FIG. 2—Blood sugar during peritoneal dialysis of Case 2. With insulin added to the dialysate, a smooth though low blood sugar level was achieved. On stopping insulin the blood sugar rapidly rose to high levels.

Case 3. Effect of Different Dialysate-Insulin Levels.—A 26-year-old man with a 25-year history of diabetes mellitus was admitted for evaluation before renal and pancreatic allotransplantation. There was a three-year history of renal failure. Initial laboratory studies included a blood urea nitrogen of 128 mg/100 ml and creatinine 18.5 mg/100 ml. During peritoneal dialysis crystalline insulin, 120 units, was added to each cycle. Blood glucose values varied from 340 to 460 mg/100 ml during the 48-hour dialysis. Four days later peritoneal lavage was reinstituted. The identical solution and techniques were used with 145 units of crystalline insulin added to each 2 l. exchange. The blood glucose values varied from 150 to 330 mg/100 ml (Fig. 1). During both dialyses the patient received his usual daily dose of 70 units of insulin zinc suspension. Except for the amount present in the dialysate, no supplemental insulin was given during either dialysis. In a 10-day period before treatment the blood glucose values had varied from 137 to 432 mg/100 ml. During the second dialysis the intraperitoneal absorption of insulin was evaluated.

Results and Discussion

Diabetic patients with renal failure are being treated in a number of medical centres with dialysis and renal transplantation. Hyperglycaemia is known to be a serious complication of peritoneal dialysis in this group of patients. Boyer *et al.* (1967) noted that after three one-hour exchanges in non-diabetic patients 32.4 g of glucose was absorbed from 1.5%

glucose solution. Even in patients with a normal carbohydrate tolerance pronounced rises in blood glucose can occur and may result in significant extracellular fluid hyperosmolality and non-ketacidotic coma (Hutchings *et al.*, 1966; Ribot *et al.*, 1966; Boyer *et al.*, 1967).

Our first case illustrates the problem in the management of diabetes mellitus during peritoneal dialysis. Urine glucose determinations are of no value and frequent blood glucose evaluations are required. In contrast there was no hyperglycaemia during the peritoneal dialysis of the second and third patients when intraperitoneal insulin was administered. Further, the blood sugar level remained very constant during the period of treatment.

The radioimmunoassay of insulin showed that 96% of the infused ^{125}I insulin was recovered in the peritoneal drainage. The remaining 4% was present in the patient's blood (see Table). During the one-hour cycle studied 35 g of glucose

Results of Radioimmunoassay of Insulin During Peritoneal Dialysis

Added to dialysate	61.0 × 10 ⁶
Total in potassium hydroxide eluate	2.4 × 10 ⁶
Given intraperitoneally	58.6 × 10 ⁶ (100%)
Recovered in effluent dialysate	56.1 × 10 ⁶
Recovered in potassium hydroxide eluate	0.2 × 10 ⁶
Total recovered	56.3 × 10 ⁶ (96%)
Present in blood	2.3 × 10 ⁶ (4%)

The counts are total counts based on measurements in aliquots minus background.

was absorbed. The dynamics of the intraperitoneal administration of insulin have not been studied in man. In view of crystalline insulin's low molecular weight, the slow absorption from the peritoneum is difficult to explain. Data from our second patient suggest that the peritoneal absorption of insulin may be proportional to the quantity instilled with the dialysate, as lower blood sugar values were obtained with larger doses of insulin.

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MEDICAL MEMORANDA

Lessons from Two Steering Wheels

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Roberts (1967) considered that ruptures of the solid viscera in vehicle occupants were caused by "direct impingement and localized stress concentration" but that the mechanism of

rupture of hollow viscera "must be one of shearing resulting from contact in the abdomen between the abdominal wall, which is thrust inward by the steering wheel, and the spinal column." This and similar research evidence has probably influenced the National Highway Safety Bureau of the United States in drafting their proposed new standards for car occupant protection (Docket No. 69-7 Notice 4). Standard 4.4.7 reads: "The force on the abdominal region shall not exceed 2,400 pounds [10.7 kg N] and the pressure on the abdominal region shall not exceed 30 pounds per square inch [2.1 kg/cm²]."

An analysis of abdominal injuries found in 483 necropsies of car occupants we have studied has shown that about 50% of the victims suffered ruptures of the solid viscera (liver, spleen, adrenals, and kidneys in that order) and all also had injuries to other body areas. From the same material there were only 14 patients with ruptures of the intestinal tract—12 of the small bowel and 2 of the stomach. Two drivers had

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both small-bowel and large-bowel injuries. It is significant, however, that 12 of the victims were car drivers and only two were front-seat passengers. No rear-seat occupant suffered this type of injury. It seemed likely, therefore, that the steering wheel was of some importance in such injuries.

Two Accidents

Within a period of four weeks this year two drivers have been admitted to our hospital with hollow visceral injuries, the mechanism of which we have been able to establish. The first driver's car was in a head-on collision with another car of comparable size, both travelling at around 35-40 m.p.h. (56-64 km p.h.). He was thrown forward on to his steering wheel (Fig. 1). The second driver crashed into the rear of a tanker at night on a motorway. The speed of the car was around 70 m.p.h. (112 km p.h.) and that of the tanker some 20-30 m.p.h. (32-48 km p.h.) less. This car driver also was thrown against the steering wheel (Fig. 2). The impact of abdomen against steering wheel seems to have been similar in both drivers. The injury sequences were probably the same but are more clearly shown in the second driver, whose skin had the unmistakable marks of the impact of his abdomen against the steering wheel (Fig. 3). When this car collided the driver continued to move forward until his abdomen hit the wheel's rim, causing the transverse bruise at the umbilical level, shown in Fig. 3. Still moving forward at speed, he hit the spoke of the steering wheel, which at one point had broken with chisel-sharp edges. Early surgery revealed a complete division of the jejunum 4 in (10 cm) below the duodenojejunal junction. We attribute this division to a shearing force caused by the chisel-sharp broken spoke of the steering wheel against the body of a vertebra. This patient also suffered a 3-in (7.5-cm) transverse split of the outer coat of his transverse colon that may have been caused by either the rim of the wheel or the broken spoke.

The other driver also suffered an abdominal impact against the steering wheel, and the spoke of the wheel broke with similar chisel-sharp edges (Fig. 1). During 24 hours of observation this driver showed no clinical signs or symptoms that would indicate an abdominal injury and there were no marks on the abdominal skin indicating a severe abdominal impact. He was very keen to return home and was allowed to do so. Ten days later he returned with an advanced retroperitoneal cellulitis that failed to respond to surgery, and a rupture of the second part of his duodenum was confirmed at necropsy. It is probable that this was a delayed rupture, and again this injury seems to have been caused by the chisel-sharp spoke of the steering wheel impacting the duodenum against its adjacent vertebra.

From these two cases and other operative evidence of duodenal and high jejunal injuries it seems that the small bowel is most vulnerable when impacted in areas where it is fixed or tied down relatively closely to the posterior abdominal wall and so can be trapped against the underlying vertebra.

Comment

Both these steering wheels might be thought to be of "safe" design. They had recessed hubs from which three spokes ascended to the rim. Both rims and spokes had tubular metal cores covered by a hard plastic material. The plastic material round the rims was thin but that over the spokes was appreciably thicker. The tubular metal cores of both rims and spokes became deformed in the predictable manner, but the thick plastic covering of the spokes broke with a sharp projecting and chisel-shaped angle. Had the covering of the spokes been as thin as that of the rims or had it been of a more flexible material, the spokes might have become deformed in a less dangerous way. When several materials are mixed, as they were in the construction of these steering wheels, each should complement the other by less dangerous deformation under impact. Perhaps the proposed American standard should be amended to cover this point.

It should be noted that both these drivers did not wear their seat belts. Had the forces of deceleration been spread over the bony structures of their chests and pelvis by closely

fitting diagonal and lap belts they would have escaped their abdominal impacts. If the steering columns had been of collapsible or telescopic types the abdominal impacts might have been less severe.

In the United States, as in Europe and the U.K., work on

FIG. 1.—Steering wheel with a broken spoke that caused a fatal retroperitoneal rupture of the second part of the duodenum. This wheel and that shown in Fig. 2 are in their post-crash positions.

FIG. 2.—Steering wheel with a broken spoke that caused a rupture of half the circumference of the transverse colon and a complete division of the jejunum. Early surgery resulted in complete recovery. (Mr. Henry Proctor's case.)

FIG. 3.—The 10-in (25-cm) transverse mark at umbilical level was caused by impact with the wheel's rim, and the higher mark by impact against the broken spoke resulting in the hollow visceral ruptures.

protective car design proceeds at three levels which are distinct and approached in the following order—research, standardization, enforcement. Whatever research evidence is used to formulate standards for protective car design that may ultimately be enforced, it seems clear that a fourth level of procedure is essential—namely, continual research into the actual crash performance of cars that have the new and developing protective designs and devices, so that the maximum degree of protection of occupants may be achieved.

We are indebted to Mr. Henry Proctor for Fig. 3.
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Scrotal Gangrene after Male Sterilization

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Since Fournier (1884) first described idiopathic scrotal gangrene there have been sporadic case reports and various theories concerning its aetiology. The present case, following the operation of vasectomy, is of interest in view of the increasing popularity of male sterilization.

Case Report

A healthy 49-year-old man was sterilized on 20 November 1969. The bilateral vasectomy was performed under local anaesthetic (1% lignocaine without adrenaline) and the initial postoperative period was uneventful. On his way to work on 3 December he felt unwell and returned home to bed with fever, headache, and rigors. That night he developed a severe pain in the scrotum and was visited by his family doctor. The scrotum was swollen and red; the incision on the left side was soundly healed but that on the right side was a little moist. The pain and swelling subsided after treatment with tetracycline, 250 mg four times a day, but suddenly over the course of 36 hours the skin started to ulcerate.

He was admitted to hospital on 15 December when he looked well and was afebrile (98.8°F; 37.1°C). The scrotal skin was gangrenous and in places it had sloughed away to expose the testes. Investigation showed a haemoglobin of 13.4 g/100 ml and a white cell count of 13,200/mm³ (80% neutrophil polymorphs). There was no glycosuria, and a midstream specimen of urine was sterile on culture. A swab from the scrotum showed pus cells and lactose-fermenting organisms.

When the necrotic scrotal skin was removed under a general anaesthetic, healthy testes and cords were exposed (Fig. 1). Histological examination of the scrotal skin showed complete necrosis with a pronounced neutrophil leucocytic infiltration of the deeper tissues and the skin itself. A few small vessels were occluded by thrombus. Many unidentified Gram-negative cocci were present.

A further scrotal swab yielded a growth of lactose-fermenting coliform and *Proteus* organisms. Both organisms were sensitive to ampicillin, and 500 mg four-hourly was given during the next week. The condition of the scrotal skin rapidly improved with daily eusol dressings and the local application of polybactrin. Suf-

FIG. 1—Appearance after debridement of scrotal slough.

ficient re-epithelization had occurred after four weeks to allow the patient to continue with dressings at home. One month later the scrotum was completely healed.

Comment

This case illustrates many of the characteristic features of Fournier's gangrene with the explosive onset of toxæmia and the rapid progression of scrotal gangrene in a previously healthy patient. Though the aetiology of the disease remains obscure, the scrotal incisions in this case must be implicated as the likely site of entry for the infection. Randall (1920), in a review of 16 cases, found that six had neighbouring local lesions which may have acted as a portal of entry. Thomas (1956) observed that the condition was secondary to such lesions as balanitis, chancroid, or minor abrasions in about one-third of the cases and noted its occurrence following circumcision, herniorrhaphy, haemorrhoidectomy, incision of perirectal abscess, and injection of a hydrocele.

The occurrence of scrotal gangrene after vasectomy has not been previously reported. It should not deter patients from this form of sterilization, as there were few complications in a recent review of 1,000 cases of male sterilization (Simon Population Trust, 1969). The risks associated with female sterilization are greater, though these will be reduced by the greater use of endoscopic sterilization (Steptoe, 1970).

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