fetal heart activity or pulsatile flow in the great vessels. If the heart valve echoes are well visualized this technique is extremely reliable. If fetal death is presumed to have occurred after the injury any other maternal injuries should be treated and the mother observed until her condition is stable.

The mother requires monitoring for evidence of disseminated intravascular coagulopathy or amniotic fluid embolism regardless of the fetal status. Such complications related specifically to the pregnancy have been reported in the immediate postresuscitation period.¹⁶ Evidence of their development is an indication for immediate induction of labour.

Conclusion

Prompt assessment of the potential fetal risks is essential in the emergency room management of pregnant women sustaining blunt trauma, for apparently minor trauma to the maternal abdomen may have serious and potentially fatal consequences for the fetus. Injuries to the uterus or the fetus, although not common, have been reported on several occasions. However, a discrete placental laceration in the absence of uterine, fetal or serious maternal injury, such as we have reported, has been described in the literature very infrequently. The results of experiments with animals suggest that three-point restraint systems protect the fetus and the mother better than the conventional lap belts. Pregnant women should therefore be instructed to wear lap belts with shoulder harnesses throughout gestation.

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Hypokalemia during the treatment of arterial hypertension with diuretics

GUY LEMIEUX, MD; MICHELLE BEACHEMIN, RN; PATRICK VINAY, MD; ANDRÉ GOUGOUX, MD

In a study of 50 patients with uncomplicated arterial hypertension the administration of hydrochlorothiazide, 50 to 100 mg daily or every other day, with or without reserpine, 0.25 mg daily, resulted in a fall in the mean blood pressure from 182/113 to 144/92 mm Hg. The mean duration of therapy was 19 months. The mean serum potassium concentration was 4.3 mmol/l before the onset of therapy. It fell during the first 6 weeks of treatment, but seldom below 3.5 mmol/l, then rose gradually and spontaneously to 4.1 mmol/l after 19 months of therapy. All the patients remained asymptomatic. These findings bring into question the routine use of potassium supplements or a potassium-sparing diuretic, such as spironolactone or triamterene, during the treatment of hypertension with diuretics such as the thiazides. The use of potassium supplements or a potassium-sparing agent may induce hyperkalemia in spite of the simultaneous administration of a diuretic that acts more proximally. Since hyperkalemia is potentially lethal, the serum potassium concentration should be carefully monitored in any patient receiving potassium supplements or a potassium-sparing agent.

Au cours d'une étude chez 50 patients souffrant d'hypertension artérielle simple l'administration d'hydrochlorothiazide, 50 à 100 mg chaque jour ou aux 2 jours, avec ou sans reserpine, 0.25 mg chaque jour, amenait une chute de la tension artérielle moyenne de 182/113 à 144/92 mm Hg. La durée moyenne du traitement a été de 19 mois. La concentration sérique moyenne de potassium était à 4.3 mmol/l avant le traitement. Elle s'est abaissée au cours des 6 premières semaines de traitement, mais rarement au-dessous de 3.5 mmol/l, puis elle s'est élevée graduellement et spontanément à 4.1 mmol/l après 19 mois de traitement. Tous les patients sont demeurés asymptomatiques. Ces données mettent en question l'emploi routinier d'un supplément de

potassium ou d'un diurétique qui bloque la sécrétion du potassium, comme la spironolactone et le triamterène, au cours du traitement de l'hypertension avec des diurétiques comme les thiazides. L'emploi d'un supplément de potassium ou d'un diurétique à action distale peut induire une hyperkaliémie malgré l'administration simultanée d'un diurétique qui agit à un site plus proximal. Étant donné que cette hyperkaliémie peut être létale la concentration sérique de potassium doit être vérifiée fréquemment chez tout patient recevant un supplément de potassium ou un agent qui bloque la sécrétion de cet ion.

In recent years a great number of drugs, such as methyl-dopa, β -blockers and clonidine, have been introduced for the treatment of arterial hypertension.^{1,2} These drugs are usually administered in conjunction with diuretics because given alone they may induce sodium and fluid retention and thus have less antihypertensive effect.³ We believe that diuretics remain the backbone of therapy for uncomplicated arterial hypertension, as the results of the study we are about to describe unequivocally demonstrate.

Potassium supplementation has long been used to control the hypokalemia associated with diuretic administration. Potassium-sparing diuretics, such as triamterene and spironolactone, are now frequently incorporated in tablets containing diuretics that act more proximally, such as the thiazides.^{4,5} Such practice is not without hazard, however.^{4,5} Our study has shown that in most cases of uncomplicated arterial hypertension these agents are unnecessary, as is potassium supplementation.

Patients and methods

The study was performed on 50 patients selected retrospectively at random from persons attending the renal clinic of Hôtel-Dieu Hospital, Montreal, because of arterial hypertension; 38 had essential hypertension, 4 unilateral renal artery stenosis, 4 chronic pyelone-

From the nephrology-metabolism division, renal clinic and renal laboratory, Hôtel-Dieu Hospital and the department of medicine, University of Montreal

Reprint requests to: Dr. Guy Lemieux, Chief, Renal clinic and renal laboratory, Hôtel-Dieu Hospital, 3840 St. Urbain St., Montreal, PQ H2W 1T8